IN THE CIRCUIT COURT OF THE EIGHTEENTH JUDICIAL CIRCUIT, DU PAGE COUNTY, ILLINOIS

PEOPLE OF THE STATE OF ILLINOIS, Plaintiff	)	
v.	)	No. 02 CF 654
RANDY LIEBICH, Defendant	)	

Petitioner's Rule 651(c) Certificate

I, Jaime Escuder, counsel for Petitioner, certify pursuant to Supreme Court Rule 651(c) that I and my co-counsel have consulted with petitioner in person, by mail, and by telephone to ascertain his contentions of deprivation of constitutional rights; I and my co-counsel have examined the record of the proceedings at the trial; and we have made all amendments to the petitions filed *pro se* that are necessary for an adequate presentation of petitioner's contentions.

Respectfully Submitted,

BY:_____
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Deputy Chief Public Defender

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RANDY LIEBICH'S SUPPLEMENTAL
PETITION FOR POST-CONVICTION RELIEF

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The Petitioner, RANDY LIEBICH, by and through his attorney JEFFREY R. YORK, DuPage County Public Defender, through his assistant, JAIME ESCUDER, and TARA THOMPSON of the Exoneration Project at the University of Chicago Law School, pursuant to 725 ILCS 5/122-1, Article I, Section 2 of the Constitution of Illinois, the Fifth and Fourteenth Amendments to the Constitution of the United States, and for the reasons set forth below, hereby supplements Mr. Liebich's prior *pro se* and amended post-conviction petitions and requests that this Honorable Court enter an order vacating his conviction and granting him a new trial. The Petitioner's statement follows.

I. EXECUTIVE SUMMARY

Randy Liebich has spent a decade incarcerated for a crime that he did not commit. Indeed, it is a crime that he *could not* have committed. As this Petition demonstrates, based on the affidavits of medical experts and witnesses who observed Steven in the days before his death, Steven Quinn died as the result of injuries that Steven sustained days before he was in Randy's care.

Randy's conviction arose out of a 2004 bench trial, in which treating physicians testified that the victim, two-year-old Steven Quinn, had been beaten during the morning and afternoon hours of February 8, 2002. Randy became the obvious suspect because he was the only person watching Steven that day.

However, Steven's autopsy revealed abdominal injuries that were days older than the brain swelling that was the immediate cause of Steven's death. This is significant because February 8 was the only day that Randy had watched Steven

and Angelique in the applicable time period. Thus, if the injuries that ultimately led to Steven's death occurred before that day, they could not have been caused by Randy.

The treating physicians did not consider whether the abdominal injuries, which included a section of necrotic (dead) bowel that was leaking into the abdominal cavity, causing peritonitis, pancreatitis and infection, could have resulted in the brain swelling that led to Steven's death. Nor were these physicians sufficiently examined on this possibility at trial, despite the fact that evidence was available showing that Steven had been fussy and eating poorly in the days before his hospitalization. Instead, the focus at trial was on the brain swelling, which the doctors insisted occurred within hours before Steven's admission to the hospital on February 8, and which they assumed must have been caused by a beating that had occurred earlier that day.

Subsequent analyses by medical experts using updated scientific methodology have confirmed, however, that Steven had predisposing conditions and newer abdominal injuries, all of which were present before February 8, that would fully explain the brain swelling. These included myocarditis (damage to the heart) and a peripancreatic hematoma with scarring, both of which were present for at least a week before hospital admission. Other abdominal injuries -- including damage to the bowel, diaphragm and liver -- were present at least two days before hospital admission. By the time of hospital admission, these injuries had led to pancreatitis, a hypoxic brain (a brain that had been deprived of oxygen), and a

coagulopathy (a bleeding/clotting disorder). This timeline is corroborated by the child's symptoms of lethargy, whininess, and poor eating in the days before his collapse. All nine experts who have re-examined the medical evidence, including the Cook County Medical Examiner who conducted the initial autopsy, reach essentially the same conclusions: Steven died from medical conditions and abdominal injuries/infection that preceded the day that Randy cared for him.

Randy Liebich was wrongly accused and wrongly convicted. Based on the facts and legal argument set forth below, this Court should reverse Randy's conviction and grant him a new trial.

II. FACTS

A. Factual Background

1. Randy meets and lives with Kenyatta Brown, Steven Quinn's mother, who is abusive towards Steven.

Kenyatta Brown bore Steven Quinn when she was fifteen years old. *See* Record on Appeal ("RA") at 113. Soon after his birth, Kenyatta and the boy's father, Steven Quinn Sr., abandoned Steven with friends. *See id.* at 990. As a result, Steven was raised by Kenyatta's mother, Karen Clark, and his great-aunts, Sadie Brown and Dorothy Herron. *See id.* at 990, 992.

Randy and Kenyatta met in February 2000. *See id.* at 33. They soon developed a relationship and lived together for most of the following two years, sometimes sharing living arrangements with friends or living with Randy's mother. From 2000-2002, Steven visited Randy and Kenyatta regularly. In the summer of

2001, Steven stayed with Randy and Kenyatta at an apartment that they shared with friends, including Crystal Zeis. *See id.* at 1896. In 2001, a few months before the birth of their child, Angelique, Randy and Kenyatta moved to their own apartment in Willowbrook. *See id.* at 34.

At trial, several of Randy's friends and relatives, including Ms. Zeis, testified that Kenyatta was very rough with Steven, punishing him inappropriately, slapping him without cause, and tossing him across a room. Ms. Zeis testified that when she lived with Randy, Kenyatta and Steven, she saw Kenyatta "screaming at Steven, and then she grabbed him and threw him out of the kitchen." Steven landed on "[h]is back or his butt." *Id.* at 1881-82. Ms. Zeis further testified that "[e]very time [Steven] did something wrong, he ended up getting hit for it;" Kenyatta would "knock him upside the head a lot, actually." *Id.* at 1885.

Denise Foster, Randy's sister, testified that she was present when Kenyatta "kept telling [Steven] to shut up and smacking him on his leg." *Id.* at 1787. Frank Belpedio, Randy's cousin, testified that in June or July 2001, while he was driving with Kenyatta and Steven in the back seat of his car, he "heard a very loud slap" and saw Kenyatta "taking her hand and slapping [Steven] in the head." A few weeks later, he saw Kenyatta slap Steven so hard that he fell off the couch. *Id.* at 1799; 1802. Mr. Belpedio described this as a slap "on the back of [Steven's] head, open hand, and then there was a slap on the back too." *Id.* at 1802. Mr. Belpedio also testified that he saw Kenyatta "walk over to Steven with her right arm and just

started shaking the kid, and his head was going like this, you know, flailing around." *Id.* at 1806.

According to the trial testimony, from 2000-02, Steven visited Randy and Kenyatta regularly. These events occurred with regularity during the summer of 2001 and continued into the days before Steven's collapse. Since Judge Jorgensen rejected this testimony on grounds of credibility, we do not discuss it further, other than to note that it is consistent with the information provided by Randy and others, including Kenyatta's mother, who told the police that, although she had never seen Randy hit Steven, Kenyatta had "slapped Steven in the face a few times in the past due to his whining and crying." (Ex. 27, DuPage County Sheriff's Police Reports at MR 19.) Karen also described her daughter as an "excellent liar." (*Id.* at MR 18.) We further note the undisputed testimony that Randy and Steven appeared to have a good relationship and that Randy was never seen to hit or discipline Steven.

On January 27, 2002, Kenyatta gave birth to Angelique. RA at 36, 997. At that time, Steven was staying with his great aunt, Dorothy Herron, who ran a home daycare. See *id.* at 37, 997. On February 2, Randy and Kenyatta brought Steven home to their apartment in Willowbrook. *See id.* at 38. At that point, Randy was working at the *Patio* restaurant in the evening and Kenyatta was working at the Yorktown Mall, where she conducted research surveys, in the day. *See id.* at 40, 88. In her victim impact statement, Kenyatta described this period as follows:

> In the month of February of 2000, I met Randy and the next two years were spent planning a life together along with Steven Jr. I finally got

> my life together. Randy and I both had jobs, a new apartment, and a new baby together. I couldn't ask for anything more. It was perfect.

Id. at 2251.

Six days after arriving at their home, Steven collapsed, and the doctors at two hospitals told Kenyatta that Randy had beaten Steven Jr. all over his body. Kenyatta could not imagine that someone she loved, trusted, planned to marry and share the rest of her life with could do such a terrible thing to her son. *Id.* at 2252. As it turns out, Kenyatta's instincts were right: Randy did not beat Steven. The doctors had simply confused the end results of earlier abdominal injuries/infection -- specifically, pancreatitis, a hypoxic brain and a secondary coagulopathy -- with a beating.

2. Events of February 2-8, 2002.

Judge Jorgensen stated in her ruling that the central issue was what happened to Steven "between when he left the care of Dorothy Herron on 2/2/02 in the late hours of February 8, 2002, when he was determined to be brain dead by Dr. Munoz." *Id* at 5. We review the events of this period in some detail.

a. February 2-6, 2002. From February 2 to February 6, Steven was primarily watched by Kenyatta. See *id.* at 42. Kenyatta testified that February 8, 2002 was "the only time" that Angelique and Steven had been left alone with Randy. *Id.* at 90. Randy's boss, Nicolas Brinias, testified that Randy was at work on February 4 and part of the day on February 5, 2002. *See id.* at 1348, 49. He was also scheduled to work on the evening of February 8. See *id.* at 1031.

From February 2-6, Randy, Kenyatta, Steven and Angelique visited, and were visited by, Randy's relatives. (Ex. 14, Foster Aff. at ¶ 2; Ex. 16, Minucciani Aff. at ¶ 2.) During the family visits, Steven was quiet and ate poorly. See *id.* In fact, Steven refused to eat the McDonald's food that Denise Foster and Debra Minucciani had brought during their visit. See *id.* As recently as February 9, 2012, Kenyatta told an investigator that Steven had a stomach ache two or three days before his collapse on February 8. (Ex. 13, Lilly Aff. at ¶ 12.) Randy noticed that Steven was quieter than usual and thought that Steven might have been a little jealous or depressed because the baby was getting a lot of attention. (Ex. 10, Randy Liebich Aff. at ¶ 10.) The last couple days, Steven wouldn't eat unless Kenyatta almost made him eat. (*Id.*)

b. February 7, 2002. On February 7, Kenyatta primarily cared for Steven and Angelique. Randy went to work, but found he had confused his schedule and returned home. (*Id.* at ¶ 12.) Before Randy returned, Kenyatta made Steven pork chops for dinner, but Steven refused to eat them. *See* RA at 48. As punishment, Kenyatta sent Steven to his room, telling him that he could not come out until he was ready to eat. *See id.* at 48, 125. After this, there are some variances in the history provided by Kenyatta and Randy. Since Randy did not testify at trial, we first summarize the information provided by Kenyatta and then note some of the variances with the information that Randy provided to investigators and others.

(1) *Kenyatta.* According to Kenyatta, on February 7, Steven came out of his room and watched television when Randy came home around 8:30 p.m. *See id.*

at 50. When she again offered Steven dinner, Steven refused to eat and was sent back to his room, whereupon he began to cry. *See id.* at 54. At trial, Kenyatta testified that Randy followed Steven into his room and that, shortly thereafter, she heard a "hollow sound." *Id.* at 55. When Steven continued to cry, she asked Randy if he had struck Steven, and Randy said no. *Id.* at 55. Eventually, Kenyatta "muffed" Steven by pushing her fingers against the child's temple. *Id.* at 62.

When Steven still continued to cry, Kenyatta told Steven that she was going to spank him, asked Randy for his belt and spanked Steven on his diaper with the belt. *Id.* at 63-64. When the crying continued, Kenyatta removed Steven's diaper and spanked him on his bare skin with her hand. *Id.* at 66. When Steven continued to cry, Kenyatta put his diaper back on and she and Randy left the room. Id. Eventually, Steven stopped crying, came out of the room, and told Kenyatta he was ready to eat. Id. Kenyatta gave him his plate, but Steven ate only half of what was offered. *Id.* at 67. The family then went to sleep for the night. *See id.* at 68-69.

(2) *Randy.* According to Randy, Steven was in his room crying when he came home, but came running out when he heard Randy return. (Ex. 10, Randy Liebich Aff. at ¶ 12.) When Randy asked why Steven was in the bedroom, Kenyatta said she made Steven go to the bedroom because he wouldn't eat his dinner. (*Id.*) Kenyatta told Steven to go back in the bedroom, which he did. (*Id.*) Kenyatta later slapped Steven on the side of his head (presumably what Kenyatta describes as "muffing"), hit Steven with the belt over his diaper, and then removed the diaper and swatted him on the rear end with her hand. (*Id.* at ¶ 13.) Kenyatta did not,

however, ask Randy for his belt; instead, she pulled the belt out of Randy's pants, which were lying on the floor. (*Id.*) According to Randy, Steven did not eat or come out of his room that night, and his plate of food, covered by plastic wrap, was still in the refrigerator the following morning. (*Id.* at ¶ 14.)

c. February 8, 2002. On February 8, Kenyatta left for work around 10:10 a.m. *See* at 75. Steven was still in bed, and Kenyatta poured cereal into a bowl and told Randy to make sure Steven ate it. *Id.* at 74.

After Kenyatta left, Randy gave Steven the bowl of cereal with milk. With encouragement, Steven ate the cereal but left the milk. After Steven played with the dog, Steven and Randy watched TV, and Randy and both children fell asleep. Randy subsequently left the house for under five minutes to borrow a cigarette from a girl who worked at McDonald's. When he returned, both children were still sleeping.

Around 3 p.m., Randy fixed Steven a hot dog, which he cut up and put on a plate with ketchup. (Ex. 10, Randy Liebich Aff. at ¶ 17.) He again had to coax Steven to eat. (*Id.*) Steven drank orange juice and ate a little of the hot dog, but started choking when Randy gave him water. (*Id.*) Randy patted him on the back and put his finger in Steven's mouth to see if he had some hot dog caught in his throat. (*Id.*) However, Steven bit down on his finger. (*Id.*) When he didn't let go, Randy slapped him lightly on the cheek to get him to let go. (*Id.*) He also patted him on the back to dislodge any food that might be stuck. (*Id.*) When Steven let go, there was a little bite mark on Randy's finger and some vomit in Steven's mouth, and

Randy cleaned him up a bit. (*Id* at ¶ 18.) Steven seemed a bit dazed but indicated he was okay. (*Id.*) Randy and Steven watched Jurassic Park, and Steven went to sleep. (*Id.*)

When Kenyatta got home at 4:30, she found Randy in the living room with Angelique asleep on Randy's chest. RA at 78. Steven was also lying down in the living room. *Id.* at 79. Kenyatta took Angelique from Randy so that Randy could get dressed for work. *Id.* at 81. Kenyatta noticed that Steven appeared to be short of breath and saw what she believed to be vomit in his mouth. *Id.* at 82. Kenyatta handed Angelique to Randy, who had returned to the living room, and began to question Steven on how he was feeling. Steven did not respond. *Id.* at 83.

Kenyatta asked Randy how long Steven had been in this condition, and Randy said, "I don't know. About an hour." *Id.* at 83. Since Steven seemed unwell, Kenyatta told Randy that she wanted to use their only car to take Steven to the hospital. *See id.* at 85. Initially, Randy said that Kenyatta could not have the car since, due to her history of prostitution, he thought that she was actually planning to pick up men in Chicago. *See id.* at 85. Kenyatta assured him that she would take Steven to the hospital. Randy said he would accompany her, but he wanted to stop by his job first so that they would know why he would not be working that evening. *See id.* at 86, 88. Kenyatta did not object to this plan, nor did she think it necessary to call an ambulance since at that time Steven did not appear to be seriously ill. *See id.* at 145.

Kenyatta drove to the *Patio*, and they arrived around 5:15 p.m. *See id.* at 1031. Randy removed Steven from the back seat so that he could show his boss that Steven was ill. *See id.* at 89. Randy showed Steven to his boss, John Georgopolous, who testified that he saw no marks on Steven and that Steven appeared to have the flu. *See id.* at 1032-33. Randy returned to the car after four or five minutes, and Kenyatta drove to Mount Sinai hospital in Chicago. *Id.* On the way to the hospital, Randy told Kenyatta about Steven choking on the hot dog, his attempt to clear Steven's airway, and Steven's biting down on his finger. *See id.* at 90. The family arrived at Mount Sinai approximately 35 minutes after leaving their home. *See id.* at 91.

B. Steven's Medical Treatment and Contemporaneous Diagnoses

1. Overview.

At Mount Sinai, Steven was viewed as ill, since there were no signs of trauma. *See* RA at 1043 (Dr. Green did not "see any signs of trauma initially, so I thought maybe this is something medical"). When a computed tomography ("CT") scan showed a subdural hemorrhage, and various marks began to appear on Steven's body, however, the diagnosis switched to abuse, specifically head trauma. *See id.* at 1062 (Dr. Green testified that the trauma team was "brought in on the case after we found out about the CT scan"). An initial misread of the CT scan by a radiology technician and a trauma consultant, Dr. Tracy Boykin, indicated that there was a large subdural hemorrhage, and Steven was transferred to Rush Medical Center, so that it could be evacuated. *See id.* at 1056, 1114 (Dr. Boykin

stated that her read of the CT scan was that it was "really bad"; Dr. Green stated that, after learning from Dr. Boykin that "there was indeed a bleed ... we got our transport team and all the facilities together to get the kid transferred to Rush"). As it turned out, the hemorrhage was insignificant but Steven's brain was severely swollen. *See id.* at 1685, 1689 (Dr. Munoz stated that, based on the CT scan, he would have thought that Steven "would have a larger subdural hematoma" but the pressure in Steven's skull was "incompatible with life"). As set forth in the expert medical affidavits attached as Exhibits 1 through 9, brain swelling is a nonspecific finding that can reflect reflect lack of oxygen (hypoxia ischemia) from any cause, including infection.

Although laboratory tests at Mt. Sinai and Rush confirmed that Steven had acute pancreatitis, no abdominal CT or exploratory surgery was done. Dr. Munoz, the Rush neurosurgeon who operated on Steven, cancelled the abdominal CT scan at Mt. Sinai when he arranged for transfer to Rush. He also cancelled the abdominal CT scan and surgery at Rush following the neurosurgery since Steven was "virtually brain dead." *Id.* at 1695. However, a penrose drain (a soft rubber tube used to drain fluids from the area of a wound) inserted into Steven's abdomen the following morning confirmed abdominal injuries and infection, and the autopsy subsequently identified 7 inches of necrotic (dead) bowel with a small perforation, peritonitis (inflammation in the area surrounding the bowel), and early-stage pancreatitis (inflammation of the pancreas). (Ex. 22, Postmortem Examination

Report.) The autopsy also confirmed that the abdominal injuries occurred days before Randy cared for Steven (*Id.*) (stating "injuries subacute and/or day ~ 5").

At trial, a great deal of testimony focused on the marks and lines that appeared on Steven's body after hospital admission. Although these were often characterized bruises and whip, lash or rope marks, some of them were simply well-known signs of pancreatitis. These included "bruising" around the umbilicus, known as "Cullen's sign," and a swollen scrotum. Other marks reflected disseminated intravascular coagulation (a bleeding/clotting disorder), as confirmed in the laboratory tests. The only marks suggestive of trauma consisted of circular marks on the child's back of undetermined age and cause.

With this overview in mind, we summarize the medical findings, as reflected in the medical records and trial testimony.

2. Initial treatment at Mount Sinai.

At Mount Sinai, Steven was treated by Dr. Paula Green, an emergency room doctor, and Dr. Tracy Boykin, a consulting trauma doctor.

a. Dr. Green. Dr. Green was the first doctor to see Steven at Mount Sinai. At trial, she testified that, as an emergency room doctor, she sees child abuse cases "all the time." RA at 1043. As Steven was being undressed by the nurses, she looked for signs of abuse on his body and saw none. *See id.* In fact, she testified that she was "stunned" by the lack of traumatic signs on the boy's body. *Id.* She assumed, therefore, that Steven's illness was metabolic rather than traumatic. *See* id. This

was consistent with an initial lab test showing exceptionally high glucose. *See id.* at 1082.

As the staff was preparing Steven for a CT scan, Dr. Green noticed a mark on Steven's scalp. The mark was red, which suggested that it had just occurred. *See id.* at 1044. She also noted that Steven's gaze was fixed to the left, that he had a bruise on his lips, and that he was starting to whine or grunt during his breathing. *See id.* at 1045. To Dr. Green, these findings indicated that Steven was suffering from a head injury. *See id.* Dr. Green also noted that Steven's abdomen was "nondistended," i.e., not bloated or tender. *Id.* at 1047. When Dr. Green asked Randy what had happened, Randy told her that the only unusual incident was that Steven had choked while eating a hotdog and drinking water. *See id.* at 1042. Randy denied striking or patting Steven. *See id.*

Dr. Green testified that arrangements were made for transport to Rush when a radiology technician identified a bleed in the CT scan. *See id.* at 1056. She testified that severe brain damage and bleeding can also be caused by lack of oxygen. *See id.* at 1071-72. Dr. Green testified that Dr. Munoz, the Rush neurosurgeon, cancelled the abdominal CT since they were focusing on the head. *See id.* at 1084-85.

Dr. Green described Randy as calm, restrained and respectful. *See id.* at 1084.

b. Dr. Boykin. Like Dr. Green, Dr. Boykin initially believed that Steven was ill or suffering from a metabolic disorder. *See id.* at 1112 (Dr. Boykin stating

"[a]t that time Dr. Green was working on the child and she had gotten a blood sugar that was high and thought maybe perhaps there could be some metabolic reason as to why this child [was] unresponsive"). However, a radiology technician told Dr. Boykin that the CT showed that his head was "full of blood." *Id.* at 1113. Dr. Boykin testified that she became angry when she saw the CT scan and told Randy and Kenyatta that it looked like Randy had been "sitting at home beating on [Steven] all day." *Id.* at 1114-15. She became even angrier when Randy failed to respond or provide additional explanations for Steven's condition after she told him that Steven's injuries were inconsistent with choking on a hot dog. *See id.* When Randy responded in a manner suggesting that he didn't know how Steven got the injuries, Dr. Boykin told him that she was going to call the police, which she did. *See id.* at 1116.

As for her medical testimony, Dr. Boykin testified that it is unlikely that a person with a subdural hemorrhage would die suddenly, and that severe abdominal injuries can lead to a hypoxic brain and heart. *See id.* at 1126, 1129 (Dr. Boykin stating that "[i]t's not likely the person person with a subdural hematoma will go on to die," and that a person who suffered severe abdominal injuries would suffer "hypoxia into your brain, which really wouldn't be as much of a problem as hypoxia to your heart"). She testified that she didn't know if much attention was paid to the abdomen as she was only a trauma consultant. *See id.* at 1129. She also did not know what Dr. Munoz found when he attempted to evacuate the subdural. *See id.* at 1136.

c. Mount Sinai labs and final report. The Mount Sinai labs confirmed Steven had acute pancreatitis. (Ex. 24, Mount Sinai Report.) These labs were not ordered until the Rush transport team asked that they be added and may not have been available before transfer. The Mount Sinai report notes various marks that appeared on the child's body during hospitalization, as seen by Dr. Green and Ms. Beasley, the attending nurse (*Id.*) The final Mount Sinai discharge record notes "pancreatic disease," but the final diagnosis was intracranial bleed; hypothermia (low body temperature); and hyperglycemia (high blood sugar).

3. Transport from Mount Sinai to Rush Medical Center.

The Rush transport notes state that the CT scan showed a "large" right subdural hemorrhage. The notes also describe bruising and ecchymosis (reddened areas) in the head, back, abdomen, scrotum and between the legs on the upper thighs, none of which had been present on admission (Ex. 25, Rush Medical Center Transport Notes.) The transport team asked Mt. Sinai to add amylase and lipase testing, indicating that at least team member suspected abdominal injuries/infection. (*Id.*). At trial, Tammy Smith, the transport nurse, testified that she had asked the Mt. Sinai transport resident, "why are we taking him to Rush? He's dead." *RA* at 1262.

4. Rush Medical Center.

At Rush Medical Center, Steven was treated by several physicians, including Dr. Paul Severin, pediatric critical care, and Dr. Lorenzo Munoz, a neurosurgeon.

a. Dr. Severin. Like Dr. Green, Dr. Severin's first impression was that Steven was ill rather than suffering from trauma. At trial, Dr. Severin described Steven as "toxic ill appearing." *Id.* at 1374. In his initial examination of Steven around 10 P.M., Dr. Severin heard bowel sounds and noted that the abdomen was soft and not distended, which indicated that abdominal issues were unlikely. *See id.* at 1383-84. However, the lab tests told a different story. When Dr. Severin reviewed the results of the 9 P.M. blood draw, he found that the levels of the pancreatic enzymes amylase and lipase were "really high" – in the thousands rather than the usual 200 range. *Id.* at 1386, 1387. When he compared these results to those from Mount Sinai, he discovered that Steven's enzyme levels had "almost doubled" since Mount Sinai. *See id.* at 1387.

The following morning, Dr. Severin observed a lack of bowel sounds and the "Cullen's Sign," a redness around the navel that indicates "hemorrhagic pancreatitis." *RA* at 1390-91. That morning, a penrose drain that was inserted into Steven's abdomen drained a large quantity (500 cc) of bloody fluid, confirming abdominal infection. (Ex. 26, Rush Medical Center Medical Records. at 84a and 84b.) The fluid was not cultured.

At trial, Dr. Severin testified that he had reviewed Steven's autopsy results and learned that there was a perforation in Steven's bowel, specifically in the portion of bowel known as the "jejunum." *See* RA at 1392. Dr. Severin explained that when blood flow to the bowel is restricted for any reason, the tissue starts to break down, leading to the leakage of stool into the abdomen. *See id.* at 1393. This

can lead to pancreatitis, which would have made Steven unable to eat. *See id.* at 1395.

At trial, Dr. Severin testified that, in his opinion, Steven's injuries occurred within 24-48 hours of his examination, a period that included the evening of February 6 and February 7. See RA at 1426. Dr. Severin admitted that he had never before been called upon to give a timing of injury. *See id.* at 1400. Dr. Severin also testified that abdominal injuries are immediately symptomatic. *See id.* at 1416. While abdominal injuries can be slowly progressive, with only minor symptoms, such as lethargy or poor eating, Dr. Severin was correct that the child would have been seriously symptomatic by the time he developed the full-blown abdominal infection evident on admission.

b. Dr. Munoz. Dr. Munoz performed neurosurgery to evacuate the "large" subdural hemorrhage identified at Mount Sinai at about 10 p.m., approximately four hours after Steven's arrival at Mount Sinai. *See id.* at 1678. Dr. Munoz did not, however, find a significant subdural hemorrhage. *See id.* at 1685. Indeed, the postoperative diagnosis states "no subdural hemorrhage." (Ex. 26, Rush Medical Center Medical Records at 77-78.) By then, however, the pressure in the skull was so great that Steven's brain began to herniate out of the skull. *See* RA at 1682. Since Steven was nearly brain dead, Dr. Munoz cancelled the abdominal evaluation, specifically suggesting that exploratory surgery not be done on Steven's abdomen. See *id.* at 1695.

At trial, Dr. Munoz continued to treat this as a head injury case. He testified that the brain injuries were "nonaccidental" and that his best estimate was that the head injury must have occurred "at most within six hours of this child arriving to Mount Sinai." *See id.* at 1692. Dr. Munoz did not consider that the brain swelling might be secondary to the abdominal injuries and infection. Indeed, it is unclear that he was aware of the lab reports or autopsy findings.

c. Discharge diagnoses. After the neurosurgery, Steven failed repeated brain-death exams, and life support was removed at approximately noon on February 11, 66 hours after hospital admission. The discharge diagnosis states that the evidence of a "large" subdural hemorrhage and intraparenchymal hemorrhage with diffuse brain swelling, bilateral retinal hemorrhage, extensive cutaneous injuries and intra-abdominal injuries are "collectively diagnostic" of child abuse, and that the mother and boyfriend were being detained by DuPage County. (Ex. 26, Rush Medical Center Medical Records at 60-62.) The medical diagnosis was, however, quite different. This diagnosis was of SIRS shock (systematic inflammatory response syndrome) with multiorgan dysfunction involving the central nervous system, lungs (pulmonary), gastrointestinal, and cardiovascular systems. (*Id.* at 62.) Unlike "child abuse," this is a nonspecific diagnosis that may be caused by infection and inflammation from any source.

5. Explanation For Lines and Marks Found on Steven's Body.

Of all the medical findings, the ones that caused the most confusion were the lines and marks that appeared on Steven's body throughout his hospitalization.

Although Steven had no noticeable signs of trauma on admission to Mount Sinai, these reddish and brownish marks and lines on his body began to appear at Mount Sinai and continued to appear at Rush. These marks and lines are carefully recorded in the hospital records and autopsy reports and were used by the State at trial to confirm a beating "with nearly 50 blows." *See* RA at 2142. At trial, the doctors did not address the causation of the marks in detail. Although the hospital notes describe one of the marks as the Cullen's sign, which is a well-known sign of pancreatitis, there seemed to be a general presumption that these were caused by abuse.

What is clear, however, is that despite the number of marks that appeared during the hospitalization, Steven did not look like a bruised or battered child. Indeed, neither Dr. Green nor Dr. Severin initially saw signs of abuse. *See* RA at 1043, 1374. Karen Clark, Kenyatta's mother, testified that, at Mount Sinai, she observed Steven's naked body and saw no bruising on his thighs. *See id.* at 999-1000. The only external abnormality that Mrs. Clark saw at Mount Sinai was a swollen testicle. *Id.* at 1001.

Mount Sinai nurse Letitia Beasley agreed that the marks on the thigh were "not visible" at Mount Sinai. *Id.* at 1168. At Rush, however, several hours after surgery, Mrs. Clark saw "whip marks, red lines on his thighs, his foot, his ankle, neck, across his stomach" and marks on his back. *Id.* at 1000, 1005. These marks continued to appear throughout the following days.

At trial, these marks and lines were interpreted as signs of a beating. However, as discussed below, a renewed assessment of Steven's medical records by a panel of medical experts demonstrates that virtually all of these marks and lines reflected pancreatitis and disseminated intravascular coagulation (DIC), both of which were confirmed by the laboratory tests. The only exceptions are the marks on the back, which may be related to abdominal injuries and infection that occurred some days before admission.

6. Autopsy.

Despite lab reports confirming that Steven had major abdominal injuries and/or infection and a surgery report confirming that he did not have a large subdural hemorrhage (or any subdural hemorrhage at all), the investigative report provided to the Medical Examiner indicated that Steven was diagnosed with head trauma and that he had a "massive" subdural hemorrhage that had been evacuated on February 8. The report indicates that Steven had various marks on his body but does not mention that he also had pancreatitis and peritonitis. The medical history indicated that he had a runny nose for the past few weeks, but no other cold symptoms. The autopsy was conducted by Dr. Darinka Mileusnic, current Chief Medical Examiner for Knox and Anderson Counties, Tennessee. (Exhibit 3, Darninka Mileusnic-Polchan Aff at ¶ 3.)

At autopsy, Dr. Mileusnic found that Steven had extensive abdominal injuries and infection, including 7 inches of necrotic, or dead, bowel with a small (.1 inch) perforation, peritonitis, peripancreatitis, pancreatitis, a healing subcapsular

hematoma on the liver, and a thin layer of hemorrhage tracking down into the pelvis and scrotum. (Ex. 22, Postmortem Report.) The brain was swollen and necrotic, with extensive evidence of surgery on the right and a forehead contusion on the left with an underlying hemorrhage. There were multiple marks and lines on the body but no fractures or soft tissue swelling. The cause of death was listed as "multiple injuries due to blunt force trauma which was a consequence of child abuse." (*Id.*) The manner of death was homicide. (*Id.* at 10.)

Dr. Teas, the defense's trial expert, generally agreed with Dr. Mileusnic's conclusions but thought that some of the findings might be a little older than five days. Dr. Teas was a former Cook County medical examiner and Chair of the Aurora County Child Death Review Team, which covered DuPage County. Prior to trial , Dr. Teas pointed out the timing, which seemed to exclude Mr. Liebich, to Mr. Ruggiero, the prosecutor. (Ex. 4, Dr. Teas Aff. at ¶ 12.)

C. Summary of Facts and Medical Findings

While set forth in considerably more detail in the accompanying affidavits, and as discussed below, the above narrative establishes the following critical facts:

1. Steven was less active and eating poorly in the days before his death, consistent with abdominal injury/infection.
2. On arrival at Mount Sinai, Steven's body displayed no signs of trauma.
3. The Mount Sinai lab reports confirmed that Steven had pancreatitis.
4. The autopsy confirmed that Steven was suffering from abdominal injury and infection for days before Randy cared for him.

5. The Mount Sinai CT scan was misread as confirming a large subdural hemorrhage caused by head trauma.

6. With the possible exception of the marks on the child's back, the marks and lines that appeared on Steven's body during his hospitalization reflected pancreatitis and disseminated intravascular coagulation rather than trauma.

7. There is no evidence of head trauma and no evidence of any traumatic events occurring on the day of hospital admission.

8. Steven died from abdominal injuries/infection occurring before he was left with Randy, culminating in pancreatitis and a hypoxic-ischemic brain.

In combination, these facts confirm that Randy did not cause – and could not have caused –Steven's death.

D. The Investigation of Randy Liebich as the Sole Suspect in Steven's Death

Despite Kenyatta's history, the abdominal injuries/infection, and the broad range of medical timing, which included the period 24-48 hours before surgery, Randy was the only suspect police every truly investigated.

1. Mt. Sinai.

The accusations against Randy began at Mount Sinai when Dr. Boykin accused Randy of "sitting at home beating all day. RA at 1115. At 8:05 p.m., Det. Figiel was told that Steven was in critical condition at Mt. Sinai with trauma to the head, scrotum and bruises throughout the body, and that the doctors "could not tell

if the injuries were recent." (Ex. 27, DuPage Co. Sheriff's Police Reports at MR 11.) Randy had already told the Chicago police officers about the hot dog incident. (*Id.*) The Rush transport notes indicate that the history was limited because the parent(s) were being questioned by the police. (Ex. 25, Rush Medical Center Transport Notes at MED 46.)

2. Initial investigation.

When Steven was in surgery, Det. Figiel and Criminal Investigator Vrbos met with Dr. Severin and Tammy Smith, R.N. (Ex. 27, DuPage Co. Sheriff's Police Reports at MR 11.) Dr. Severin said Steven had a bleed in the brain on the right, internal abdominal injury, bruising around the head, and marks on his back and inner legs. The bruising looked relatively recent and Dr. Severin estimated that it occurred "sometime between 24 to 48 hours," though this was a guess. (*Id.* at MR 12.)

3. Interrogation at Rush, 2/8-2/9.

Figiel and Vrbos began interrogating Randy at about 11:20 p.m. (Ex. 11, Randy Liebich Supp. Aff. at ¶ 2.) In his affidavit, Randy states that he was placed between Figiel and Vrbos and pushed back into his chair when he tried to leave. (*Id.* at ¶ 3.) Randy answered their questions and told them about the events of the day over and over, including the hot dog incident. (Ex. 10, Randy Liebich Aff. at ¶ 2.) Even official police reports reflect that Randy said that "Steven hadn't been eating, he wasn't saying much and he was not as active as he usually was . . . he still moved

around and played but not as much." (Ex. 27, DuPage Co. Sheriff's Police Reports at MR 13-14.)

After about an hour, Figiel and Vrbos told Randy that he was not to leave the conference room and a police officer was placed outside the door to ensure he could not leave when they left the room. (Ex. 11, Randy Liebich Supp. Aff. at ¶ 4.) When Figiel and Vrbos went to Steven's room, Tammy Smith told them that Steven had been declared brain dead and was on life support. (Ex. 27, DuPage Co. Sheriff's Police Reports at MR 14.) At about 1:05 a.m., Figiel and Vrbos gave Randy a paper listing his rights. (Ex. 11, Randy Liebich Supp. Aff. at ¶ 5.) Randy objected to signing the paper but Figiel told him that signing the paper just meant he had read it. (*Id.*) Vrbos told him to sign the paper and tell them what happened to Steven or he would ensure that Child Services would take his daughter and he would never see her again. (*Id.*) Randy signed the form. (Ex. 27, DuPage Co. Sheriff's Police Reports at MR 217.)

In the continued interrogation, Randy's story was the same with one exception: when he was told that the bruises on Steven's head were inconsistent with what he was telling them, Randy said that Steven fell and hit his head when he choked, but it wasn't hard. (*Id.* at MR 15.) When Randy said he no longer wanted to answer questions and tried to leave, Figiel said he was not going anywhere near Steven. (*Id.* at MR 15; *see also* Ex. 11, Randy Liebich Supp. Aff. at ¶ 6.) Randy's requests to use the phone to ask his family to contact an attorney were denied. (Ex. 11, Randy Liebich Supp. Aff. at ¶ 7.)

At about 2:15 a.m., Lt. Szalinski and Sgt Kuntz took Randy to the emergency room and allowed him to hold Angelique briefly. Szalinski told him to take a good look at her because it would be the last time he would see or hold her unless he gave a better explanation of what happened to Steven. (*Id.* at ¶ 8.) Sgt. Kuntz then took Randy outside for a cigarette and questioned him further. (*Id.* at ¶ 9.) After that, Randy was placed in an isolated room and prevented from leaving. (*Id.* at ¶ 10.)

About an hour later, Figiel and Vbros took Randy to Steven's room. (*Id.* at ¶ 11.) They told him that Steven was clinically brain dead and "questioned him as to why this happened." They also told him "that his story on the events that occurred Friday were inconsistent with the severe injuries sustained by Steven." (Ex. 27, DuPage County Sheriff's Office Police Reports at MR 15.) According to Randy, Vrbos placed his hand on the back of Randy's neck, squeezing hard, and asked repeatedly, in an accusing manner, "what did you do?" "[w]hy did you do this?" and "[y]ou had better give us some answers." (Ex. 11, Randy Liebich Supp. Aff. at ¶ 11.) Randy told Vrbos and Figiel that he was tired of being intimidated and demanded a phone call to contact an attorney, but his request was denied. (*Id.*)

4. DuPage Interrogation, 2/9.

At about 7 a.m., Figiel and Vrbos handcuffed Randy and took him to the DuPage County Sheriff's office. (*Id.* at ¶ 12.) At about 8:40 a.m., Randy threw up in a wastebasket. (*Id.* at ¶ 13.) He was questioned by different detectives for about three hours. (*Id.* at ¶ 14.) He told them repeatedly that he didn't do anything,

needed to get his medication, and didn't want to answer any more questions, but was not allowed to leave. (*Id.*)

At about 12:15 pm, Officer Richard O'Brien, a polygraph examiner, also questioned Randy. (*Id.*) Randy signed a Miranda form for this purpose. (Ex. 27, DuPage Co. Sheriff's Office Police Reports at MR 223.) According to O'Brien's February 12 report, Randy again described the hot dog incident. He also described Kenyatta "popping" Steven in the head a few times and hitting him with a belt on the evening of February 7. (*Id.* at MR 212-13.) Ultimately, Randy told O'Brien and the detective that he was sick, didn't want to answer any more questions, and didn't want to take any test under these circumstances. (*Id.* at 16; Ex. 11, Randy Liebich Supp. Aff. at ¶ 15.)

Randy also told Lt. Szalinski that he didn't have anything to say and that he had already asked for a lawyer. Szalinski said that the longer it took Randy to tell them what they wanted to know, the longer he would suffer. (Ex. 11, Randy Liebich Supp. Aff. at ¶ 16.) According to Szalinski, Randy described Kenyatta popping Steven on the head on 2/7 and illustrated the "pops" as similar to an open hand slap but with the impact coming from the palm of the hand. (Ex. 27, DuPage Co. Sheriff's Office Police Reports at MR 48.) According to Szalinski, Bradford told Randy that there was no doubt that the injuries to Steven occurred when Randy was caring for him and that Randy needed to explain how they occurred. (*Id.* at 50.)

By then, Randy had been questioned for over 15 hours. (Ex. 11, Randy Liebich Supp. Aff. at ¶ 17.) When he was finally given a phone, he called his mother

and stepfather, and asked them to come to the Sheriff's Office and refused to answer any more questions. (*Id.* at ¶ 18.) O'Brien and Kenyatta then came to the interrogation room, and Kenyatta accused Randy of hurting Steven. Randy responded that he hadn't harmed Steven in any way. After a few "heated exchanges," the officers removed Kenyatta, and Szalinski and Bradford continued the interrogation. (*Id.* at ¶ 20.)

At 4:40 p.m., the State's Attorney Office decided that no criminal charges would be filed against Randy or Kenyatta at that time. (Ex. 27, DuPage Co. Sheriff's Office Police Reports at MR 16.) Randy was released after 17½ hours of detention. (Ex. 11, Randy Liebich Supp. Aff. at ¶ 20.) Randy later learned that his mother and stepfather had gone to the sheriff's office soon after he called, but were told that he had been charged with first degree murder and would not be going anywhere. (*Id.* at ¶ 21.)

5. February 12.

According to the police reports, Randy called Fiegel at 8:15 a.m. on February 12 and left a message saying he heard Figiel wanted to speak with him and that he would call back. (Ex. 27, DuPage Co. Sheriff's Office Police Reports at MR 71.) At 3 p.m., Figiel called Randy's sister, Denise, who said she would get a message to Randy that Figiel wanted to speak to him. (*Id.*)

6. February 13 interrogation.

According to the police reports, when Randy called Figiel at 9:30 a.m., he indicated he did not mind speaking in person. (Ex. 27, DuPage Co. Sheriff's Office

Police Reports at MR 71.) Figiel, Bradford and an Assistant State's Attorney came to Randy's parents' home. (Ex. 11, Randy Liebich Supp. Aff. at ¶ 33.) There is some disagreement over whether Randy accompanied them voluntarily. (Compare *Id.* at ¶ 22 with Ex. 27, DuPage Co. Sheriff's Office Police Reports at MR 72.) In any event, Randy was taken to the police department in a police car. (Ex. 11, Randy Liebich Supp. Aff. at ¶ 23.) At 11:20, Randy signed an interrogation waiver. (Ex. 27, DuPage Co. Sheriff's Office Police Reports at MR 72, 224.) The detectives told Randy of the medical evidence against him and said they knew he was responsible; Randy continued to insist that he hadn't hurt Steven. (*Id.* at MR. 72.) This was discussed "in a very repetitive manner" for 2 ½ hours. (*Id.*)

7. February 14 polygraph discussions.

After continued discussions on taking a polygraph, Randy called Figiel at 2:19 p.m. and told him that an attorney, Dennis Born, had advised him not to take the polygraph. (*Id.* at 73; Ex. 11, Randy Liebich Supp. Aff. at ¶ 27.)

8. February 14 interrogation (Robert Liebich).

Shortly after the polygraph discussions ended, Randy's cousin Dion Liebich told Randy that a cousin, Robert Liebich, a police officer, was looking for him because he was reportedly "on the run" in a murder investigation. (Ex. 11, Randy Liebich Supp. Aff. at ¶ 24.) Dion convinced Randy to go to the Roselle Police Department to resolve this erroneous information. (*Id.*) At the station, Randy told Robert that he was not on the run and didn't know why Robert wanted to see him.

(*Id.* at ¶ 26.) Although Robert is Randy's cousin, they had only met on two previous occasions. (*Id.*)

Once Dion and Randy arrived at the police station, Robert took them into an interrogation room and proceeded to interrogate Randy. According to Dion, Robert was angry about the possibility that Randy could have let something happen to a child (Ex. 12, Dion Liebich Aff. at ¶ 5) and he questioned Randy aggressively. (*Id.; see also* Ex. 10, Randy Liebich Aff. at ¶ 26.) Robert asked detailed questions about the hot dog incident and insisted that Randy reenact what he did when Steven bit down on his finger. Randy showed him light slaps on the cheek, just to get Steven to open his mouth. (Sex. 10, Randy Liebich Aff. at ¶ 26; Ex. 12, Dion Liebich Aff. at ¶¶ 6-7.) Robert became frustrated when Randy gave consistent answers to his questions. Since he believed that Randy was lying, he kept pressing the issue. (Ex. 12, Dion Liebich Aff. at ¶¶ 12, 14.) Even so, Randy's account did not vary.

Robert asked if Randy had told the police about slapping Steven to get him to let go of his finger. (Ex. 10, Randy Liebich Aff. at ¶ 27.) Randy didn't think he had because the police weren't interested in the hot dog incident. (*Id.*) Dion's account of the same interrogation is set forth in his affidavit (described below). Notably, all three people in the room, agree that what Randy demonstrated would not have caused Steven any harm. This includes Robert, since, after Randy demonstrated the hit, Robert testified that he still didn't believe that had not "hit Steven hard enough to do this." RA at 1498.

9. February 27.

On February 27, Robert provided a handwritten report on his interview with Randy. (Ex. 28, Robert Leibich Report.) According to his report, the last time Randy looked at the floor and Robert was uncomfortable believing his answer, so he asked him to swear on his father's grave. Randy looked him straight in the eye and said he didn't hit the kid that hard. Robert said that he and Dion assumed Randy had been arrested, and that he called Fiegel to tell him of the conversation when he learned that Randyh had not been arrested. (Ex. 27, DuPage County Sheriff's Police Reports at MR 103-104.)

10. Robert Liebich's trial testimony.

At trial, Robert testified that on February 13 at about 6 p.m. he had seen a copy of a state-wide LEADS message saying that Randy was "missing and suicidal." RA at 1482. The remainder of his testimony was the same as his earlier statements though a little more specific. For example, Robert testified that Randy told him that on February 7 he had seen Kenyatta hit Steven in the head four times, using her right hand to hit Steven on the left side of the head. *See id.* at 1486-87. Robert said that, in the hot dog incident, Randy "smacked [Steven] in the right side of his head with his left hand, an open hand, twice to get him to let go of the finger." *Id.* at 1488. Randy said that he didn't tell the detectives about the bite or about striking Steven. Robert told Randy that if he was lying, "the police were going to come for him." *Id* at 1490.

On cross, Robert agreed that Randy looked him "right in the eye" and said he did not hit the kid that hard after Robert asked him to swear on his father's grave.

Id. at 1497. On redirect, Robert testified that when he asked Randy several times if he had hit Steven hard enough to do this, Randy was often slumped forward with his eyes on the floor "and I didn't believe him." *Id.* at 1498. The Judge overruled Mr. Holman's objection to Robert's belief on the ground that "it's what he said to the defendant at the time. His belief is not the issue." *Id.* This testimony proved to be extremely important at trial as it was viewed as an admission by Randy that he had struck Steven. *See id* at 24 (The Court stating, "But what I found to be telling and I still can see Robert Liebich sitting in that chair testifying, is that the defendant looked me straight in the eye and he said, 'i didn't hit the kid that hard.' To me that speaks volumes; that is the defendant's admission that he hit Steven Quinn.").

11. February 28.

Randy was arrested on first degree murder charges at 11:55 p.m. on February 28. (Ex. 27, DuPage Co. Sheriff's Police Reports at MR 823.)

12. March 1.

When Randy refused to sign a *Miranda* waiver form at approximately 1 a.m., the police the detectives "explained that the evidence gathered in this case showed he was involved in the death of Steven." (*Id.*) Randy was told to think about what he was told and to knock on the door when he was ready to speak to them again. At 2:10 a.m., Randy said he had thought about it and wanted an attorney. (*Id.*)

13. Autopsy findings.

Randy was arrested on the same day that the medical examiner signed the autopsy report, which identified the cause of death as multiple blunt force injuries

due to child abuse and the manner of death as homicide. At that point, however, there was no information on timing. On information and belief, Dr. Mileusnic received the slides of the organs (excluding the brain) on February 25 and returned them on March 6 She received the tissues of the brain on March 7 and received the slides on March 14, returning them on March 29. These dates confirm that Dr. Mileusnic could not have provided the pathology information on timing until sometime in March, probably towards the end of the month. As noted, Dr. Mileusnic found that the key injuries were subacute and/or approximately 5 days old at the time of death, placing them well before the day that Randy cared for Steven. (Ex. 22, Postmortem Report.)

14. May 14, 2002.

On May 14, 2002, Dr. Munoz wrote to Mr. Reidy, DuPage County State's Attorney, in response to an update from Mr. Reidy. Dr. Munoz confirmed that it was his opinion that Steven's injuries occurred within six hours of admission to Mt. Sinai and that the injuries could not have occurred before that if the child was talking and eating/drinking just prior to his mother's departure.

15. Autopsy timing.

There is no indication in the materials we have reviewed indicating when the state or Dr. Munoz became aware of the pathology findings. Dr. Teas makes clear in her affidavit, however, that she told prosecutors of these findings well before the trial. (Ex. 4, Dr. Teas Aff. at ¶ 12.)

E. Facts as Adduced at Trial

For purposes of this filing, the Petitioner adopts the recitation of facts as set forth in the appellate court's Rule 23 Order filed December 12, 2007, as an accurate representation of the facts as they were adduced at trial. This order is attached as Exhibit 20.

In its Order, the Court summarized the facts set forth above, emphasizing the "the severity of the head injury" Steven had suffered and describing the marks and discolorations that appeared after hospital admission, including a "swollen and red testicle" and marks "like whip marks, red lines on his thighs, his foot, his ankle, neck, across his stomach **** on his back and like pressure marks on his neck." Order at 2-3. Since Randy did not testify, the Court accepted Kenyatta's testimony as given (*Id.* at 7) but noted that Kenyatta's mother had told a police officer that Kenyatta was an "excellent liar." *Id.* at 3. The Court noted that Kenyatta's mother and aunt, who were Steven's primary caretakers, had not noted any prior signs of abuse and that Kenyatta agreed that she had never seen Randy strike Steven. *Id.* at 2-3, 7. Randy's boss testified that Steven looked sick when Randy brought him in on February 8 and that he did not see any marks on him. *Id.* at 7.

The Court summarized the testimony of the Mt. Sinai doctors and nurse (Dr. Green, Dr. Boykin and Letitia Beasley) and the Rush transport nurse (Tammy Smith). This testimony includes extensive discussions of the CT scan, which Dr. Boykin was told was "really bad" and showed a head that was "full of blood," and of

the bruises and marks that appeared on Steven's body throughout his hospital stay. *See, e.g., Id.* at 8-10, 12.

Det. Figiel described his interrogations of Randy, stating that as he and Randy repeatedly went over Randy's story, the substance of the story remained, for the most part, consistent. *Id.* at 15. Filipiak, another detective, testified that Randy was initially "aloof and nonchalant" but became "very scared" as it became clear that Steven's injuries were serious. Filipiak did not know how much information Randy had about Steven's condition when he appeared "nonchalant and aloof." *Id.* at 15-16.

The court placed the greatest emphasis on the testimony of Dr. Severin and Dr. Teas. Dr. Severin described Steven's physical condition in detail. He testified that the bruises and marks on Steven's body appeared to be of approximately the same age and could have occurred within the last 48 hours; however, bruising is difficult to time. *Id.* at 16-17. Dr. Severin did not think that abdominal injuries were likely on initial examination; however, his enzymes were elevated and by the following day he had the "Cullen's sign," a bruise-like appearance around the belly button that is an indication of hemorrhagic pancreatitis. *Id.* at 17. Dr. Severin testified that the injuries resulted from nonaccidental external trauma occurring within 4-6 hours before arrival at Mount Sinai. *Id.* He further testified that the timing shown on the pathology slides would not change his opinion. *Id.*

The court summarized Robert Liebich's testimony, including Robert's statement that Randy told Robert that Kenyatta hit Steven in the head four times

the night before he collapsed and that Steven lay in his bed awake the next morning, instead of getting right up, as he would usually do. The court also summarized Robert's description of the "hot dog incident," including his testimony during cross-examination that Randy swore "on his father's grave" that he did not hit Steven that hard when he "smacked" him to get him to let go of his finger. *Id.* at 20.

The court summarized Dr. Mileusnic's testimony on timing, in which she testified that Steven's head injuries were inflicted approximately five days, plus or minus a day, from his time of death. She believed that the injuries could have occurred on the 8th but could have easily occurred before that, even as early as the 5th, since abdominal injuries are often slow to manifest. *Id.* at 24. In this case, Dr. Mileusnic saw mononuclear cell fibrin in the abdominal injuries that typically occur "five to seven days after an injury." *Id.* at 25.

The Court also summarized Dr. Munoz's testimony. Dr. Munoz testified that when Steven was transferred to Rush, he was "neurologically very sick" and that a CT scan had revealed "a lot of blood in his head." Dr. Munoz found that the CT findings spoke "of a trauma to the whole brain." Dr. Munoz testified that at surgery, the brain started to herniate (come out of the opening of the skull) and that the blood that he saw was "bright red," indicating that it constituted a fresh clot. Dr. Munoz characterized the amount of blood as "massive." *Id.* at 25-26. Dr. Munoz opined that Steven's injuries were not accidental and that they occurred no more than six hours before Steven arrived at Mount Sinai since he could not have walked,

talked, eaten or drank anything after sustaining these injuries. *Id.* at 27. Munoz acknowledged that he had only been board certified for nine months and that this was the first criminal case in which he had testified. He agreed that history was important in timing an injury but that the history he considered was that Steven was "doing okay" before Kenyatta left for work and that he was not when she returned to work. Dr. Munoz further testified that he had contacted DCFS "hundreds" of times regarding suspected child abuse and that on each occasion DCFS asked him for his opinion on timing. *Id.* at 28.

The Court also summarized the testimony of Denise Foster, Frank Belpedio and Crystal Zeis, all of whom testified that they had seen Kenyatta hit or throw Steven. *Id.* at 29-32. Kenyatta's mother agreed that she had told a detective that "Kenyatta slapped Steven in the face a few times in the past due to his whining and crying" but later clarified that she did not actually see Kenyatta do this. *Id.* at 30-31.

Finally, the Court summarized the testimony of Dr. Shaku Teas, a forensic pathologist that the court recognized as an expert in the area of forensic pathology and child abuse. Dr. Teas had previously testified for the state "[p]robably hundreds of times" and on behalf of defendants only about 20 times. *Id.* at 32. Dr. Teas opined that Steven's injuries were about five days old. They could have been six days old and it was possible that they were only four days old, but due to the amount of healing, they could not have been less than four days old. *Id.* at 34. Dr. Teas stated that it was more likely that Steven's injuries were seven days old as opposed to

three days old. *Id.* Dr. Teas also noted that the red lines on Steven's foot could have been caused by tubes from a blood pressure cuff on Steven's leg. *Id.* at 35.

F. Facts Relating to the Performance of Counsel

1. Advice Regarding Randy's Right to Testify.

During the course of the trial, Randy told attorney Ricky Holman that he wished to testify. (*See* Ex. 10, Randy Liebich Aff. at ¶ 41.) Randy explained that he wanted to clear up some information that was incorrect or incomplete, such as when his cousin, Robert, failed to mention that Randy had demonstrated the force with which he had hit Steven on the occasion when Steven had bitten down on Randy's finger, and Robert had commented that the hit was not that hard. (*See id.*) Randy also wanted to let the court know that, contrary to Kenyatta's testimony, he had not smoked PCP the night before Steven's hospitalization. (*See id.* at ¶ 42.) Randy also wanted to correct statements made by the police, such as that Randy said that Steven could not feel Kenyatta hitting him through his diaper. Randy never said that; Kenyatta did. (*See id.* at ¶ 43.)

Randy told Mr. Holman that he wanted to testify about these things, but Mr. Holman said that he would not let Randy testify. (*See id.* at ¶ 45.) Randy did not insist, because he did not know that the decision of whether or not to testify was his to make. In fact, Mr. Holman does not remember ever advising Randy of this right, and Mr. Casey remembers that he did not advise Randy of this right. (*See* Exs. 18 and 19, Ricky Holman Aff. at ¶ 3, and John Casey Aff. at ¶ 3.) Also, nowhere in the record does it appear that the Court informed Randy of his right to testify.

2. Performance of Counsel at Trial.

During the course of their investigation, defense counsel learned of the existence of witnesses who might have been favorable to Randy's defense. For example, Robert Liebich's handwritten report states that Dion Liebich was present when Randy explained how he hit Steven to get him to release his bite. (*See* Ex. 28, Robert Liebich Report). Despite this, trial counsel never contacted Dion. (*See* Ex. 12, Dion Liebich Aff. at ¶ 22.) Had they done so, they would have learned that the hit that Randy demonstrated, was really "a tapping from a few inches away that should not have have hurt anyone." (*Id.* at ¶ 7.) Since Dion was not called at trial, the court never heard this information.

In addition, both Denise Foster and Marlene Szafranski had knowledge relating to Kenyatta's violent nature. In particular, they were aware of an occasion when Kenyatta pushed Linda, Randy's mother, and knocked her unconscious (Ex. 15, 3/3/12 Foster Aff. at ¶¶ 3 & 4; Ex. 16, Szafranski Aff. at ¶ 11.) Not only did defense counsel fail to question Denise and Marlene about this incident, they did not call Linda, who is now deceased.

3. Pre-trial Motions to Suppress.

No motion to suppress Randy's statement to Robert Liebich was filed in this case. On June 9, 2004, Mr. Holman stated that he chose not to file any such motion for strategic reasons. *See* RA at 905 (Mr. Holman stating "I have reviewed all of the discovery and all of the conversations and it is in my view that, and speaking with Mr. Liebich, and Mr. Liebich and I have talked about suppressing statements; and

it is our view, the defense view, that no Motion should be filed in this particular matter"). Randy's appellate counsel did not raise the issue of whether Mr. Holman was ineffective for failing to file such a motion before the appellate court.

III. PROCEDURAL HISTORY

A. Trial

Following a bench trial, Randy was convicted of the First Degree Murder of Steven Quinn on July 16, 2004.

B. Verdict

The trial court found that the issues to be decided were what happened to Steven between February 2 and February 8 and, more specifically, "who caused the [blunt trauma] injuries, and when did they occur." RA at 5. The court stated that the defense was claiming that Kenyatta had beaten Steven so often and so severely that he was "mortally wounded" early in the week of February 2, perhaps as early as February 4-6, followed by a "lucid interval" that lapsed within an hour or so of Kenyatta returning home at approximately 4:30 P.M. on February 8. *Id.* at 6. The court found that the primary support for this theory was presented through Frank Belpedio, Crystal Zeis and Ruben Martinez, who testified that Kenyatta struck Steven on various occasions from various points and vantages, including grabbing him by the arm, throwing across the room, and pushing him off a couch. *Id.* The court found that these witnesses were not credible based on their manner of testimony and motive to fabricate, i.e., their relationship with Randy and disdain for Kenyatta. *Id.* at 9. The court also found that Kenyatta's mother, Karen Clark,

and aunts, Sadie Brown and Dorothy Herron, were "extremely credible" and that their testimony that they had never seen an injury on Steven when he returned from being with his mother was compelling. *Id.* Based on this testimony, the Court stated that, while Kenyatta was not an ideal mother, there was no credible evidence that she abused Steven, chronically or at all. This testimony was, however, equally applicable to Randy, who lived with Kenyatta and also cared for Steven for most of his life.

With regard to the medical evidence, the court found that, based on the pathology, Dr. Teas offered at best a "great guess" that the injuries occurred anywhere between February 4 and February 8. In fact, this finding was contrary to Dr. Teas' testimony, in which she made clear that the injuries preceded February 8. *See* RA at 1983 (When asked whether the injuries took place on February 8, Dr. Teas responding "[t]hey probably could have taken place earlier. If I was going to go to the narrowest part, but there was a lot of healing, I would actually be more inclined to say it went beyond the 5th or the 6th, then to go closer, because there was a lot of healing"). The court did not mention the findings of Dr. Mileusnic, the Cook County Medical Examiner who conducted the autopsy and timed the injuries to a period before Randy cared for the child.

Although the court did not address this issue, the court likely had difficulty grappling with the notion that Steven was severely beaten on February 2-6, causing bruises and whip marks that did not appear for several days. What the court did not know – since it was not presented – was that most of these marks were signs of

pancreatitis and a coagulopathy (specifically, disseminated intravascular coagulation, or "DIC"), both of which were confirmed by laboratory testing.

The court's ruling also did not address the fact that internal injuries or infections can have "lucid intervals," or periods of relative normality. In most injuries and natural disease processes, however, "lucid intervals" are the rule, not the exception. *See id.* at 1127 (Dr. Boykin stating, "[b]ut generally the person will lose consciousness at the scene of an accident. It's actually quite classic, and they'll have what's called a lucid interval, and within an hour they'll usually go on, and a lot of those patients will actually then go on to die").

Without any explanation of how lucid intervals impacted Steven's symptoms, the Court adopted the state's theory that Randy had beaten Steven about the head and abdomen on February 8, knowing that these acts would cause a strong probability of great bodily harm and death. The court made the following findings in support of its conclusion:

1) The child's posturing and deviation of the eyes to the left shortly after arrival at Mt. Sinai indicated a significant brain injury.
2) The brain injury was a whole brain injury.
3) The pathology findings were consistent with injuries occurring between February 5-9.
4) Various marks and lines appeared at Mt. Sinai or during transport (flat red mark on the right forehead, bruises on the groin, red abdomen, and marks on the legs and thigh), with more marks

appearing at Rush (raised welts on left foot, more defined marks on legs, thighs and abdomen, marks on chin, and Cullen's sign, a diffuse red mark around the belly button that indicates severe injury to the pancreas).

5) Dr. Munoz saw "acute, fresh, red" blood during surgery and was "absolutely steadfast" in his opinion that the head injuries occurred within 4-6 hours of hospital admission.

6) Dr. Severin characterized the injuries as recent.

7) The injuries were the equivalent of falling from 20-30 feet or blunt trauma with a bat, brick, foot or fist to the abdomen, and would have precluded eating, walking, talking or playing if sustained before February 8.

8) There is no lucid interval or delay in symptoms with abdominal injuries.

9) Randy was calm at Mt. Sinai (variously described as respectful, aloof and nonchalant by the medical staff) and unresponsive to Dr. Boykin's accusations, becoming concerned and "scared" only when the serious nature of Steven's injuries was communicated to him.

10) Steven's abdomen was not distended at Rush, indicating that abdominal injuries or infection were unlikely.

11) The Mount Sinai labs showed liver and pancreatic injury, and the doubling of the levels of amylase and lipase between Mt. Sinai and Rush indicated extremely recent injury.

12) A McDonald's employee from whom Randy borrowed a cigarette said that Randy "appeared to be somebody that I would party with" and that he could have been under the influence of drugs, then or previously.

13) When questioned by Robert Liebich (Randy's cousin and a police officer), Randy described slapping Steven on the side of the head during the choking episode, stating that he didn't hit Steven that hard.

14) The injuries to the pancreas, liver and bowel were in a straight line, indicating the force that had to be used.

15) The Cullen's sign appeared on February 9, indicating that the injury occurred very shortly before arrival at Mt. Sinai.

16) Since Randy failed to counter the information provided by Kenyatta and others on his attitude and/or statements, the Court accepted them as fact.

C. Dr. Teas' letter

On August 26, 2004, Dr. Teas, dismayed at learning that there had been a guilty finding, wrote to Judge Jorgenson pointing out that the pathology slides showed that Steven's injuries could not have occurred on February 8 or even February 7. (Ex. 21, Letter from Dr. Teas to Judge Jorgenson.) She noted that it

was unlikely that Steven was well on February 8 since acetaminophen and aspirin were found in his blood, and he had lost more than 4 pounds since his November 2001 checkup. She noted that the initial lack of abdominal findings on admission to Rush was attributable to the sedating and paralaytic drugs that Steven had received and that signs and symptoms of abdominal injuries in children can be delayed for as much as 2-3 days. Finally, Dr. Teas noted that the "large" subdural hemorrhage noted in the hospital records was not present at surgery. *Id.*

In addressing the letter from Dr. Teas (misspelled Dr. Zeis), Counsel for Mr. Liebich joined counsel for the State in arguing that Dr. Teas' letter was an improper *ex parte* communication and should not be considered. The court agreed that she would not consider the letter and that it would not go into her file. In its Rule 23 Order, the appellate court determined that the letter did not necessarily alter the trial court's determinations regarding the timing of the injuries.

D. Motion for a New Trial

In a hearing on a motion for new trial based on ineffective assistance of counsel ("IAC"), the court declined to appoint new counsel for Mr. Liebich and further found that there was evidence beyond a reasonable doubt of Randy's guilt.

E. Sentencing Hearing

In a victim impact statement, Kenyatta said that she and Randy had spent two years planning a life with Steven, and that they had finally gotten their lives together. When she learned at the hospital that Steven had been "beaten all over his body by someone I loved, trusted and planned to marry and share the rest of my

life with," she could not imagine that someone she loved and trusted could do such a terrible and violent thing to her son. As this suggests -- and as their earlier statements confirmed -- Kenyatta and her family had no reason to believe -- and could not imagine – that Randy would ever harm Steven.

Numerous relatives and family friends testified that Randy had always been very good with children, including Steven, Angelique, various nephews, a younger brother and children of other family friends. He was a regular babysitter for many of these children, including neighborhood children, including overnight stays. Those who had seen Randy with Steven uniformly described them as having an excellent relationship, and the social services caseworker testified that Randy was equally good with Angelique.

In sentencing Randy to 65 years imprisonment, the court emphasized Robert Liebich's description of the choking incident and Randy's prior drug use. A motion to reconsider the sentence was denied.

F. Randy's *Pro Se* Post-Trial Motions Raising Ineffective Assistance of Counsel

Following his conviction. Mr. Liebich made an oral *pro se* motion claiming ineffective assistance of counsel ("IAC"). This motion was summarily rejected. On November 4, 2004, Mr. Liebich filed a second *pro se* IAC motion that fell into several categories, summarized by the Court during the hearing to include:

Failure to prepare witnesses and present key factual evidence

- failure to prepare defense witnesses prior to trial
- failure to interview or call Dion Liebich, who was present at Randy's interrogation by Robert Liebich

- failure to introduce evidence that February 8 was not the first time Randy had been alone with Steven
- failure to introduce evidence from the polygraph examiner that Kenyatta admitted that she slapped Steven across the face and hit him with a belt and comb
- failure to introduce Kenyatta's diary, which discussed her stepfather beating her younger siblings
- failure to interview or introduce evidence from Kenyatta's coworkers at Carlene Research who observed Kenyatta's conduct towards Steven
- failure to introduce evidence from Steven's biological father that Kenyatta would lose her patience with Steven and that he had seen her at her worst
- failure to insist that the entire videotapes of Crystal Zeis and Frank Belpedio be played, rather than sentences excerpted by the State
- failure to introduce Kenyatta's admission that they had been at Frank Belpedio's house days before hospital admission (Mr. Belpedio was impeached on that issue because he couldn't remember the exact date)
- failure to object to damaging hearsay testimony by the state's witnesses
- failure to object to improper and prejudicial statements made by the prosecutor in closing

Failure to present medical evidence

- failure to introduce evidence of Steven's weight loss between November 2001 and February 2002
- failure to introduce evidence on the Tylenol and aspirin in Steven's system on hospital admission, which confirmed that he was unwell prior to February 8
- failure to introduce evidence that the paralyzing drugs would have prevented Dr. Severin from diagnosing abdominal injuries based on physical examination.
- failure to review slides with Dr. Mileusnic prior to trial

Right to testify

- failure to inform defendant of his constitutional right to testify on his own behalf and refusal to allow the defendant to testify on his own behalf, described by counsel as a strategic choice

Plea bargains

- failure to disclose a plea deal offered by the state before trial

RA at 2414-31.

At the December 7 hearing on Randy's IAC claims and on his attorney's request for reconsideration of the sentence, Mr. Casey, one of Randy's attorneys, testified that Mr. Liebich's facts were correct, but that the decision to introduce, or not introduce, certain witnesses and evidence were strategic decisions made by defense counsel. Mr. Holman testified that:

> ... when Mr. Liebich had addressed ineffective assistance I think before the sentencing, the Court had ruled that when he had indicated there were several people that were not called to testify, that it was trial strategy and that he wanted us to call some people after the state had put on some witnesses, and that I didn't call those witnesses . . . I believe the Court had made a ruling on that, that it would be hard for us to be able to counteract the state's case for bringing in additional witnesses. I can tell you that the witnesses that we did not call, it was because of trial strategy. I have read those witnesses' reports, and there was both good and bad that could have been brought in on those witnesses. And I chose not to bring them in to testify.

RA at 2420.

In response to the court's questions regarding a plea offer, Mr. Holman testified that he received an offer after a finding of guilty, which he passed on to Mr. Liebich with the promise that if he were to take that sentence, he would not file any post-conviction motions whatsoever. He and Mr. Liebich agreed that they were not going to accept that offer. Mr. Holman did not disclose the contents of the plea offer. *See id.* at 2422.

With respect to pre-trial plea offers, Mr. Holman stated that, so far as he knew, it was Mr. Liebich's desire, and Mr. Holman's and Mr. Casey's desire, to go forward without receiving an offer since "we wanted to have a trial on this matter because we were maintaining our absolute innocence in this case." *Id.* When the

prosecuting attorney suggested discussing an offer, Mr. Holman told them that, in exchange for dismissing the charges, Mr. Liebich would testify against Kenyatta. *Id.* Mr. Holman testified that he told Mr. Liebich of the state's overtures and what he had told them on several occasions. The following discussion followed:

> Liebich: . . . Mr. Holman told me that after Mr. Ruggiero got off the phone speaking with Dr. Teas he came forward, he wanted to make a plea offer. He wanted to resolve this. Mr. Holman told me he told him straight out no, we want to go to trial. We don't want to know what an offer is. Facing the kind of time that I'm facing and that I received I would have been willing to take an offer had it been reasonable.
>
> Court: No specific offer was ever made to you that you rejected?
>
> Liebich: Mr. Holman didn't even give him a chance to hear it.
>
> Court: You never made an offer?
>
> State: There was never an offer made in this case prior to trial.
>
> Liebich: Mr. Holman stopped him and told him "I don't want to hear it. We're going to trial. That's all there is to it." At least that's what Mr. Holman told me.
>
> Court: None was ever communicated from the state attorney's office, simply no offer, there was some discussion after the conclusion of the trial?
>
> Liebich: This was before the trial, Judge.
>
> Court: I appreciate that. The only discussion, so the record is crystal clear, the only discussion about a possible disposition came after a finding of guilty?
>
> State: That's correct.

Id. at 2423-24.

With respect to Mr. Liebich testifying, Mr. Holman told the Court that Mr. Liebich's allegation that he told Mr. Holman of his desire to testify "would not be true." *Id* at 2423. Later, the Court followed up as follows:

Court:	What about testifying?
Liebich:	I feel that I should have been able to take the stand to testify because I'm the only person that can tell what happened that day, and rebut any of the things that were said about me by Kenyatta Brown. I felt it was important that I take the stand. Mr. Holman told me not to take the stand.
Court:	All right. Anything else?
Holman:	Not from me, Judge.
Court:	Mr. Casey?
Casey:	No, your Honor.

Id. at 2425.

The court concluded that none of the witnesses would have affected her decision, focusing again on Robert Liebich's testimony. The court again described Robert's statement that Randy said "I didn't hit the kid that hard" as a "critical piece of evidence." *Id* at 2427. The court did not see the relevance of Steven's weight loss, which she felt would depend on whether the same scale was used, or of the fact that Randy and Steven had spent considerable time alone together in the past. *Id* at 2428-29.

In addressing the plea agreement issue, the court found that "there was no offer made by the state period. It's always their prerogative, state is under no obligation to make an offer in any case. For whatever reason they chose to not make

an offer in this case until after a finding of guilty was made by the Court." *Id* at 2430.

The court then addressed the issue regarding Mr. Liebich's ability to testify at trial as follows:

> [The defendant] says now he should have been able to testify. He should have testified to tell his side of the story, but now it appears in retrospect that he says his attorney told him not to testify, which is totally contrary to what was represented by Mr. Holman, who was the lead counsel at the time that he never told the defendant not, that he should not testify. And I have to say that I don't have an independent recollection of it, but generally I make an inquiry, the Court itself makes an inquiry to determine whether or not the defendant wishes to testify.

Id. at 2431.

In conclusion, the court found that Mr. Liebich had not presented sufficient information for the appointment of independent counsel based on her investigation in open court and her own recollection of the trial testimony. Instead, these issues were preserved for appeal.

At the same hearing, Mr. Holman asked the court to reduce the sentence based on the court's insistence that Robert Liebich's description of the hot dog incident constituted an admission of criminal conduct. Mr. Holman argued that:

> There were substantial grounds tending to excuse or justify the defendant's criminal conduct though failing to establish a defense. Where we are basically indicating that his conduct of trying to dislodge his finger and his thumb from the child's mouth is not in fact criminal conduct. And if the Court is relying on the action of trying to dislodge his fingers from the child's mouth by perhaps slapping the child on the side of the cheek for him to let go, that that conduct again we don't believe is criminal, but we believe that the Court may have been relying on that action in its ruling, and we do not believe that that conduct is enough to justify criminal conduct.

Id at 2433-34.

Mr. Holman reiterated that Mr. Liebich had always been good with children and had little history of prior delinquency or criminal activity. He understood that the court did not give any credibility to Ms. Zeis' testimony that she had seen Kenyatta beating Steven and throwing him from one room to another, but that testimony was in the record. In contrast, not a single person had seen Mr. Liebich strike Steven, indicating a low likelihood of recurrence. The State responded by describing Mr. Liebich's actions as a "flogging" with the intent to murder. *Id* at 2437.

The court concluded by stating that "setting aside what the pathologist said" – which must of course be done if Mr. Liebich were to be found guilty – the treating doctors found that the injuries were acute, meaning recent. *Id*at 2439. She repeated that she found the testimony of the Mount Sinai and Rush doctors to be "compelling if not chilling" in their description of the extent of the injuries and the amount of force that had to be used on a child that size to produce the documented injuries. *Id.* The court concluded that Mr. Liebich "should never be put in the presence of a child again." *Id.* To achieve this end, she reaffirmed the 65 year sentence, with all time to be served.

G. Appeal

Mr. Liebich's appeal raised four grounds: (1) a request for remand on Mr. Liebich's *pro se* IAC claim since the trial court failed to conduct an adequate inquiry into the basis for the claims; (2) failure to prove guilt beyond a reasonable doubt; (3)

the admission of testimony of the treating physicians on the timing of injury in the absence of sufficient expertise to make this determination under Frye; and (4) IAC based on the failure to object and exacerbation of inadmissible expert testimony; the failure to effectively impeach the State's witnesses; error in arguing that Dr. Teas' letter was not to be considered by the trial court; failure to argue a lesser included offense; and filing a certificate and making statements that contained material misrepresentations.

The appellate court's ruling is attached as Exhibit 20. The court of appeals held that it was for the trial court to decide which was more credible: the timing of injuries on pathology (slides), or eyeballing the color of blood during surgery. The court held that, in response to Dr. Severin's testimony on timing, "defendant presented countervailing scientific evidence (the histology slides), and it was for the trial court to resolve the conflict between Severin's testimony and that scientific evidence just as it would have been for the trial court to attribute additional weight to Severin's opinion had it been supported by corroborating scientific evidence." The court agreed that it was possible to infer intent from the "quantity and severity" of Steven's injuries, including the "number of blows" directed to Steven's head and abdomen. The court concluded that substantial evidence supported the finding of guilt, "not the least of which was Kenyatta's testimony that Steven was fine when she left for work, Steven spent the day alone with defendant, and when Kenyatta returned, Steven was severely injured," and that the Court was not persuaded that Mr. Liebich was not proven guilty beyond a reasonable doubt.

Citing *Strickland*, the court of appeals held that the failure of defense counsel to object to medical testimony, effectively cross-examine and impeach witnesses, argue a lesser included offense or present the Teas' letter did not constitute ineffective assistance of counsel since it was not established that defense counsel had fallen below an objective level of reasonableness or that this deficient performance prejudiced the defendant.

The Court also addressed whether Dr. Munoz' testimony that he could time the injury based on the "bright red" color of the blood met *Frye*. The Court held that *Frye* did not apply to nonscientific testimony, such as that offered by Dr. Munoz. Instead, Dr. Munoz' testimony that bright red blood indicates a recent injury is not subject to *Frye* because it is based on his own experience. His expertise was, in short, akin to that of a beekeeper, and his expertise was "not derived from the abstract application of scientific principles but was based instead on what he had observed in his years as a doctor." (Ex. 20, Rule 23 Order at 49-50.) Since Dr. Severin was relying on Dr. Munoz, the challenge to Dr. Severin was derivative. Since Dr. Munoz's testimony was proper, Dr. Severin's reliance on Dr. Munoz was also proper. (*Id.* at 50.) It was then up to the trial court to resolve the conflict between the opinion testimony offered by Dr. Munoz (and derivatively by Dr. Severin) and the scientific evidence offered by Dr. Teas and Dr. Mileusnic. (*Id.*) Later, the Court emphasized that basing an opinion on the color of blood is not an accepted scientific methodology or a scientific methodology at all; instead is based

on Dr. Munoz' skill and experience based upon his observations made in the course of his career. (*Id.* at 62.)

The Court also held that the State had sufficiently proven the intent to kill or do great bodily harm from the quantity and severity of the injuries Steven sustained, specifically, the number of blows directed at Steven's head and his abdomen, both of which are vital parts of the body. (*Id.* at 55.)

Finally, the Court held that the five arguments on ineffective assistance of counsel (IAC) were insufficient to support an IAC claim. These included trial counsel's: (1) failure to object to Dr. Munoz' and Dr. Severin's testimony on timing; (2) agreement that Dr. Teas' letter to the court after the trial concluded should be impounded; (3) failure to argue that Randy was guilty of a lesser-included offense, namely, involuntary manslaughter; (4) failure to effectively cross-examine and impeach several witnesses (based on string citations); and (5) provision of material inaccuracies in a Rule 604(d) certificate. With the exception of Dr. Teas' letter, on which the Court spent some time, each of these claims was summarily dismissed. The Court held that trial counsel's failure to endorse the Teas' letter did not constitute ineffective assistance of counsel since some of the information had already been presented and the defendant had not shown that a reasonable probability that a different outcome would have followed had this evidence been presented.

Justice O'Malley dissented, stating that, in her letter, Dr. Teas identified what she considered to be relevant exculpatory evidence that was not adduced at

trial. Justice O'Malley specifically stated, "[Dr. Teas] wondered why Steven had aspirin in his blood if he had been feeling well, she wondered why Steven had lost four pounds in the four months prior to his death, she noted that some of the drugs doctors administered to Steven may have caused his abdomen to be soft, and she noted that it was not uncommon for symptoms of children's abdominal injuries to be delayed as much as 2-3 days after the injury." (*Id.* at 71.)

Justice O'Malley felt that Mr. Liebich's allegation that his counsel ignored these facts presented a potentially meritorious claim of ineffective assistance of counsel. He further noted that counsel admitted being unaware of at least one of these medical facts (the presence of aspirin and Tylenol in the child's blood). Justice O'Malley felt that the other cited facts were also sufficient for Mr. Liebich to be given the opportunity to argue that their exclusion was not a matter of reasonable trial strategy. (*Id.* at 71-72.)

H. Prior Post-Conviction Pleadings

On February 23, 2009, Randy Liebich filed a *pro se* Petition for Post-Conviction Relief raising as claims (1) his Fifth, Sixth and Fourteenth Amendment Rights were violated by police questioning, and his Sixth Amendment right to counsel was violated by his counsel's failure to move to suppress his statements; (2) Trial counsel's failure to allow him to testify at trial violated his rights under the state and federal constitution; (3) Appellate counsel was ineffective by failing to raise the sufficiency of the evidence; (4) Trial counsel was ineffective by failing to effectively investigate his defenses and to effectively cross-examine and impeach

state witnesses; (5) Cumulative error denied him the right to due process and a fair trial; and (6) He is actually innocent of the crime for which he was convicted. (Ex. 29, *Pro Se* Post-Conviction Petition.) Mr. Liebich subsequently moved to supplement his Petition with additional exhibits supporting those claims. (Ex. 30, April 1 Supplemental *Pro Se* Filing.) Mr. Liebich later added additional exhibits to this petition - those exhibits were incorporated into his Amended Petition for Post-Conviction Relief, filed June 11, 2008, which he filed with the assistance of counsel. (Ex. 31 Amended Petition for Post-Conviction Relief.) His Amended Petition was a two-page brief, adding additional exhibits and adding as additional claims that (1) Mr. Liebich was denied the effective assistance of counsel where his attorney failed to investigate and litigate a motion to suppress, and (2) Mr. Liebich was denied the effective assistance of counsel where his attorney prevented him from testifying at trial. (*Id.* at 2.) This Court later appointed current counsel to represent Mr. Liebich and allowed counsel the opportunity to further amend Mr. Liebich's prior claims. Mr. Liebich incorporates the claims and evidence raised in his prior *Pro Se* and Amended Post-Conviction Petitions, and supplements those pleadings with the attached Supplemental Petition and attached Exhibits filed in support of his petition.

IV. NEW EVIDENCE SUPPORTING POST-CONVICTION RELIEF

A. Overview

Randy Liebich was convicted of the murder of Steven Quinn primarily because medical evidence appeared to establish that Steven Quinn's death was

caused by injuries sustained shortly before his death. Because Randy Liebich was admittedly watching Steven during this time period, he was the one and only possible suspect. This theory doomed Randy. It meant that all of the other evidence he had demonstrating his good relationship with Steven, demonstrating that Kenyatta had a history of abusing Steven, and demonstrating that Steven had prior injuries, seemed inconsequential. It made this case an easy conviction. With the discovery of new exculpatory evidence, however, this case has completely changed.

Below, we explain how new medical understanding reveals that Steven's head trauma was a secondary result of the abdominal injury that was sustained days earlier - and that could not have been caused by Randy. This argument can be summarized as follows: Abdominal Injury/Ischemic Bowel (unknown origin) → Peritonitis → Pancreatitis → Hypoxia (lack of oxygen to the brain) → Brain Swelling and Bleeding → Death.

B. Affidavits from Medical Experts

The new evidence presented in this Petition comes from a wide variety of medical experts who have reviewed the records related to Steven's death. As set forth in their affidavits, there is no evidence that Steven had a traumatic head injury, and no evidence that he was beaten on the day of hospital admission. Instead, his death was due to abdominal injuries/infection that were present days before hospital admission, leading to pancreatitis, a hypoxic brain and a secondary coagulopathy (disseminated intravascular coagulation). The affidavits were provided on a *pro bono* basis by Dr. Patrick Barnes (pediatric neuroradiologist); Dr.

Michael Laposata (clinical pathologist with expertise in coagulopathy); Dr. Darinka Mileusnic (forensic and anatomic pathologist); Dr. Shaku Teas (anatomic, clinical and forensic pathologist); Dr. George Nichols (anatomic, clinical and forensic pathologist); Dr. Waney Squier (pediatric neuropathologist); Dr. Ronald Uscinski (neurosurgeon); and Nathan Felix (trauma medic).

1. Dr. Patrick Barnes (Exhibit 1).

Dr. Barnes is a Professor of Radiology at Stanford University Medical Center and Chief of Pediatric Neuroradiology and Medical Co-Director of the MRI/CT Center at Lucile Packard Children's Hospital at Stanford. He has practiced and taught on head injury in children for thirty years, and has published over a hundred articles, reviews and book chapters on this subject. His affidavit, curriculum vita and most recent article on this subject are attached as Exhibit 1.

Dr. Barnes agrees with the Mt. Sinai radiologists that the CT scan does not show fractures, soft tissue swelling or other abnormalities that would suggest head trauma. There are thin hemorrhages (subdural, subarachnoid, intradural and/or intraparenchymal), the beginning of a hypoxic-ischemic brain (*i.e.,* a brain that lacks oxygen), suspicion of thrombosis (clotting) in the dural sinuses (the large veins that draina the brain), and sinus disease. The hospital x-rays show bilateral pulmonary (lung) disease.

Dr. Barnes concluded that the conviction appeared to rest largely on the initial misinterpretation of the CT scan and outdated medical literature. He points out that Steven died less than a year after publication of the Geddes research

(2001), which found that the swollen brains in infants reflects hypoxia ischemia (lack of oxygen to the brain) rather than traumatically torn axons, and that thin subdural hemorrhages are also found in natural deaths. By 2004, some of the prosecution witnesses were aware of the shift in the literature, but others continued to rely on the basic tenets of shaken baby syndrome, which held that the subdural hemorrhages and brain swelling can only be caused by tremendous force (often described as equivalent to a multistory fall or motor vehicle accident) and are immediately symptomatic. Since 2004, the child abuse literature has recognized that there are many natural and accidental causes for these findings and that there can be lucid intervals of up to 72 hours.

Dr. Barnes noted that the lab reports, discharge diagnosis and autopsy report established that the child had abdominal injury/infection with systemic inflammatory response syndrome (SIRS) and multiple organ dysfunction syndrome (MODS). The CT scan was consistent with these diagnoses and did not suggest head trauma. The dense dural sinuses were suspicious for thrombosis (abnormal clotting), which would not be unexpected in the presence of abdominal injury/infection and would account for the brain findings and thin hemorrhages. The child had multiple risk factors for thrombosis, including sinusitis, inflammatory bowel disease, poor appetite, vomiting, lethargy, unsteady gait and seizures.

Dr. Barnes states that there is no medical or scientific basis for the testimony that it is possible to time an injury based on the color of the blood. To the contrary, it is relatively common to find evidence of older injuries or processes under the

microscope that cannot be seen on CT or by the naked eye. There is also no medical or scientific basis for the testimony that there can be no lucid interval following abdominal or head injuries, and that the force to create Steven's injuries would be the equivalent of falling from 20-30 feet or of blunt trauma with a bat, brick, foot or fist to the abdomen. Abdominal injuries may be caused by relatively minor trauma and may not become seriously symptomatic for days. Head injuries may also be slow to develop.

Dr. Barnes agreed with Dr. Munoz and Dr. Severin that the hypoxic brain was a recent development, likely occurring around the time of collapse. This finding is, however, likely secondary to pre-existing abdominal injuries and infection. There is no radiological evidence of head trauma and nothing in the records suggesting that the abdominal injuries/infection began on the day of collapse. Any attempt at dating should be coordinated with the clinical symptoms and pathology.

Since Dr. Barnes did not review the hospital and autopsy photographs, he did not comment specifically on the marks and lines that appeared after hospital admission. He noted, however, that the fact that these marks were not evident on arrival suggests that they might reflect a coagulopathy such as disseminated intravascular coagulation, rather than trauma. A coagulopathy would also explain the thrombosed sinuses suggested on the CT scan.

Dr. Barnes concluded that the CT scan was consistent with thrombosis and hypoxia-ischemia secondary to the abdominal injuries/infection confirmed in the lab tests and at autopsy. The choking episode was likely a symptom of the abdominal

injury/infection and may have triggered or accelerated the collapse. There is also a possibility of aspiration (*e.g.*, inhalation of food into the lungs during the choking episode).

2. Dr. Michael Laposata (Exhibit 2).

Dr. Laposata is Pathologist-in-Chief and Director of Laboratory Medicine and Clinical Laboratories at Vanderbilt. He is also a leading national coagulation expert who teaches and publishes regularly in this area. His affidavit, *curriculum vitae*, one of his articles and two of his presentations are attached to this Petition as Exhibit 2. After reviewing the laboratory reports and hospital and autopsy photographs, Dr. Laposata pointed out that:

> Given the history and autopsy findings, the child's illness likely began with an ischemic bowel. As the walls of the ischemic bowel deteriorated, the contents leaked into the peritoneal cavity, affecting the surrounding organs, including the pancreas.
>
> As the inflammation spread, the body would have produced additional platelets to help stop the process of bleeding. The platelets were consumed in the process known as DIC, which produces bleeding, bruising and thrombosis.
>
> A child in DIC may bruise spontaneously or from minor trauma, including medical intervention or handling. It is not possible to determine visually whether bruises or contusions are caused by trauma or a coagulopathy. Since the marks on the child were not apparent on admission but appeared during hospitalization, some or all were likely due to DIC and handling of the child as care was being provided, rather than pre-existing trauma.
>
> DIC also produces thrombosis, which can cut off the blood supply or return from arteries or veins, producing ischemia. If the thrombosis occurs in the arteries or veins that supply or drain the brain, this can cause a hypoxic ischemic brain.
>
> The most likely progression in this case is an ischemic bowel progressing to peritonitis and pancreatitis. The final stage (severe pancreatitis and a

> hypoxic ischemic brain) is likely what brought him to the hospital. Before that, he may have been only mildly symptomatic (lethargy, cold symptoms, refusal of food, etc.).
>
> . . . Ischemic bowel, *i.e.,* decreased flow of blood to the bowel, can result from many different causes. In my experience, ischemic bowels are most often natural in origin. . . . Other causes of ischemic bowel include impacted food or thrombosis, *i.e.,* formation of a blood clot in the blood vessels supplying blood to the bowel. Pneumonia has also been implicated as a possible cause, suggesting that an ischemic bowel may result from a reduced oxygen supply from any source. . . . If a child has been kept on life support for some days before autopsy, it may be difficult or impossible to determine the source of the ischemic bowel since intussusceptions, impacted food and/or thrombosed veins or arteries may have been present and then resolved.
>
> . . .
>
> The best way to determine when the bowel became ischemic and the order in which these events occurred is to examine the pathology (microscopic slides). Since the process continues after hospitalization until the child is taken off life support, it is important to look for the earliest findings on pathology as these will provide the best indications of when the process began. The slides may also give some idea of relative timing.
>
> Irrespective of the pathology, I would not expect this entire process (ischemic bowel, peritonitis, pancreatitis, liver inflammation and DIC) to occur within approximately eight hours of hospitalization. Instead, I would expect the process to evolve over a period of days.

(Ex. 2, Dr. Laposata Aff. at ¶¶ 26-35.)

Dr. Laposata pointed out that the ischemic bowel in this case may have been natural, accidental or abusive in origin. If traumatic, he would look for a traumatic event occurring at least a day before hospital admission and possibly longer. He also pointed out that the small bowel perforation may have occurred during hospitalization as the walls of the bowel continued to break down. From a clinical perspective, since the abdominal injuries/infection had progressed to pancreatitis by

the time of hospital admission, the process likely began at least a day before hospital admission and possibly earlier. (*Id.* at ¶ 37.)

3. Dr. Darinka Mileusnic-Polchan (Exhibit 3).

Dr. Mileusnic is the Cook County medical examiner who conducted Steven's autopsy. Dr. Mileusnic is currently the Chief Medical Examiner for Knox County and Anderson County, Tennessee. She is also an Assistant Professor in the Department of Pathology, University of Tennessee Graduate School of Medicine. She is board certified in anatomic and forensic pathology Her affidavit is attached to this Petition as Exhibit 3.

In February 2012, Dr. Mileusnic reviewed the autopsy slides and medical records for Steven Quinn in light of new information, including a surgical report that establishes that the massive subdural hemorrhage reported in the investigative report did not exist, laboratory tests confirming pancreatitis shortly after hospital admission, and new stains of autopsy slides that show injuries occurring well before February 8.

These slides establish that Steven had two conditions that preceded his arrival at the Liebich home on February 2: (1) myocarditis (damage to the heart), which would have reduced his circulation and increased his vulnerability to trauma or infection; and (2) a healing hematoma in the area outside the pancreas that was at least 10 days old and was most likely 2-3 weeks old. These are new findings that were not addressed at trial. The slides, including the new stains, establish that the remaining abdominal injuries occurred before February 8, most likely around

February 6. Since myocarditis and the peripancreatic hematoma would have made the child susceptible to trauma or infection, these injuries would not require major trauma and are consistent with a push, shove or inappropriate punishment.

The surgical report establishing that the "massive" subdural hemorrhage described in the investigative report was minimal or did not exist eliminated the basis for a finding of head injury. Massive subdural hemorrhages almost always represent ruptured bridging veins (the relatively large veins that drain the brain) and are usually caused by significant impact (accidental or abusive). Thin subdural hemorrhages in children may, however, may be secondary to natural causes, including infection in other parts of the body. In this case, the thin subdural seen on the CT and described in the surgical report is consistent with the abdominal infection and does not suggest head trauma. The forehead bruise is older (*i.e.*, occurred before February 8) and is of unknown significance.

The laboratory reports reviewed in February 2012 further confirmed that Steven had pancreatitis on admission. Dr. Mileusnic is quite certain that she did not receive these reports before the trial as she would have remembered the extraordinarily high levels of amylase and lipase. The lab reports also show that Steven's platelets dropped rapidly after admission. When confronted with injury from trauma or infection, the body sends platelets to attempt to "plug" or repair the damage. Once the platelets are used up, the body cannot regulate the bleeding/ clotting process, resulting in hemorrhage, thrombosis and/or easy bruising. This is known as disseminated intravascular coagulation (DIC). In this context, the

majority of the lines and marks that appeared during the hospitalization likely reflect pancreatitis and disseminated intravascular coagulation (DIC). The exception is the bruising on the back, which is likely associated with the earlier abdominal injuries. Although these marks were not noted on admission, it can be difficult to identify bruising on African-American children, and it is not clear that Steven's back was examined thoroughly before the CT scan. These marks would have increased in size and deepened in color from DIC.

Dr. Mileusnic noted that during the trial, she was shown hospital photographs that she had not seen previously. In these photographs, some of the marks seen in the hospital had disappeared by the time of autopsy while other marks that were not seen in the hospital appeared at autopsy. This further suggests that some of the marks were associated with DIC rather than trauma.

Dr. Mileusnic confirms that her original report on the tissue slides states that Steven's injuries were subacute (five days or more) or approximately five days old. When Dr. Mileusnic returned to Illinois for the trial, the prosecutor urged her to place the injuries three days before death or to testify that this was possible. She made clear that this was very improbable given the stage of healing and made clear that her best estimate of timing was five days or slightly longer. The prosecutor understood her position and did not question her on the timing of the injuries.

Since the new information made it possible to provide more accurate information on timing (particularly relative timing, *i.e.*, which findings came first and which came later), Dr. Mileusnic reviewed all of the slides. This review

confirms that Steven had myocarditis (damage to the heart), most likely caused by a virus 2-3 weeks earlier, damaging his circulation and making him vulnerable to traumatic or hypoxic ischemic injury. He also had a healing hematoma outside the pancreas that was at least 10 days old and likely 2-3 weeks old. Approximately five days before death, Steven developed an ischemic bowel with liver involvement and hemorrhage in the diaphragm. While there are natural causes for the abdominal findings, the bruises on the lower back are suspicious for trauma given their location and extent. There is no evidence of head trauma and no evidence of any trauma occurring on the day of collapse. Instead, the events on the day of admission represent a natural progression of the earlier injuries, which culminated in pancreatitis and a hypoxic-ischemic brain. The majority of the findings on the scalp, skull, meninges and brain were the consequences of surgical intervention.

4. Dr. Shaku Teas (Exhibit 4).

In a recent affidavit, attached as Exhibit 4, Dr. Teas confirms that the pathology established that the abdominal injuries most likely occurred by noon on February 6 or earlier. It is possible that the injuries occurred early on February 7, but some portions of the injuries appeared to be closer to the seven day range. Dr. Teas states that when she spoke with Dr. Mileusnic before the trial, Dr. Mileusnic confirmed that the injuries were approximately 5 days old, as set forth in her written postmortem report. Since this precluded Mr. Liebich, Dr. Teas conveyed this information to Mr. Ruggiero, the prosecutor, pointing out to him the pages in the autopsy report that provided timing. It was Dr. Teas' impression that Mr. Ruggiero

had not been aware that the medical examiner had timed the injuries to a period before Mr. Liebich cared for Steven.

When Mr. Ruggiero proceeded to trial despite pathological evidence that excluded Mr. Liebich, Dr. Teas advised Mr. Holman and Mr. Casey that, among other things, they needed to (1) understand how pathological timing is done; (2) review the slides with Dr. Mileusnic so they could understand the basis for her opinion and refresh her memory as she would not have had access to the slides after leaving the Cook County M.E.'s office in 2002; and (3) establish that Steven had been symptomatic in the days before his collapse, as evidenced by his weight loss and the Tylenol in his system. None of these steps were taken, and the latter information was not introduced at trial.

Dr. Teas further states that Mr. Casey incorrectly stated in her testimony in his closing argument. Specifically:

> I just learned that Mr. Casey suggested in his closing argument that I was not sure whether I saw older injuries, *i.e.*, injuries occurring before February 8. This was not my testimony. While pathological dating is not precise and in medicine one can rarely say "never," my testimony was that the healing and reaction seen in the slides represented injuries that occurred on or before the morning of February 7. This testimony was based on established pathological principles.

(Ex. 4, Dr. Teas Aff. at ¶ 13.)

This mischaracterization was unfortunate since the Court's verdict relied heavily on Dr. Teas' alleged agreement that the injuries could have occurred on February 8. (*Id.* at ¶¶ 15-16.)

Dr. Teas also reviews three types of new evidence in her affidavit. First, Dr. Teas had new stains done on the slides at her own expense after the trial. These stains confirmed that the abdominal injuries were at least five days old.

Second, the lab tests confirm that Steven had pancreatitis and a coagulopathy (disseminated intravascular coagulation) at the time of hospital admission. These would explain the marks and lines on Steven's body with the possible exception of the bruises on the lower part of Steven's back, which could represent a push or shove, resulting in a crush injury (consistent with the abdominal findings). DIC might also explain the head findings since the small subdural/subarachnoid hemorrhages and hypoxic brain are consistent with a coagulopathy (bleeding/thrombosis).

Third, Dr. Teas reviews the major changes in the medical literature since Steven's death in 2002 and the trial in 2004. These changes – which are also summarized by Dr. Stephens (through 2008) and Dr. Barnes – are important since they explain why the treating physicians reached the conclusions that they reached in 2002. Dr. Teas' description of the changes in the literature is succinct and worth repeating in its entirety:

> There have been major changes in the medical literature since Steven's death in 2002 and the trial in 2004. In the early 2000s, it was widely believed that swollen brains were caused by the traumatic tearing of axons (the nerve fibers that connect the cells of the brain) throughout the brain and that subdural hemorrhages were caused by the traumatic rupture of the bridging veins that connect the brain to the superior sagittal sinus (the large vein that drains the brain). It was further believed that such traumatic tearing would require a major force, often described as equivalent to a major motor vehicle accident or fall from a multistory building.

In 2001, a position paper published in the journal of the National Association of Medical Examiners (NAME), the professional organization for forensic pathologists, adopted these hypotheses and suggested that the force was caused by violent shaking. This position paper was not approved by the reviewers and was accompanied by an editorial caveat intended to make clear that it was not an official NAME position paper but rather represented the views of the authors. Despite these red flags, this paper became the foundation of many criminal prosecutions.

While this case was not a shaken baby case, the Rush diagnosis included shaken baby syndrome and the state's key trial witnesses relied heavily on the underpinnings of this theory, as set forth above.

Soon after the NAME paper was published, a series of research and review papers established that many of the assumptions in this paper were incorrect. Perhaps most important, neuropathological research papers published in 2001 confirmed that the brain swelling in infants was hypoxic-ischemic rather than traumatic and is also found in natural deaths. The same papers found that the subdurals seen in allegedly abused infants are small and thin, and are similarly seen in natural deaths. A third paper, also published in 2001, found that short falls can produce the same findings, disproving the former belief that it required the force of a fall from a multistory building. A 2003 paper published in the NAME journal found that there was no scientific or evidence-based research support for the shaken baby hypothesis. The current consensus is that there are numerous accidental and natural causes for the medical findings previously attributed to shaking or abuse, and that such findings may be secondary to other injuries or illnesses. There is also considerable consensus that children may have lucid intervals (periods of normality or relative normality) of up to 72 hours after a head injury that ultimately proves fatal.

In October 2006, the 2001 NAME position paper expired and the NAME annual meeting included papers with titles such as "The Use of the Triad of Scant Subdural Hemorrhage, Brain Swelling, and Retinal Hemorrhage to Diagnose Non-Accidental Injury Is Not Scientifically Valid."

In 2009 and 2010, new research confirmed that the small subdurals seen in allegedly abused children are too small to represent traumatic bridging vein rupture and that retinal hemorrhages are related to brain swelling and life support, rather than the traumatic rupture of retinal veins. Other research has further supported the role of hypoxia.

There have been similar changes in our understanding of abdominal injuries. Forensic medicine has long recognized that slow collapse from abdominal

injuries is common. This is a well-known phenomenon in children who hit the handlebars of bicycles or are impacted by a seatbelt and who present with abdominal injuries a day or more after the event. In her judgment, the Court stated that there is no lucid interval concept or theory with abdominal injuries and that there is an immediate onset of symptoms. It is my understanding that this was based on the testimony of a pediatric intensivist. In 2006 and 2009, however, the leading textbooks written or edited by child abuse pediatricians confirmed that abdominal injuries may progress slowly.

In view of these major changes in the literature, the courts are beginning to review child cases from the late 1990s and early 2000s that were based on misunderstandings of the progression of injury and disease in children. While Mr. Liebich was not accused of shaking Steven, the prosecutors and prosecution witnesses relied heavily on the tenets of shaken baby syndrome to support their claims, including the outdated beliefs that swollen brains and subdural hemorrhages represent torn axons and ruptured bridging veins, requiring the force of a multistory fall or major motor vehicle accident, and that there are no lucid intervals for head or abdominal injuries.

(*Id.* at ¶¶ 34-41 (internal citations omitted.))

5. Dr. George Nichols (Exhibit 5).

Dr. Nichols was the Chief Medical Examiner for Kentucky from 1977-97 and is currently a Clinical Professor in the Department of Pathology and a Clinical Associate Professor in the Department of Pediatrics at the University of Louisville School of Medicine. He is also emeritus staff at Kosair Children's Hospital in Louisville, Kentucky. His affidavit is attached as Exhibit 5.

Based on his review of the materials, with emphasis on the glass microscopic tissue slides and a recent review of the photographs, Dr. Nichols concluded that Steven had intra-abdominal injuries caused by blunt force trauma occurring at least 5-7 days before removal of life support. The brain findings reflected hypoxia-ischemia (lack of oxygen to the brain) with progressive cerebral edema and were a delayed reaction to the abdominal injuries with no independent significance given

the extent of the surgery and the time on life support (respirator brain). With the exception of the marks in the area of the spine and lower back, the marks and lines on Steven's body were consistent with normal childhood bruising, abdominal infection, hospital interventions and/or a coagulopathy. The marks in the area of the spine and lower back were consistent with being hit on the back or with falling on the back after being hit, pushed or shoved, and were likely associated with the abdominal injuries. In the absence of a report of an accidental injury, the injuries were likely inflicted. Based on the histology, it was not possible that the injuries were inflicted three days before removal of life support.

6. Dr. Peter Stephens (Exhibit 6).

Dr. Stephens, a former Deputy Iowa State Medical Examiner, provided an in-depth review of the records in 2009. His affidavit is attached as Exhibit 6. Dr. Stephens summarized the medical evidence as follows:

> The medical records confirm that Steven had a severe abdominal infection (peritonitis), leading to systemic inflammatory response (SIRS), sepsis, septic shock and multi-organ failure including the pulmonary, gastrointestional, cardiovascular and central nervous systems. He also had hypoxic/ischemic encephalopathy (brain swelling due to lack of oxygen), probably secondary to the abdominal infection. The abdominal infection was confirmed at the hospitals by laboratory tests and serosanginous drainage from the abdominal area. The autopsy report confirmed peri-pancreatitis (infection outside the pancreas), pancreatitis, liver damage and a small perforation in the small bowel.
>
> Abdominal infections in a young age group are generally associated with impact, either accidental (often bicycle accidents with impact against the handlebars) or inflicted (e.g., punch to the stomach). In this case, the younger age of this child (just under 3 years) and reported rough treatment by the mother suggests inflicted injury, but accidental causes cannot be excluded.

> Regardless of cause, the pathology establishes that the abdominal infection was present at least 7-10 days before death (4-7 days before collapse and hospital admission). It is not possible that it began as late as February 8, 2002. This infection progressed until the child's collapse on February 8 and continued after hospitalization.
>
> As determined at autopsy, the injuries to the brain were hypoxic-ischemic in nature (*i.e.,* due to lack of oxygen). This likely represented a natural progression of the abdominal infection, possibly triggered or aggravated by choking on a hot dog around 3 p.m. on February 8.
>
> I do not see significant signs of trauma in the hospital or autopsy photographs. Many of the signs interpreted as trauma are well-known indicia of abdominal injuries or artifactual (*i.e.,* attributable to medical interventions). The only significant marks are a series of marks down the child's spine that were small at the first hospital but that grew in size at the second hospital. These cannot be definitively identified as to causality but may have been caused by a fall, accidental or from a push, or other types of pressure. They may also represent hemorrhage from the pre-existing abdominal infection.
>
> At this point, it is not possible to determine the origin of the abdominal infection. Based on the evidence, the most one can say is that it began no later than February 6 (and likely earlier), progressing into sepsis, shock and multi-organ failure, as documented in the hospital records.
>
> . . . the caretaker accounts of the child's reluctance to eat on February 7 and the choking incident on February 8 are consistent with the pre-existing abdominal infection, followed by hypoxia-ischemia.

(Ex. 6, Dr. Stephens Aff. at ¶¶ 6-12.)

Based on the medical evidence, Dr. Stephens concluded that: (1) the abdominal infection and/or injury identified at autopsy were present at least 5-7 days prior to death (2-4 days prior to hospital admission), and likely longer; (2) it is not possible to determine whether the infection was accidental, abusive or natural in origin; (3) the brain findings are secondary to the abdominal infection and may have been triggered or aggravated by the choking incident; (4) there are no specific

indicators of head trauma; (5) the bruises and linear marks that appeared at the hospital are related to abdominal infection, sepsis and hospital intervention rather than recent abuse; (6) there is no medical evidence that any injuries occurred on the day of hospital admission.

7. Dr. Waney Squier (Exhibit 7).

Dr. Squier is a pediatric neuropathologist affiliated with Oxford University. In her affidavit, which is attached as Exhibit 7, Dr. Squier confirmed that the brain findings are largely consistent with the timing of Steven Quinn's collapse and hospitalization. Many of the vessels contain small fibrin thrombi, with a small thrombus in one dural sinus that may represent a portion of a more established clot in the draining veins or sinuses. These changes are consistent with venous outflow obstruction, including thrombosis, and altered coagulation secondary to hypoxia/ischemia. Other changes are nonspecific and may be seen with respirator brain or cardio-respiratory arrest of any kind, including choking. Although this cannot be determined fully without special stains, there is no evidence of primary traumatic damage. As this suggests, the "mangled" or traumatized brain described by the prosecutor in his closing argument did not exist, even under the microscope; instead, the pathology showed a hypoxic ischemic brain with evidence of thrombosis (abnormal clotting).

8. Dr. Ronald Uscinski (Exhibit 8).

Dr. Uscinski is a neurosurgeon who practices in the Washington, D.C. area. He is a Clinical Associate Professor in the Department of Neurological Surgery,

George Washington University School of Medicine; a Clinical Assistant Professor, Department of Pediatrics, Georgetown University School of Medicine; and a Senior Adjunct Fellow at the Potomac Institute for Policy Studies. He has special expertise in shaken baby syndrome/abusive head trauma, and has published, lectured and served as an expert witness on this subject on multiple occasions. His affidavit is attached as Exhibit 8.

Dr. Uscinski was asked to do a blind review of the CT scan and to address three questions: what did the CT scan show? was surgery appropriate and, if so, what kind? and what were the most likely causes for the CT findings?

Dr. Uscinski stated that the CT scan indicated an anoxic insult to the brain (*i.e.*, a brain that has been deprived of oxygen, with a breakdown of grey white differentiation), more on the left. There was some subdural hemorrhage along the cerebellum and occipital poles, very thin on the right, some subarachnoid hemorrhage and possible blood in the ventricles. There were no indicators of trauma (fractures, tissue swelling, etc.).

Based on the CT scan, he would not expect Steven to survive. The critical factor was the anoxic brain, not the thin hemorrhages, which were likely a side effect of anoxia. There was insufficient hemorrhage to drain surgically. Realistically, he would not expect any neurosurgical procedure to affect the outcome. The CT findings were consistent with any process that deprives the brain of oxygen. Since the narrow airway in young children can be obstructed by food or foreign body, one obvious possibility is upper airway obstruction. Other causes

include heart dysfunction, respiratory distress (from any cause), and shock. After being provided with a brief history, Dr. Uscinski indicated that the CT scan was consistent with pre-existing abdominal injuries/infection, possibly aggravated by choking.

9. Nathan Felix (Exhibit 9).

Nathan Felix, an Army medic with extensive experience in trauma cases, including aeromedical evacuations, provided an initial review of the labs and photographs. As set forth in his affidavit, attached as Exhibit 9, he noted immediately that the high glucose, amylase and lipase levels indicated that Steven had pancreatitis or a severe endocrine problem that would require emergency treatment, usually with antibiotics, insulin and an abdominal CT scan. He would not expect Steven to survive without prompt treatment, and the subsequent labs confirmed that all of Steven's organs appeared to be failing.

Mr. Felix also reviewed the photographs of the circular and linear bruises or marks on Steven's body. He noted that while bruises can come from external impact or systemic abnormalities, the circular bruises looked too small for punches and the lines were too thin for a belt. The lines could be from IV tubes or a hanger, but the delayed timing struck him as inconsistent with an earlier beating with a hanger, and some of the lines seemed to be in different places in different pictures. Mr. Felix therefore conducted an experiment. Since his skin color was close to Steven's, he was hit hard with a hanger repeatedly, breaking one or more hangers. This caused lines to appear within seconds. Within minutes, there were raised welt-like red

lines. Within 30-45 minutes, all marks had disappeared. This does not, of course, preclude the possibility that the lines could appear again if Steven developed a coagulopathy, but it does make the likelihood that they were from an earlier beating less likely. Mr. Felix further noted that the small bowel perforation found at autopsy might have been caused by the penrose drain, which can cause small perforations, particularly in the presence of abdominal infection.

B. Changes in Medical Understanding of Pediatric Head Injury

As several reviewing doctors noted, there have been tremendous changes in the literature on pediatric head injury since Steven's death in 2002 and Randy's trial in 2004. Since many of these are covered in the affidavits of Drs. Barnes, Teas and Stephens (Exs. 1, 4 and 5), we will briefly summarize the changes that are most relevant to this case.

In this case, the abdominal findings are clearcut: they began two days or more before hospital admission and gradually progressed, consistent with Steven's symptoms. The head findings, however, are a different story. In 2002, the Mount Sinai and Rush doctors assumed that brain swelling and thin hemorrhages were caused by trauma, and in 2004 all of the medical witnesses testified that Steven had a head injury. The only issue, therefore, was timing: when was Steven abused? And could there be a lucid interval – a period of relative normality in which Steven ate, drank and interacted? If there could be no lucid interval, Randy must be guilty. If there could be a lucid interval, then the evidence pointed away from Randy and toward either an accidental cause or toward Kenyatta.

To understand the changes over the past decade, it is necessary to review the history of shaken baby syndrome/abusive head trauma (SBS/AHT). SBS was advanced in the early 1970s as a hypothesis to explain three medical findings: (1) subdural hemorrhage (bleeding between the dura and the brain); (2) retinal findings; and (3) cerebral edema (brain swelling).[1] Together, these features are known as the "triad." Although the SBS hypothesis initially included signs of trauma (broken ribs, bruises, etc.), it was soon applied to children who had no such signs of trauma. The SBS hypothesis was simple: under this hypothesis, subdural hemorrhages were caused by the traumatic tearing of bridging veins; retinal hemorrhages were caused by traumatic traction or tearing within the eye; and cerebral edema was caused by traumatically torn axons in the brain. It was further assumed that each element of the triad required forces equivalent to a multistory fall or major motor vehicle accident, causing injuries so severe that the last person with the child must have caused the injuries.

1987. The first serious challenge to the SBS hypothesis arose in 1987, when an experiment at the University of Pennsylvania concluded that the force of shaking fell far below established injury thresholds and was approximately 1/50 the

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[1] This theory is generally credited to Dr. Caffey and Dr. Guthkelch. *See* John Caffey, *The Whiplash Shaken Infant Syndrome: Manual Shaking by the Extremities with Whiplash-Induced Intracranial and Intraocular Bleedings, Linked with Residual Permanent Brain Damage and Mental Retardation*, 54 Pediatrics 396 (1974); A. N. Guthkelch, *Infantile Subdural Haematoma and its Relationship to Whiplash Injuries*, 2 Br. Med. J. 430 (1971).

force from impact.[2] The paper also noted that the subdural hemorrhages in SBS cases were small and that the real problem was brain swelling.

1997. The SBS hypothesis was not seriously revisited until the 1997 trial of Louise Woodward, an English nanny who was charged with shaking the child of an American ophthalmologist and her husband.[3] Based on the triad, the prosecution experts testified that the child must have been violently shaken immediately before his fatal collapse, while others testified that the subdural hemorrhage resulted from a chronic (old) subdural, accompanied by re-bleeding.[4] At the time, the notion of a chronic subdural with rebleeding was viewed as a courtroom diagnosis; today, chronic subdurals with rebleeds are accepted.[5]

1998. In 1998, a study by Dr. Gilliland found that in approximately 25% of alleged abuse cases, there was an interval of more than 24 hours (and sometimes more than 72 hours) between the alleged abuse and the onset of severe symptoms.[6] This contradicted the mainstream belief that the severity of the injuries caused by shaking, shaking/impact or impact was inconsistent with a "lucid interval" and that the last person with the child was therefore the perpetrator. In the same year, an editorial in *The Lancet* noted that "[i]f 26 years after Caffey's description, doctors

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[2] Ann-Christine Duhaime et al., *The Shaken Baby Syndrome a Clinical Pathological and Biomechanical Study*, 66 J. Neurosurg. 409 (1987).

[3] *See Commonwealth v. Woodward*, 694 N.E.2d 1277 (1998).

[4] The *Woodward* case also involved a pre-existing skull fracture, not attributed to Ms. Woodward, that passed almost unnoticed in the general furor over shaken baby syndrome.

[5] Marguerite M. Caré, *Neuroradiology, in* Abusive Head Trauma in Infants and Children, a Medical, Legal, and Forensic Reference 73, 81 (2006) (septations or membranes that develop within chronic hematomas may predispose infants to repeated episodes of bleeding within these collections; such rebleeding can occur with little or no trauma).

[6] M.G.F. Gilliland, *Interval Duration Between Injury and Severe Symptoms in Nonaccidental Head Trauma in Infants and Young Children,* 43 J. For. Sci. 723 (1998).

are still undecided about the "shaken-baby syndrome," the difficulties faced by experts in presenting medical evidence in court, and by the judge and jury in making sense of it, are readily imaginable."[7]

1999. In 1999, Dr. Piatt identified external hydrocephalus (large heads due to CSF collections) as a risk factor for subdural and retinal hemorrhages from minor trauma, such as a bump on the head, providing a natural explanation for these findings, in some instances.[8]

2001. 2001 was both the peak of the SBS hypothesis and the beginning of its unraveling. In this year, the American Academy of Pediatrics (AAP) and the National Association of Medical Examiners (NAME) issued policy statements on SBS/AHT. The AAP paper endorsed the SBS hypothesis and suggested that child abuse be presumed whenever a child younger than 1 year suffers intracranial injuries.[9] The NAME paper, which did not pass peer review but was published as an opinion piece, stated that inflicted head injuries in children under the age of 4 or 5 usually create shearing injuries of the brain and blood vessels, resulting in diffuse axonal injury and subdural, subarachnoid and retinal hemorrhages.[10] In recent

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[7] Editorial, *Shaken Babies*, 352 The Lancet 335 (1998).

[8] Joseph H. Piatt, *A Pitfall in the Diagnosis of Child Abuse: External Hydrocephalus, Subdural Hematoma, and Retinal Hemorrhages*, 7 Neurosurg. Focus 4 (1999).

[9] Comm. on Child Abuse and Neglect, Am. Acad. of Pediatrics, *Shaken Baby Syndrome: Rotational Cranial Injuries – Technical Report*, 108 Pediatrics 206 (2001).

[10] Mary E. Case et al., *Position Paper on Fatal Abusive Head Injuries in Infants and Young Children*, 22 Am. J. Forensic Med. Pathol. 112 (2001); Email from Vincent Dimaio to NAME-L@Listserve.cc.emory.edu (Feb. 7, 2002) ("As editor of the AJFMP, I had serious misgiving about publishing this paper, not because of its contents but in that it is described as a position paper . . . If one bothers to read the box in the lower left corner of the first page of the article, one will see that the paper was rejected as a position paper by the three reviewers . . . As an aside, the paper in its

years, the NAME paper has been withdrawn and the AAP paper substantially modified.[11]

In the same year, Dr. Geddes published the first careful neuropathological studies of infants who were allegedly the victims of nonaccidental head injury (Geddes I and II). Geddes I found that, unlike the findings in older children, the subdural hemorrhages in infants were typically "trivial" in terms of quantity and almost invariably described as "thin film." Even more striking, the brain swelling (edema) in infants was hypoxic (*i.e.*, due to lack of oxygen) rather than traumatic in nature.[12] Geddes II found that the scientific evidence for the proposition that the triad is traumatic in origin was "scanty" and that the subdural hemorrhages and brain swelling in infants who died natural deaths were virtually indistinguishable from the findings in allegedly abused infants.[13] An accompanying editorial described this research as "meticulous" and noted that, given the findings of hypoxia, the vascular complications of hypoxia and/or raised intracranial pressure should be considered.[14] It took nearly a decade, but Geddes I and II are now part of the mainstream.

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original form was rejected by 4 of 5 reviewers . . . Shaken baby syndrome is controversial in that a number of individuals doubt its existence . . .) (e-mails on file with author).

[11] As addressed below, the NAME paper was rescinded in 2006; the AAP paper was modified in 2009.

[12] J. F. Geddes et al, *Neuropathology of Inflicted Head Injury in Children, I. Patterns of Brain Damage,* 124 Brain 1290 (2001) (often referred to as "Geddes I").

[13] J.F. Geddes et al, *Neuropathology of Inflicted Head Injury in Children, II. Microscopic Brain Injury in Infants,* 124 Brain 1299 (2001) (often referred to as "Geddes II").

[14] David I. Graham, *Editorial: Paediatric Head Injury,* 124 Brain 1261, 1261 (2001) (Geddes and her colleagues conducted a "meticulous clinicopathological correlation in 53 cases of non-accidental paedatric head injury").

In 2001, Dr. Plunkett, a forensic pathologist, addressed the common courtroom testimony that the triad could not be caused by anything less than a major motor vehicle accident or multi-story fall (anywhere from 3-10 stories, depending on the witness). In an article published in the NAME journal, Dr. Plunkett described witnessed short falls that resulted in some or all of the triad, including a videotaped fatal fall of a 23 month old from a plastic gym set (28 inches high) in the carpet-covered garage of her home.[15] The findings included subdural and retinal hemorrhages and brain swelling. This videotape established dispositively that short falls can be fatal and can cause the triad.

2002. In 2002, a review of the biomechanical literature in the British Journal of Neurosurgery concluded that it was improbable that manual shaking could produce the triad and that the assumptions made in the shaking model were unvalidated, ambiguous and/or incorrect.[16] In the same year, Dr. Jenny and other supporters of the SBS/AHT hypothesis published an article that recognized that subdural hemorrhages in children have a wide range of causes, including prenatal, perinatal and pregnancy-related conditions; birth trauma; metabolic diseases; congenital malformations; genetic diseases; oncologic diseases; autoimmune

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[15] John Plunkett, *Fatal Pediatric Head Injuries Caused by Short-Distance Falls,* 22 Am. J. Forensic Med. Pathol. 1 (2001).

[16] K. Ommaya et al., *Biomechanics and Neuropathology of Adult and Paediatric Head Injury,* 16 Br. J. Neurosurg. 220 (2002). Experiments by Professor Carole Jenny, a leading proponent of the SBS/AHT hypothesis, produced similar results. Jenny et al., *Development of a Biofidelic 2.5 kg Infant Dummy and Its Application to Assessing Infant Head Trauma During Violent Shaking,* 2002 Injury Biomechanics Research, Proceedings of the Thirtieth International Workshop, sponsored by the National Highway Traffic Safety Administration (on file with author).

disorders; clotting disorders; infectious diseases; the effects of poisons, toxins or drugs; and other miscellaneous conditions.[17]

In 2002, Dr. Barnes revisited the evidence base for shaken baby syndrome in view of the new evidence, including the Geddes research.[18] In this article, Professor Barnes pointed out that "[f]rom an evidence-based medicine perspective, quality of evidence ratings for diagnostic criteria regarding the literature on SBS reveal that few published reports merit a rating above class IV . . . Such quality of evidence ratings hardly earn a diagnostic criteria recommendation level of 'optional,' much less as a 'guideline' or a 'standard.'" Professor Barnes emphasized that radiologists must be aware of conditions that may mimic abuse, including accidental injury, coagulopathies, vascular diseases, infectious or postinfectious conditions, metabolic disorders, neoplastic diseases, certain therapies, and some congenital and dysplastic disorders.

Given the increasing controversies over shaking as a mechanism of injury, the National Institutes of Health held a conference in October 2002.[19] While the conference was attended largely by SBS/AHT supporters, there was general agreement that the research supporting the SBS hypothesis was largely circular,

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[17] Kent P. Hymel, Carole Jenny & Robert W. Block, *Intracranial Hemorrhage and Rebleeding in Suspected Victims of Abusive Head Trauma: Addressing the Forensic Controversies,* 7 Child Maltreatment 329 (2002).

[18] Patrick D. Barnes, *Ethical Issues in Imaging Nonaccidental Injury: Child Abuse,* 13 Topics Magnetic Resonance Imaging 85 (2002).

[19] *Inflicted Childhood Neurotrauma,* Proc. of a Conference Sponsored by Department of Health and Human Services, National Institutes of Health, National Institute of Child Health and Human Development, Office of Rare Disease and National Center for Medical Rehabilitation Research. The conference papers were published by the American Academy of Pediatrics in 2003.

that the current knowledge was limited, that there were many alternative diagnoses, and that additional research was needed.

2003. In 2003, Dr. Mark Donohoe reviewed the evidence base for shaken baby syndrome from 1966-1998 and concluded that the data in the medical literature was of poor quality and inadequate to support diagnostic assessment.[20]

In 2003, Dr. Geddes reported on 50 nontraumatic infant deaths (intrauterine to 5 months), with causes of death including infection, hypoxia, infant death syndrome (SIDS) and SBS. Since most of the natural deaths and all three SBS deaths showed intradural rather than subdural bleeding, Dr. Geddes suggested that the mechanism for the bleeding may be hypoxia-related leakage from veins within the dura rather than traumatic rupture of bridging veins, resulting from a cascade of events including raised intracranial pressure, central venous and systemic arterial hypertension, combined with immaturity and hypoxia-related vascular fragility.

In 2003, a case fatality report in the NAME journal documented a 72 hour delay between an accidental short fall and collapse, during which period the child had been asymptomatic.[21] This case involved a skull fracture, subdural and subgaleal hemorrhage, cerebral edema, and focal axonal injury, with no retinal hemorrhage.

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[20] Mark Donohoe, *Evidence-Based Medicine and Shaken Baby Syndrome Part I: Literature Review, 1966-1998*, 24 Am. J. Forensic Med. Pathol. 239 (2003).

[21] Scott Denton & Darinka Mileusnic, *Delayed Sudden Death in an Infant Following an Accidental Fall, A Case Report with Review of the Literature*, 24 Am. J. Forensic Med. Pathol. 371 (2003).

2004. In 2004, an article by Dr. Starling analyzed 81 perpetrator admissions to inflicted traumatic brain injury in children.[22] In 91% of the cases, the symptoms appeared immediately; in 5 cases, the timing occurred within 24 hours. The authors did not examine the validity of the admissions or the techniques used to elicit them, and their source material is unavailable.

In the same year, Dr. Lantz published a case report finding several ocular findings that had previously been considered to be diagnostic of abuse in an accidental television tipover.[23] Based on a literature review, Dr. Lantz concluded that the association of ocular findings with abuse suffered from selection bias, inappropriate controls, lack of precise criteria, a fallacy of assumption, unsystematic reviews, and consensus statements that mingled opinion with facts.

In 2004, Professor Goldsmith, a biomechanical engineer at the University of California at Berkeley, and Dr. Plunkett published another article addressing the biomechanical shortcomings of shaken baby theory.[24] The article concluded that the experimental biomechanical data indicated that the mechanism for retinal hemorrhage is functional or mechanical venous occlusion and suggested that this might be related to cerebral edema rather than trauma.

2005. A 2005 article by Dr. Bandak, a biomechanical engineer, found that the levels of force suggested by proponents of shaken baby theory would exceed the

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[22] Suzanne P. Starling et al., *Analysis of Perpetrator Admissions to Inflicted Traumatic Brain Injury in Children*, 158 Arch. Pediatr. Adolesc. Med. 454 (2004).

[23] P. E. Lantz et al., *Perimacular Retinal Folds from Childhood Head Trauma*, 328 Br. Med. J. 754, 756 (2004).

[24] Werner Goldsmith & John Plunkett, *A Biomechanical Analysis of the Causes of Traumatic Brain Injury in Infants and Children*, 25 Am. J. Forensic Med. Pathol. 89 (2004).

tolerance of the neck, causing total neck failure, and that the force from manual shaking is of the same order as a fall from approximately three feet.[25] While some of his calculations are disputed, Dr. Bandak's conclusions are consistent with the other biomechanical studies.

In the same year, an article by Dr. Leestma, a neuropathologist, found that the documented confessions in the medical literature (11 cases involving shaking only) did not permit valid statistical analysis or support for many commonly stated aspects of shaken baby syndrome.[26]

2006. In 2006, a textbook by leading supporters of the SBS hypothesis recognized that many medical disorders "mimic" SBS/AHT.[27] These include prenatal, perinatal and pregnancy related conditions; birth trauma; congenital malformations; various forms of childhood stroke; accidental injury; genetic and metabolic disorders; hematological diseases and disorders of coagulation and clotting; infectious diseases; autoimmune and vasculitis conditions; oncological processes; toxins, poisons and nutritional deficiencies; and medical and surgical complications. While some of these conditions can be confirmed or ruled out through diagnostic testing, others are indistinguishable from AHT or can only be ruled out by tests conducted while the child is living or, in some cases, after the child has died.

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[25] Faris A. Bandak, *Shaken Baby Syndrome: A Biomechanics Analysis of Injury Mechanisms* 151 Forensic Science International 71 (2005).

[26] Jan E. Leestma, *Case Analysis of Brain-Injured Admittedly Shaken Infants: 54 Cases, 1969-2001,* 26 Am. J. Forensic Med. Pathol. 199 (2005).

[27] Andrew P. Sirotnak, *Medical Disorders that Mimic Abusive Head Trauma, in* Abusive Head Trauma in Infants and Children, a Medical, Legal, and Forensic Reference 191 (2006).

In the same year, Dr. Lantz presented a study of retinal hemorrhages in 425 deaths (birth to age 96).[28] Retinal hemorrhages were found in 17% of all deaths, including a wide variety of natural and accidental deaths. Of the retinal hemorrhages found in 19 children, only 4 were related to abuse.

In October 2006, the NAME Board of Directors withdrew the 2001 position paper, which it had previously extended.[29] By then, the NAME annual meeting included presentations with titles such as "Use of the Triad of Scant Subdural Hemorrhage, Brain Swelling, and Retinal Hemorrhages to Diagnose Non-Accidental Injury is Not Scientifically Valid" and "Where's the Shaking?: Dragons, Elves, the Shaking Baby Syndrome and Other Mythical Entities."[30]

2007. In a 2007 article, Professor Barnes addressed the forensic controversies from an evidence-based perspective and urged radiologists to thoroughly familiarize themselves with the imaging, clinical, surgical, pathological, biomechanical and forensic literature and the principles of evidence-based medicine in providing a differential diagnosis for radiological findings previously associated with shaking or abuse.[31]

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[28] Patrick E. Lantz & Constance A. Stanton, *Postmortem Detection and Evaluation of Retinal Hemorrhages,* 12 Proc. of the Am. Acad. For. Sci. 271 (2006). Like Drs. Squier and Mack, Dr. Lantz has presented his research at meetings conducted by those on both sides of the "shaken baby" debate and in neutral settings, including the American Academy of Forensic Sciences.

[29] E-mail from Gregory G. Davis, Bd. of Directors, NAME, to John Plunkett, MD, and R. Wright (Oct. 17, 2006) (on file with authors). The 2001 NAME position paper had originally been scheduled to sunset in 2006; however, the Board extended it to 2008. In October 2006, the Board rescinded the renewal.

[30] Scientific Program, 40th Annual Meeting, National Association of Medical Examiners, San Antonio, TX (Oct. 13-18, 2006 (on file with author).

[31] P.D. Barnes & M. Krasnokutsky, *Imaging of the Central Nervous System in Suspected or Alleged Nonaccidental Injury, Including the Mimics,* 18 Topics Magnetic Resonance Imaging 53 (2007).

2008. In 2008, Dr. Rooks found that 46% of asymptomatic term newborns have subdural hemorrhage on MRIs taken within 72 hours of birth, confirming that subdural hemorrhages are not always symptomatic.[32] In the same year, Dr. Cohen, a histopathologist, presented a paper in which she reviewed the anatomy of the infant brain and identified several "myths" that had been adopted by the medical and legal professions but did not comport with the anatomy and pathology of the infant brain.[33] Dr. Cohen's research confirmed that, in young infants, the bleeding often referred to as "subdural" was in fact intradural; that it is also found in natural deaths; and that it is unlikely in many cases to result from tears in bridging veins. Instead, the common strand in these cases was often hypoxia/ischemia, or lack of oxygen to the brain, from any source, including natural disease processes.

In 2008, the legal system began to recognize the "new learning" on pediatric head injury, and the Wisconsin Court of Appeals reversed a 1996 first-degree reckless homicide conviction against Audrey Edmunds, holding that "a significant and legitimate debate in the medical community has developed in the past ten years over whether infants can be fatally injured through shaking alone, whether an infant may suffer head trauma and yet experience a significant lucid interval prior to death, and whether other causes may mimic the symptoms traditionally viewed as indicating shaken or shaken impact syndrome."[34] The Court held that this

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[32] V. J. Rooks et al, *Prevalence and Evolution of Intracranial Hemorrhage in Asymptomatic Term Infants,* 29 Am. J. Neuroradiol. 1082 (2008).

[33] M. Cohen, *Myths and Facts of the Subdural Haemorrhage in the Perinatal Period,* Third International Congress of Pathology, Barcelona, Spain (May 17-22, 2008).

[34] State v. Edmunds, 746 N.W. 2d 590, 596 ¶ 15 (2008).

legitimate and significant dispute within the medical community required a new trial. Ultimately, all charges were dismissed.

In October 2008, Commissioner Goudge issued his report on the Inquiry into Pediatric Forensic Pathology in Ontario, Canada.[35] While many of his findings focused on the misdiagnoses by a single pediatric pathologist, Commissioner Goudge concluded that the problem was systemic and that "the changes in pathology knowledge concerning shaken baby syndrome and pediatric head injuries over the last two decades provide cogent reason for a carefully constructed review of the cases" since some convictions may have been based on pediatric pathology that today would be seen as unreasonable.

2009. In 2009, Dr. Mack, Dr. Squier and Dr. Eastman published an article on the anatomy and development of the meninges in infants, particularly the dura.[36] In this article, they note that since the bridging veins are relatively large caliber vessels that would produce larger, more localized bleeds, traumatic bridging vein rupture is an unlikely source of the small thin film bleeds identified in the SBS/AHT literature. In the same year, Dr. Squier and Dr. Mack published an article on the neuropathology of infant subdural hemorrhage in which they concluded that it is unlikely that the widespread bilateral thin film subdural hemorrhage seen in infants has the same causality as the thick, space-occupying and often unilateral clot seen in older children and adults after trauma and that

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[35] Goudge, Inquiry into Pediatric Forensic Pathology in Ontario (Sept. 2008) *at* http://www.attorneygeneral.jus.gov.on.ca/inquiries/goudge/index.html *supra* note 4, Executive Summary at 48-49.

[36] Julie Mack, Waney Squier & James T. Eastman, *Anatomy and Development of the Meninges: Implications for Subdural Collections and CSF Circulation,* 39 Pediatr Radiol. 200 (2009).

alternative causes should be considered.[37] If correct, there is no longer any basis for the ruptured bridging vein hypothesis that formed the underpinning of SBS/AHT theory.

In the meantime, Dr. Cohen and Dr. Scheimberg published the results of a two year study that confirmed the association between hypoxic ischemic encephalopathy and intradural/subdural hemorrhage in fetuses and neonates. This study found that the degree of the hemorrhage was closely related to the degree of hypoxia.[38]

In May 2009, the AAP updated its policy statement on Abusive Head Trauma in Infants and Children, stating that the "advances in the understanding of the mechanisms and clinical spectrum of injury associated with abusive head trauma compel us to modify our terminology to keep pace with our understanding of pathologic mechanisms."[39] While continuing to endorse shaking or shaking/impact as a mechanism of injury, the Committee acknowledged that the "mechanisms and resultant injuries of accidental and abusive head injury overlap" and that medical diseases can also "mimic" the presentation of abusive head trauma. The following month, an editorial in Pediatric Radiology by leading supporters of the SBS/AHT

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[37] Waney Squier & Julie Mack, *The Neuropathology of Infant Subdural Haemorrhage*, 187 Forensic Sci. Int. 6 (2009).

[38] M.C. Cohen & I. Scheimberg, *Evidence of Occurrence of Intradural and Subdural Hemorrhage in the Perinatal and Neonatal Period in the Context of Hypoxic Ischemic Encephalopathy: An Observational Study from Two Referral Institutions in the United Kingdom*, 12 Pediatric & Developmental Pathology 169 (2009).

[39] Cindy W. Christian, Robert Block and the Committee on Child Abuse and Neglect. *Abusive Head Trauma in Infants and Children*, 123 Pediatrics 1409 (2009).

hypothesis acknowledged that "[m]aybe our current understanding of the actual pathogenesis of subdural bleeding (tearing of bridging veins) is incorrect."[40]

By then, it was apparent that all three components of the SBS/AHT hypothesis were wrong: brain swelling is not caused by traumatically torn axons, subdural hemorrhages are not caused by traumatically ruptured bridging veins, and retinal hemorrhages are found in a wide array of conditions. In September 2009, this "new learning" was reflected in a law review article entitled "The Next Innocence Project: Shaken Baby Syndrome and the Criminal Courts."[41] After reviewing the literature, Professor Tuerkheimer, a former child abuse prosecutor, concluded that "[a]s a categorical matter, the science of SBS can no longer support a finding of proof beyond a reasonable doubt in triad-only casess" and that "the presence of retinal hemorrhages and subdural hematoma cannot conclusively prove that injury was inflicted." Professor Tuerkheimer noted that even doctors who continue to defend the legitimacy of SBS "are willing to concede that the science has evolved – and that even mainstream thinking has changed in a number of areas."

2010. By 2010, it was widely understood that brain swelling reflected hypoxia rather than trauma, that there are many causes of subdural hemorrhage, and that the thin subdurals seen in young children were unlikely to have been caused by traumatically ruptured bridging veins. Supporters of the SBS/AHT hypothesis therefore increasingly relied on the size and shape of retinal

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[40] Steven Chapman & Thomas L. Slovis, *Response to Galaznik, Cohen & Scheimberg, and Rorke-Adams & Christian,* 39 Pediatric Radiology 770 (2009).

[41] Deborah Tuerkheimer, *The Next Innocence Project: Shaken Baby Syndrome and the Criminal Courts,* 87 Wash. Univ. L. Rev. 1 (2011) ("Tuerkheimer").

hemorrhages and other ocular findings to diagnose abuse. In February 2010, however, Dr. Evan Matshes reported that a retrospective review of retinal hemorrhages at the Dallas Medical Examiner's Office found that eye evaluations are of "limited value" in child death investigations.[42] His review confirmed that retinal hemorrhages are found in natural and accidental deaths as well as homicides and that their severity appeared to be linked to edema and life support rather than trauma. In the same month, retinal hemorrhages of a type previously viewed as diagnostic of abuse were found in two children who died from a common community-acquired pneumonia in children.[43]

2011. 2011 brought two major reviews of the literature on SBS/AHT.[44] In January 2011, the Radiological Clinics of North America published a major review on the imaging of nonaccidental injury and the mimics.[45] In this review, Dr. Barnes identified the lack of an evidence base for much of the SBS/AHT literature as well as the increasing list of alternative causes. A September 2011 review of the SBS

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[42] Matshes, *Retinal and Optic Nerve Sheath Hemorrhages Are Not Pathognomonic of Abusive Head Injury,* 16 Proc. of the Am. Acad. For. Sci. 272 (2010)

[43] Juan Pablo Lopez et al., *Severe Retinal Hemorrhages in Infants with Aggressive Fatal Streptococcus Pneumonia Meningitis,* 14 J. Am. Ass. Ped. Ophthal. Strab. 97 (2010). Severe retinal hemorrhages have also been reported in children with leukemia.

[44] A third review, this time from an emergency medicine perspective, was also published in 2011. Steven C. Gabaeff, *Challenging the Pathophysiologic Connection between Subdural Hematoma, Retinal Hemorrhage and Shaken Baby Syndrome,* 12 Western J. Emergency Medicine 144 (2011).

[45] A third review, this time from an emergency medicine perspective, was also published in 2011. Steven C. Gabaeff, *Challenging the Pathophysiologic Connection between Subdural Hematoma, Retinal Hemorrhage and Shaken Baby Syndrome,* 12 Western J. Emergency Medicine 144 (2011).

literature by Dr. Squier focused on the anatomy of the dura and the evolving neuropathology literature.[46]

While there are still disagreements, even the strongest advocates of the SBS/AHT hypothesis now recognize that there are many alternative causes for the triad. For example, at a September 2011 training conference, Dr. Jenny stated that "No trained pediatrician thinks that subdural hemorrhage, retinal hemorrhage and encephalopathy equals abuse. The "triad" is a myth!" Instead, abuse is now a "rule out" diagnosis requiring an extensive medical evaluation, detailed laboratory evaluations, a radiology review, and a review of all medical records.[47] In conducting this review, one must consider a differential diagnosis that includes trauma, metabolic disease, coagulopathies, genetic disease and infectious disease.

In 2011, the national media also began to address the issues. In February 2011, the New York Times Magazine published an article titled "Has a Flawed Diagnosis Put Innocent People in Prison? A Re-Examination of Shaken-Baby Syndrome," and in June 2011 PBS Frontline, NPR and Pro Publica ran a series on false convictions in cases involving child deaths, including SBS/AHT cases.

At the same time, the courts began to consider these issues. In October 2011, six of the nine U.S. Supreme Court justices upheld the SBS conviction of Shirley Ree Smith, whose grandson Etzel died in 1996, but indicated that "[d]oubts about whether Smith is in fact guilty are understandable" and suggested that a clemency

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[46] Waney Squier, *The "Shaken Baby" Syndrome: Pathology and Mechanisms*, 122 Acta Neuropathol. 519 (2011).

[47] Carole Jenny, Presentation, *The Mechanics: Distinguishing AHT/SBS from Accidents and Other Medical Conditions*, New York City Abusive Head Trauma/Shaken Baby Syndrome Training Conference (Sept. 23, 2011) *at* http://www.queensda.org/SBS_Conference/SBC2011.html

petition (now pending) might be appropriate. Justice Ginsberg, joined by Justices Breyer and Sotomayor, dissented, listing the criticisms of SBS/AHT theory and pointing out that "[i]n light of current information, it is unlikely that the prosecution's experts would today testify as adamantly as they did in 1997." The following month, Judge Posner addressed the changes in the literature on lucid intervals as well as the problem of false confessions.[48]

By 2011, many members of the medical community were having similar doubts. For example, in a September 2011 email, a forensic pathologist testifying on behalf of the prosecution in a criminal case privately advised the prosecutor that "I don't know what the breakdown is, but I would not be surprised to learn that it is close to 50/50 among neuropathologists, neurologists, and forensic pathologists as to whether any given case represents non-accidental trauma."[49]

2012. In 2012, the prediction of the dissenters in *Smith* that "it is unlikely that the prosecution's experts would today testify as adamantly as they did in 1997" is coming to pass. In February 2012, in another post-conviction case, Dr. Norman Guthkelch, often described as one of the founders of shaken baby syndrome, provided a declaration stating that:[50]

> I am aware that my 1971 article has been cited by doctors and researchers in support of a prosecutorial suggestion that babies who have subdural

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[48] *Aleman v. Village of Hanover Park*, 662 F.3d 897 (7th Cir. 2011) (Posner, J.).

[49] E-mail from Mark Peters, MD, to Sharyl Eisenstein, Assistant State's Attorney, McHenry County, IL (Sept. 15, 2011) (on file with authors) (regarding Sophia Avila Case #08-073, which resulted in conviction, Oct. 14, 2011). The same e-mail noted that infants can have a lucid interval of several days after head trauma and that a number of medical conditions can cause cerebral hemorrhage, retinal hemorrhage and bone fractures. These conditions should be ruled out before concluding that the injuries are the result of inflicted trauma.

[50] Declaration of A. Norman Guthkelch, M.D., *State of Arizona v. Drayton Shawn Witt*, Feb. 3, 2012.

> hematomas, retinal hemorrhages, and brain swelling can be *assumed* to be suffering from "Shaken Baby Syndrome," even when there are no other signs of abuse. However, I consider that this is a distortion of the article I wrote in 1971, resulting in that article being taken as support of a diagnosis of criminal liability in circumstances which I never envisaged.
>
> The term "Shaken Baby Syndrome" is an undesirable phrase. . . there was not a vestige of proof when the name was suggested that shaking, and nothing else, causes the triad.
>
> It is my understanding from reading the recent medical literature that the hypothesis that the triad can be caused only by shaking or shaking plus impact is still open to serious doubt. We know that a number of other conditions – natural and non-accidental – may lead to the triad. These conditions include metabolic disorders, blood clotting disorders, and birth injury, to name a few. . .
>
> In November 29, 2011, I was deposed by volunteer lawyers from the Arizona Justice Project. In that deposition I was asked whether I thought there was enough evidence in this case to say that Steven Witt was a victim of homicide. My answer is an unequivocal "No." I believe that his father, Drayton Witt, has been convicted of murder on insufficient grounds.

This affidavit was accompanied by an affidavit from Dr. A. L. Mosley, the medical examiner who conducted the autopsy.[51] Based on the triad, Dr. Mosley concluded in his autopsy report that the cause of death was "Shaken/Impact Syndrome" and the manner of death was "homicide," and he testified to this effect at trial. In his affidavit, Dr. Mosley states:

> Since I conducted Steven's autopsy and reached a conclusion that he was a victim of SBS, there have been significant developments in the medical community's understanding of SBS, most of which serve to undermine the reliability of the SBS diagnosis.
>
> There is no longer consensus in the medical community that the findings I reported in my autopsy report are reliable proof of SBS or child abuse. It is now understood that multiple conditions other than shaking can, through

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[51] Declaration of A. L. Mosley, M.D., *State of Arizona v. Drayton Shawn Witt*, Feb. 3, 2012, pgs. 2-3.

> their impact on multiple body systems, create the very symptoms and injuries once thought to be nearly exclusively attributable to SBS. . .
>
> Based on my review of these materials from an expansive body of post-2000 SBS literature, as well as the significant developments in the medical and scientific community's understanding of SBS and several of the conditions that mimic its symptoms, I have determined that I cannot stand by my previous conclusion and trial testimony that Steven Witt's death was a homicide. Steven had a complicated medical history, including unexplained neurological problems. He had no outward signs of abuse. If I were to testify today, I would state that I believe Steven's death was likely the result of a natural disease process, not SBS.

Even more recently, Senior Deputy Medical Examiner James Ribe of the Los Angeles Coroner's Office has broken ranks with his own office and concluded that abuse was misdiagnosed in the *Shirley Ree Smith* case (addressed above).[52] At a March 16, 2012 pathology conference, Dr. Colin Smith, a senior pathologist at the University of Edinburgh, advised that while doctors usually inferred shaking from the presence of the triad, there was growing evidence that other conditions (including seizure and/or breathing obstruction) could cause the same findings.[53] He noted that since doctors were trained to diagnose shaken baby syndrome until proven otherwise, they "may have difficulty accepting alternative explanations."

D. Additional Affidavits from Fact Witnesses

The affidavits and expertise of the medical experts who have received the evidence in this case and provided expert affidavits demonstrating Randy's

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[52] Joseph Shapiro and A.C. Thompson, NPR (Morning Edition), ProPublica and PBS *Frontline, New Evidence in High-Profile Shaken Baby Case* (March 29, 2012) www.npr.org/2012/03/29/149576627/new-evidence-in-high-profile-shaken-baby-case; Carol J. Williams, *New reports suggest wrongful conviction in 'Shaken Baby' case,* Los Angeles Times (March 29, 2012).

[53] Melissa Davey, The Sydney Morning Herald, *Doctor queries shaken baby symptoms* (news report), Presentation by Dr. Colin Smith at the Royal College of Pathologists of Australasia (March 16, 2012).

innocence are the best source of evidence for this Court to consider, but it is also relevant that these experts' findings are supported by the affidavits of fact witnesses.

1. Affidavits from Randy Liebich.

Randy's observations on Steven's activity level and the events of February 2-8 in his affidavits are consistent with the information that he provided to the doctors, the police and his attorneys at the time of Steven's collapse and shortly thereafter. In his 2009 affidavit, Randy describes Steven as follows:

> That week, Steven was quieter than usual. I thought he was a little jealous or depressed because the baby was getting a lot of attention. He also didn't seem to feel well. He whined and cried more than usual that week, often for no reason. He was always a little slow moving, but this week he was slower than usual. The last couple days, he wouldn't eat unless Kenyatta almost made him eat.

(Ex. 10, Randy Liebich Aff. at ¶ 10.) His description is consistent with his statements to the police at the hospital immediately after Steven's collapse:

> Randy related that Steven hadn't been eating, he wasn't saying much, and he was not as active as he usually was. It was indicated that he still moved around and played but not as much.

(Ex. 27, DuPage Co. Sheriff's Reports at MR 13.) It is also consistent with Kenyatta's descriptions of Steven crying for no reason and refusing to eat on the evening of February 7. It is also a classic description of a child with myocarditis and abdominal injuries/infection.

Randy's description of Kenyatta's treatment of Steven is also consistent with the descriptions provided by others:

> I did not like how Kenyatta treated Steven on his visits. She often hit him, sometimes cuffing him on the head, hitting him with broken plastic hangers, pushing or throwing him, or poking him in the head or stomach with her fingers. Kenyatta was very upset about how her stepfather treated her siblings but seemed to think that how she treated Steven was normal. She would not let me interfere.

(Ex. 10, Liebich Aff. at ¶ 5.)

Randy's description of the police efforts to get him to provide an explanation for Steven's injuries, reflected in his supplemental affidavit attached as Exhibit 11 to this Petition are virtually identical to those described by Judge Posner in *Aleman.* Randy explained that during over seventeen hours of interrogation, he was repeatedly accused of hurting Steven, and he repeatedly told police that he had not harmed Steven--there was nothing more he could tell them. (Ex. 11, Randy Liebich Supp. Aff. at ¶¶ 11-12.) His descriptions of what occurred largely track the official police version of events. Police reports reflect that during those interrogations police insisted that they knew he had caused the injuries and that he needed to explain them:

> Randy was advised of the medical evidence against him. That we knew he was responsible for the tragedy to Steven. That he was at the apartment by himself with the children. That he probably didn't think the injuries Steven sustained were as serious as they were. How this could have been an accident on his part and that he didn't intend for this to happen. That he was the only one who could help himself. Randy would sit and listen for long periods of time and on occasion when the subject of harming Steven was brought up he would say that he'd never hurt Steven like that. Randy would say that he loved Steven and wouldn't do that to him.

(Ex. 27, DuPage Co. Sheriff's Police Reports at MR 72.) Randy, an uneducated layperson, was in no position to explain to authorities Steven's pre-existing, undiagnosed medical conditions (including myocarditis and a preripancreatic

hematoma with scarring), his more recent medical conditions (ischemic bowel leading to pancreatitis, a hypoxic brain and a secondary coagulopathy), Rush Hospital's mistaken identification of a large subdural hematoma, or the true cause of Steven's symptoms. Ultimately, however, Randy was arrested and convicted because he could not explain complex medical processes that doctors did not yet understand.

2. Affidavit Concerning Kenyatta Brown.

As the new medical evidence described *supra* makes clear, Kenyatta Brown is the obvious suspect in Steven Quinn's death. It is therefore not surprising that Kenyatta Brown has declined to assist Randy Liebich in the present petition. Despite that, however, she confirmed in a recent interview that she never saw Randy hit or discipline Steven, and further stated that Steven had complained of stomach pain a few days before his collapse. (Exhibit 13, Lilly Aff. at ¶ 12.)

3. Affidavit of Dion Liebich.

In his 2009 affidavit, attached as Exhibit 12 to this Petition, Dion Liebich, Randy's cousin, describes Randy's interrogation by his cousin, Robert, at the Roselle Police Department. This affidavit contradicts Robert Liebich's trial testimony, described *infra*, that Randy Liebich admitted during this interview that he struck Steven on February 8. This interview occurred the day after Randy had exercised his *Miranda* rights and told DuPage authorities that he did not want to speak with them. In Dion's affidavit he describes going with Randy to see their mutual cousin,

Robert Liebich, a Roselle police officer, to address accusations that Randy was somehow "fleeing" authorities - in fact, Randy was staying with his parents.

When Dion and Randy came to the station, Robert proceeded to interrogate Randy. Dion describes the interrogation as follows:

> Robert took us in an interrogation room. Robert was very angry, and there were a lot of raised voices. There were no Miranda warnings of anything like that. This was a full interrogation, with a lot of anger directed at Randy. Robert and I wanted answers on how Steven died, and Randy wasn't giving us answers. Robert and I had kids, and we were angry with Randy for letting this happen to child. We thought he had to know what happened since he was there.

(Ex. 12, Dion Liebich Aff. at ¶ 5. In this interrogation, Randy illustrated for Robert how he had struck Steven in his efforts to release his finger when Steven choked on the hot dog:

> Robert questioned Randy on how he struck Steven, and Randy illustrated. He showed a tapping from a few inches away that should not have hurt anyone. It was almost like a push, more pressing than hitting. Robert went over this with Randy several times. Randy's description seemed like a natural instinctive reaction, exactly what I think most people would have done if someone was biting their finger. It did not seem like anything that would have hurt Steven. . . .
>
> Robert asked Randy over and over if he could have hit Steven hard enough to hurt him or kill him. Randy always said no . . . He kept saying that he didn't know what happened to Steven. Robert and I were angry that a three year old had been killed and no one could give an explanation.

(*Id.* at ¶¶ 7, 15.) Dion states that "Randy talked about Steven not feeling well the week before he collapsed" (*Id.* at ¶ 12), however, this is not included in Robert's report on the interrogation. Dion also describes a later visit by detectives:

> Months after Steven died, detectives came to my home. They told me that Randy had hit Steven on the head so hard that his brain swelled and there was bleeding on his brain, and that this was the only explanation for his

> injuries. Since I was present when Robert interrogated Randy and heard what Randy had to say, I thought that Randy's attorneys would also want to talk to me, but they never did.
>
> . . . In my gut, I always believed, and will always believe, that Randy is innocent. I couldn't see Randy hurting a child or not telling what he had done, even when interrogated. The only problem was that he couldn't explain why the child died.

(*Id.* at ¶ 22.) Finally, Dion describes another incident in which Kenyatta was rough with Steven:

> When I visited Randy . . it was over ninety degrees in their apartment, and Kenyatta was frying chicken in the kitchen. It was much too hot for the children, so I went out and bought an air conditioning unit and put it in the window. When Steven went over and started to play with the knobs, Kenyatta grabbed him by the left arm, opened the door to the bedroom and chucked him in, like she was throwing a baseball.
>
> I saw this from the kitchen table, and I jumped up and started going after Kenyatta, saying "how could you do this?" Randy got in the middle, and I yelled at him too, saying "how can you let this happen?" I told both of them I would call the department of child services if I ever saw or heard about anything like this again. I told my wife about this after I got home.
>
> Earlier, Randy told me that Kenyatta would use broken coat hangers to hit Steven. I didn't know why Randy didn't stop it but I don't think he thought he could. Kenyatta was a strong personality, and I don't think she would have listened to him. I was mad with Randy because I felt he was condoning it by staying with her. However, it may have been worse if he left.

(*Id.* at ¶¶ 17-19.)

4. Affidavit of Marlene Szafranski.

Marlene Szafranski, Randy's aunt, was the officer manager at Carlene Research, and has completed an affidavit attached to this Petition as Exhibit 16. Mrs. Szafranski had hired Kenyatta to do part-time consumer research at the Yorktown Mall. Mrs. Szafranski describes Randy as being excellent with children

(including her own son and nephew, with whom he spent a great deal of time) and getting along very well with Steven. For example:

> If Randy had Steven for the day, he would sometimes call me the night before and ask me to bring children's videos to the office. He and Steven would watch videos together. Randy would pick up food for Steven and eat with him, then he would take Steven out in the mall to walk around and ride the train. Randy was good with children, and he and Steven got along very well.

(Ex. 16, Szafranski Aff. at ¶¶ 6-7.) Mrs. Szafranski had only one problem with Kenyatta at work:

> I only had one significant problem with Kenyatta at work. Since I took weekends off, I assigned Kenyatta to work with Erin on a couple weekends. After one of the weekends, Erin told me that she wouldn't work with Kenyatta when I wasn't there because she didn't like Kenyatta hitting Steven.

(*Id.* at ¶ 8.) She also described an incident between Kenyatta and Randy's mother:

> I was concerned about Randy's relationship with Kenyatta because she wasn't mature and didn't seem ready to settle down. Much later, my sister Linda (Randy's mother) told me that when Randy and Kenyatta lived with her, Kenyatta had pushed her and caused [her] to hit her head and lose consciousness. Linda told me that she didn't want to make a report but insisted that Kenyatta move out. I believe that Linda told Randy's attorneys about this incident.

(*Id.* at 11.)

Like the police, the doctors and the rest of the family, Mrs. Szafranski thought that Randy should be able to explain Steven's injuries:

> When Randy was in jail I visited him and told him that I needed to know what happened to Steven. I told him words to the effect of, "you're not saying that you did it, you're not saying that Kenyatta did it, and this doesn't make any sense. Since you were there, you have to know what happened, and you need to tell us." He just kept saying that he didn't know. I told him that the family didn't see how he couldn't know since he was there.

(*Id.* at 13.) However, Mrs. Szafranski didn't think what the police were saying made sense either; she felt that something was missing. And she was right: what was missing were the pre-existing conditions, the abdominal injuries/infection in the days before the collapse, and the ensuing hypoxic brain and coagulopathy.

5. Affidavit of Denise Foster.

In her first affidavit, attached as Exhibit 14 to this Petition, Randy's sister Denise, a daycare worker at a health club, stated, as she had at trial, that Kenyatta regularly hit Steven, sometimes to make him stop crying. She also described the incident with her mother:

> The incident with my mother caused me real concern for Steven. We already knew that Kenyatta regularly hit and shoved Steven. My concern was that she would do with Steven as she had done with my mother, that is, push him, or shove him, or slam him into something, with serious consequences. The incident with my mother, who was not a fighter, showed me that Kenyatta did not have much self-control and that she did not think of the consequences of her actions.

(Ex. 14, 3/30/12 Foster Aff. at ¶¶ 3, 6.) In her second affidavit, Denise also describes visiting Randy, Kenyatta, Steven and Angelique shortly before Steven's collapse:

> My aunt and I brought McDonald's food to the apartment, but Steven refused to eat.
>
> I went into the bedroom and tried to talk Steven into eating, but he still refused.

(Ex. 15, 4/3/12 Foster Aff. at ¶¶ 3-4.)

6. Affidavit of Debra Minucciani.

Like Denise Foster, Debra Minucciani has submitted an affidavit (attached as Exhibit 17) in which she describes the same visit as Denise Foster:

> Approximately three days before his death [collapse], my niece, Denise Foster, and I visited Randy, Kenyatta, Steven and newborn daughter Angelique at their Willowbrook apartment.
>
> When we arrived, Steven appeared to be whining and crying for no reason.
> . . .
>
> We offered Steven some of the food from McDonald's, but he refused to eat it . . .
>
> I do recall an occasion in which I was riding in the car with Kenyatta and Steven and Kenyatta slapped Steven on the leg because he was crying.

(Ex. 17, Minucciani Aff. at ¶¶ 2, 3, 5, 7.)

7. Affidavits from Trial Counsel.

In affidavits attached to this Petition as Exhibits 18 and 19, Randy Liebich's trial counsel both aver that at no time in their representation do they remember advising, or remember the other person advising, Mr. Liebich that he had a right to testify at trial. (Ex. 18, Holman Aff. at ¶ 3; Ex. 19, Casey Aff. at ¶ 3.) Both also averred that they reviewed their notes and neither have any notes reflecting that Mr. Liebich was advised of his right to testify. (*Id.* at ¶ 4.)

V. CONSTITUTIONAL CLAIMS FOR RELIEF

COUNT ONE: NEWLY-DISCOVERED EVIDENCE ESTABLISHES THAT RANDY LIEBICH IS ACTUALLY INNOCENT

Petitioner re-alleges all of the earlier sections of this Petition and expressly incorporates them as if they were fully set forth herein.

Randy Liebich is actually innocent of the murder of Steven Quinn. It is well-established that Illinois has no interest in wrongfully incarcerating innocent persons. Procedurally, doing so "would be fundamentally unfair." *People v.*

Washington, 171 Ill.2d 475, 487 (1996); see also U.S. Const. amends. V, XIV. Substantively, imprisoning the innocent would be "so conscience shocking as to trigger the operation of substantive due process." *Washington*, 171 Ill.2d at 487-88; see also U.S. Const. amends. V, XIV.

Thus, a defendant who is actually innocent of the offense for which he stands convicted may bring a free-standing claim of actual innocence, seeking reversal of his conviction. To prevail, the defendant must present supporting evidence which is new, material, and non-cumulative, and which would probably change the result on retrial. *Washington*, 171 Ill.2d at 489. "New" evidence is "evidence that has been discovered since the trial and that the defendant could not have discovered sooner through due diligence." *People v. Ortiz*, 235 Ill.2d 319, 334 (2009). Randy Liebich presents such evidence in this Petition.

Steven Quinn died in February 2002. In the decade since his death, there have been dramatic changes in the understanding of pediatric head injury. In February 2002, it was widely believed that brain swelling in children represented the violent tearing of axons (the nerve fibers that connect the cells of the brain); that subdural hemorrhage in children represented the violent rupture of bridging veins (the veins that connect the brain to the large veins, or sinuses); and that retinal hemorrhages in children represented the violent rupture of retinal veins (the veins in the back part of the eye). By definition, each of these findings could only be caused by extreme violence. Now, it is understood that all three findings have a wide array of causes, including natural causes.

In 2002, however, it was common for doctors to diagnose shaking or abuse if a child presented with a subdural hemorrhage and brainswelling and the parents could not provide a history of trauma equivalent to a motor vehicle accident or major fall. The fact that Steven's CT scan was misinterpreted as showing a large subdural hemorrhage made matters worse, as did the appearance of strange marks and lines that appeared on his body shortly after hospital admission. The confirmation of severe brain swelling at surgery was interpreted to mean that the axons were torn throughout the brain. The lab tests became secondary: instead, the focus was on who had beaten him, and when. And since no one can eat, drink or interact with a brain that consists of torn axons and torn bridging veins that are dumping blood in the area outside the brain, the damage must have occurred immediately before hospital admission, and it must have been inflicted by the person who was with the child when he collapsed.

In 2012, however, these underlying assumptions have been disproven. We now know that brain swelling is not caused by torn axons: it is caused by lack of oxygen to the brain from any cause. We also know that thin subdural hemorrhages do not represent torn bridging veins; instead, they appear in a wide array of settings, ranging from trauma to natural causes. In this new context, why Steven died is a preliminary question that must be answered before resolving who might have caused his death. The answer to this question is contained in the objective medical evidence: the lab reports, the meticulous recording of the marks that

appeared and disappeared at the hospital, and the autopsy findings, all of which can now be correctly interpreted using advancements in medical science.

The reviews by doctors in several specialties produced uniform results: Steven died from abdominal injuries/infection that preceded hospital admission by at least two days, with no evidence of a head injury or a beating. While it is not possible to determine the precise course of events, it is possible to determine the approximate timing of when the abdominal injuries began and to roughly reconstruct their progression.

Based on the new evidence, we have learned that Steven had two predisposing conditions: myocarditis (damage to the heart), which would have made him react disproportionately to trauma or infection, and peripancreatitis (an encapsulated hematoma with scarring) that occurred before he arrived at Randy's and Kenyatta's on February 2. These were not addressed – or known – at the time of trial. On or about February 4-6 (and no later than February 7), some event or events occurred that caused a forehead bruise and damage to the bowel, mesentery, diaphragm and liver. By February 8, Steven had developed pancreatitis, a hypoxic brain and a secondary coagulopathy (bleeding/clotting disorder), all of which were confirmed by CT scan, lab tests and surgery. There is no evidence of head trauma and no evidence of a beating: instead, the hypoxic brain and the marks and lines that appear throughout the hospital stay are typical of infection/inflammation and a secondary coagulopathy, specifically DIC, which causes bleeding and thrombosis.

While Randy was present at the end of this process, he was not present at the beginning.

Since the appropriate time period – the days and weeks before Steven's collapse – was never investigated, we are unlikely to be able to determine with certainty what happened in the weeks before Steven's collapse to cause the damage to the heart, the peripancreatic hematoma or the newer abdominal injuries/infection. If the trial judge was correct that Kenyatta was not abusive, the injuries are likely due to an accident that passed unnoticed at the time and/or a natural disease process. With these factors in mind, we briefly review the new evidence supporting his innocence.

A. New Scientific Evidence Demonstrates that Steven Quinn Had a Peripancreatic Injury that Pre-Dated Randy Liebich's Care

New evidence in the form of expert affidavits from numerous medical professionals establishes that Steven Quinn had a peripancreatic injury that predated the time that Randy Liebich watched Steven on February 8. Because this injury predated February 8, this evidence is new, material, non-cumulative, and would likely change the result on retrial.

At trial, Randy was only able to present a few pieces of evidence to support his theory that Steven was injured before he was in Randy's care. First, he was able to point to the few symptoms Steven had earlier, including a runny nose and a cough on February 5. See RA at 43, 119. He also was able to present evidence from Dr. Teas, who testified that Steven's injuries were caused in an earlier time period.

This evidence would have been viewed completely differently when accompanied by the new scientific understanding about infant head injuries that Randy presents with this Petition. Randy has set forth a detailed description of the advancements in medical understanding in this area, and it is this new medical understanding upon which his new affidavits rely to reach new medical conclusions.

First, Dr. Barnes explained that for many years the medical community did not understand and accept that swollen brains in infants reflected lack of oxygen to the brain rather than traumatic injury, and that thin subdural hemorrhages were not necessarily indicators of traumatic death. These principles did not become widely accepted in medical literature until 2008-11. (Ex. 1, Dr. Barnes. Aff.) That means that at the time of Randy's trial, the medical witnesses, the Court, and the lawyers for both sides could not have understood that Steven's injuries were not consistent with shaken baby syndrome, but rather a stomach injury. In addition, the medical community, the Court, and counsel also did not understand that there can be lucid intervals of up to 72 hours for injuries that cause brain swelling, meaning that brain swelling is not always an immediate symptom. This outdated scientific understanding also played a huge role in this trial, because the Court could not have had the evidence before it to avoid the conclusion that because Steven was symptomatic at the hospital, those symptoms must have been based on an immediately recent injury.

Because Dr. Barnes examined the medical evidence in this case free from the outdated assumption that brain swelling indicates brain trauma, he was able to

opine that Steven's CT scan was consistent with a diagnosis of abdominal injury/infection with systemic inflammatory response syndrome and multiple organ dysfunction syndrome (*Id.*) He was able to explain Steven's brain findings and thin hemorrhages as a result of thrombosis (abnormal clotting) and not as a result of trauma caused by Randy striking Steven. (*Id.*) Dr. Barnes was also able to refute the testimony at trial that the "fresh" color of the blood indicated a recent injury - Dr. Barnes explained that this has no medical or scientific basis, and that "fresh" blood may be present when evidence of older injuries or processes cannot be seen in a CT or by the naked eye. (*Id.*) In short, Dr. Barnes provides critical new evidence that Steven's injuries had a lucid interval, and that these injuries were not caused by blunt trauma or a fall. (*Id.*)

Dr. Laposata, an expert pathologist and coagulation expert, agrees, explaining that Steven mostly likely had an ischemic bowel that progressed to peritonitis, pancreatitis, and hypoxic bowel (Ex. 2, Dr. Laposata Aff.) Steven's ischemic bowel was natural in origin, and Dr. Laposata explained that the answers about the timing and cause of the ischemic bowel would be best found by reviewing the autopsy slides. (*Id.*) Dr. Laposata explained that the timing of the development of ischemic bowel would likely have "evolve[d] over a period of days" as opposed within eight hours of hospitalization, which was the prosecution's theory at the time. (*Id.*)

Dr. Laposata explained that the answers lay in the slides, and Dr. Mileusnic, who did testify at Randy's trial, provides that new opinion after re-reviewing her

original analysis. (Ex. 3, Dr. Mieusnic Aff.) Her re-review, which would not have been available to Randy at the time of trial because it was based on the new medical developments in the understanding of child head injury, concludes that Steven's abdominal injuries occurred prior to February 8, and are consistent with even just a "push, shove, or inappropriate punishment." (*Id.*) She also identified for the first time myocarditis (damage to the heart) which would have reduced Steven's circulation and increased his susceptibility to trauma or infection (*Id.*) Based on her review, "There is no evidence of head trauma occurring on February 8 and no evidence of inflicted head trauma occurring earlier," a medical finding which completely undercuts the State's theory and the testimony of the medical doctors who opined that Steven's death was caused by a recent head injury that only Randy could have inflicted. (*Id.*) Like the other medical experts who have reviewed the case, she concludes that the pancreatitis and hypoxic-ischemic brain "are a natural progression of earlier injuries" and "do not suggest trauma occurring on the day of hospital admission (*Id.*) These conclusions exclude Randy Liebich as the cause of Steven's injuries and death.

Other experts are all equally clear that these injuries were old. Dr. Stephens explains in his affidavit that while it is not possible to determine the origin of the abdominal infection - it could have been caused by a natural process or by pressure from a fall, accident, or push - "it began no later than February 6 (and likely earlier)." (Ex. 6, Dr. Stephens Aff.) Dr. Stephens is clear - there is no medical evidence of injuries on the day of admission, there are no specific indicators of head

trauma, and there is no scientific way to determine the origin of the abdominal infection (*Id.*) Again, these conclusions would have had a clear impact on Randy's trial.

Most striking is that Randy's actual innocence claim is not based on the testimony of one or two medical experts, but on the corroborated conclusions of experts from various parts of the medical field who all agree that Steven died of an old stomach injury. Dr. George Nichols, a pathologist, agrees with the above experts (Ex. 5, Dr. Nichols Aff.) as do Dr. Ronald Uscinski, a neurosurgeon (Ex. 8, Dr. Uscinski Aff.), Dr. Waney Squier, a pediatric neuropathologist (Ex. 7, Squier Aff.), and as does Nathan Felix, a military expert on trauma cases. (Ex. 9, Felix Aff.)

Finally, Randy Liebich presents a new affidavit from Dr. Shaku Teas. She testified for the defense at trial that the timing of Randy's injuries were at least five days old, but was not able to testify with the benefit of the medical advancements in this area. Her testimony was misstated in the closings and the Court viewed her testimony as agreeing that Steven's injuries could have occurred on February 8. In her affidavit, she also confirms what these other experts have opined about new scientific advancements that support her initial conclusions about the timing. (Ex. 4, Dr. Teas Aff.) At trial, Dr. Teas appeared to be in the vanguard, the sole witness who would provide evidence of a timing on Steven's injuries from days before. In fact, her initial conclusions are squarely within the mainstream and supported by the medical establishment. Her testimony does not render this new evidence

cumulative; instead, it would have caused her trial testimony to be believed and properly understood.

B. New Affidavits from Fact Witnesses Confirm the New Scientific Evidence

The affidavits of these medical experts are confirmed by new affidavits from fact witnesses, who provide evidence that Steven had symptoms of a peripancreatic injury days before he was admitted to the hospital.

Randy's affidavit explains that in the days before Steven's death, he was "quieter than usual," didn't want to eat, and "didn't seem to feel well." (Ex. 10, Randy Liebich Aff.) He provided this affidavit in 2009 before these experts gave their opinions. Although this affidavit might be viewed as the self-serving statements of a defendant, it is non-cumulative and material because it demonstrates all the symptoms that Steven would have been experiencing as a child with abdominal injuries/infection and myocarditis. Although these experts did not rely on Randy's affidavit in reaching their conclusions, it and the medical affidavits are mutually supportive. This affidavit explains why Randy Liebich should have testified in this case. Had Randy testified, he would have provided believable and credible testimony that the Court would have considered because it would have been supported by neutral medical experts.

Randy's observations are also confirmed by others who saw Steven in the days before his death. Even Kenyatta, who is a likely suspect in Steven's death given the timing of these injuries, admitted to an investigator that Steven was not feeling well in the days before his death. (Ex. 13, Lilly Aff.)

Randy's cousin Dion Liebich related that during his interview with Robert Liebich, Randy told Robert that Steven hadn't been feeling well in the week before his collapse. (Ex. 12, Dion Liebich Aff.) Dion himself also witnessed an earlier incident in which Kenyatta threw Steven into a bedroom (*Id.*) Again, while standing on its own at the time of trial this testimony might have seemed self-serving, when supported by the new medical evidence it is credible. It is also consistent with the testimony of other people who saw Steven - Denise Foster and Debra Minucciani both saw Steven shortly before his collapse, and both recalled that Steven was refusing to eat. (Exs. 14 & 15, Foster Affs., and Ex. 17, Minucciani Aff.) Collectively, numerous fact witnesses provide support for the fact that Kenyatta on numerous occasions had treated Steven roughly, in a manner that could have caused his stomach injuries.

C. This New Evidence Entitles Randy to a New Trial

Collectively, this evidence of Randy's innocence, evidence of the timing and cause of Steven's injuries, entitle Randy to a new trial. This evidence is new, material, and non-cumulative of the limited defense Randy was able to present back in 2004. He could not have discovered it earlier since medical science did not advance to accept these principles until well after Randy's conviction. It would also probably change the result on retrial. At trial, Randy was not able to present a convincing alternative explanation for the Court about when Steven's injuries were caused, and how they were caused. The Court was left to accept the testimony of the treators and hospital doctors who had seen Steven and erroneously concluded that

he was suffering from a recent head trauma. At a new trial on this matter, Randy would be able to demonstrate that their testimony is outdated and medically incorrect. Randy is entitled to a new trial to allow the Court to fully consider this new medical evidence.

COUNT TWO: TRIAL COUNSEL PROVIDED INEFFECTIVE REPRESENTATION TO RANDY LIEBICH

Petitioner re-alleges all of the earlier sections of this Petition and expressly incorporates them as if they were fully set forth herein.

Both the Illinois Constitution and the United States Constitution guarantee criminal defendants the assistance of legal counsel. Those rights are violated where the attorney's representation "fell below an objective standard of reasonableness" and there is a "reasonable probability that, but for counsel's unprofessional errors, the result of the proceedings would have been different." *Strickland v. Washington*, 466 U.S. 668, 688 (1984); Ill. Const. Art. I sect. 8. A reasonable probability is a "probability sufficient to undermine confidence in the outcome." *Id.* In Randy Liebich's case, his counsel was constitutionally deficient in several ways, which prevented the trial court from being able to consider all the evidence in the case. These deficiencies, both individually and cumulatively, affected the outcome of Randy Liebich's trial.

A. Randy Liebich's Trial Counsel was Ineffective for Failing to Counsel Randy About His Right to Testify

Petitioner re-alleges every paragraph of this petition and expressly incorporates them as if they were fully set forth herein.

A criminal defendant has an absolute right to testify on their own behalf. *People v. Brocksmith*, 162 Ill.2d 224, 227 (1994); *People v. Thompkins*, 161 Ill.2d 148, 177 (1994). That right stems from the Fifth, Sixth and Fourteenth Amendments to the United States Constitution. *Rock v. Arkansas*, 483 U.S. 44, 51-53 (1987). Trial counsel can provide ineffective assistance by giving "incomplete or inaccurate information to the defendant regarding the defendant's right to testify." *People v. Nix*, 150 Ill. App. 3d 48, 51 (3d Dist. 1986). Because this right belongs exclusively to the defendant, it "is not one of those matters which is considered a strategic or tactical decision best left to trial counsel." *People v. Seaberg*, 262 Ill. App.3d 79, 83 (2d Dist. 1994).

Here, Mr. Liebich wanted to testify but did not do so because, although he wanted to testify, his trial counsel refused his request and never informed him that he had a right to do so.

In his March 3, 2009 notarized petition, Randy asserts that he was never informed of his right to testify in his defense. He further asserts that his lawyer specifically denied his request to testify. (Ex. 29, March 3, 2009 *Pro Se* Post-Conviction Pet. at 6-7.)

In affidavits attached to this Petition, Randy's trial counsel, Ricky Holman and John Casey admit that neither of them ever recollect informing Randy of his right to testify on his own behalf. (Exs. 18 & 19, Holman & Casey Affs.)

The Defendant wanted to testify to correct factual errors in the evidence that was given to the court. For example, Kenyatta testified that she never disciplined

Steven, other than to spank him on his buttocks or "muff" his head. In fact, Randy wanted to tell the court that he had personally observed Kenyatta strike Steven numerous times, in places other than what she described. Randy also wanted to inform the court that he had not ingested PCP with Kenyatta, as she described. Randy also wanted to make it clear that he never told the police that he encouraged Kenyatta to hit Steven harder. Finally, had he testified, Randy could have explained the gesture he made with regard to attempting to get Steven to release his bite on Randy's fingers, during Randy's meeting with his cousins Dion and Robert at the Roselle Police Department.

Randy claims with regard to the denial of his right to testify are similar to other cases that the Illinois courts have deemed to be judicable. *See People v. Piper*, 272 Ill. App. 3d. 843, 848 (5th Dist. 1995) ("[T]he allegations pled by petitioner here are factual and not conclusory and are sufficient to state a meritorious claim of a substantial deprivation of a constitutional right: '[Petitioner] asked his trial lawyer to allow him to testify. Counsel told the defendant that he could not testify. Defendant did not know that he could bring this matter to the attention of the trial judge.'").

Randy wanted to testify because he feared that, were he not to testify, his statements to Robert would be misconstrued. Randy was right: his failure to testify and inability to explain adverse testimony played a major role in the court's decision. In addition to key points that were interspersed throughout the decision, the Court spent 4-5 pages of her decision specifically addressing "information that

was contributed by the defendant" via the state's witnesses, to which Randy could not respond. Had he testified, the trial court's findings would have been much different. See *Piper*, 272 Ill. App. 3d at 849 ("At this point in these postconviction proceedings, we are not in a position to find harmless error, especially given the importance of a defendant's fundamental constitutional right to testify in his own behalf at trial. At this stage of the proceedings, we are unaware of what defendant's testimony at his trial might have been and cannot evaluate how that testimony might have impacted the jury.")

B. Randy Liebich's Counsel Was Ineffective for Failing to Present Key Evidence That Would Have Undercut the State's Case

The State prevailed at trial because they were able to convince the Court that Steven Quinn died as a result of injuries sustained on February 8 when Steven was in Randy's sole care, and because the Court believed that it was Randy who had caused Steven's injuries. Despite knowing this was the State's theory, Randy's trial counsel failed to present testimony from witnesses who could have undercut that evidence and established to the Court that these propositions were untrue. Trial counsel's failure to do so cannot be attributed to any trial strategy, and altered the outcome of the trial.

First, as discussed in detail in Part A above, counsel failed to call Randy Liebich. Randy Liebich was willing--and in fact wanted--to testify on his own behalf. He would have been able to testify and refute much of the evidence presented against him, and to provide a compelling counterstory to the State's evidence. Since this was a bench trial, there was far less risk of juror prejudice against Randy.

Despite this, counsel failed to call Randy. Had they done so, the outcome of the trial would have been different.

Second, counsel failed to ask witnesses Denise Foster and Marlene Szafranski the right questions. As demonstrated by their attached affidavits, attached to this Petition as Exhibits 14, 15 & 16, and as discussed with respect to Mr. Liebich's actual innocence claim above, both Ms. Foster and Ms Szafranski could have provided evidence that Steven was sick earlier in the week, testimony that would have corroborated the defense's argument that Steven sustained his fatal injuries much earlier than February 8. These witnesses also would have provided further evidence that it was Kenyatta and not Randy who had a pattern of abusing Steven Quinn.

Third, and most importantly, counsel failed to call Dion Liebich. As demonstrated in his affidavit, attached as Exhibit 12, Dion Liebich would have supported Ms. Foster and Ms. Szafranski's testimony. More importantly, the Court found significant the perceived admissions that Randy Liebich made to Robert Liebich during their conversation, where Randy supposedly acknowledged that he had struck Steven in the past. Dion, however, was present for those conversations and could have clarified that Randy gave the police evidence which supported an earlier injury theory, and did not admit to mistreating Steven during that conversation. As it was, trial counsel's failure to call Dion or Randy left Robert Liebich's testimony on this issue unrebutted, and gave the impression to the Court that defense counsel conceded the accuracy of Robert Liebich's statements. Had

counsel rebutted that testimony, the Court would not have concluded that Randy Liebich incriminated himself, and the outcome would have been different.

Even if Randy was not in custody during his interrogation with Robert Liebich, no reasonable trial strategy can be shown for the decision not to call Dion Liebich, who was also present for the interrogation, and who could have explained that the hit that Randy administered to Steven was merely "a tapping from a few inches away that should not have hurt anyone." (Ex. 12, Dion Liebich Aff. at ¶ 7.) Not only did defense counsel not call Dion, they never even spoke with him, despite his presence in the police reports. Because counsel never investigated what Dion would say, there can be no argument that they made a strategic decision not to call him. *Strickland*, 466 U.S. at 690-91 ("[S]trategic choices made after less than complete investigation are reasonable precisely to the extent that reasonable professional judgments support the limitations on investigation. In other words, counsel has a duty to make reasonable investigations or to make a reasonable decision that makes particular investigations unnecessary."). *See also People v. Jacobazzi*, 398 Ill. App. 3d 890, 919 (2d Dist. 2009) (holding that where the failure to present exculpatory evidence is "the result of a lack of diligence in investigating the facts and law, rather than a drawback or strategy, counsel may be ineffective").

In addition, even though the defense suggested strongly that Steven's beatings were actually administered by Kenyatta, rather than by Randy, the defense failed to question Denise Foster about her being present when Kenyatta knocked Linda Liebich, Randy's mother, unconscious. This event was corroborated

by Marlene Szafranski, yet she was not asked about it. Nor was Linda called to testify about the incident.

A criminal defendant is entitled to effective assistance of counsel, and that entitlement includes the right to have exculpatory witnesses called at trial. Defense counsel was ineffective for failing to question these witnesses on this matter, as it pertained to a critical element of the defense that Kenyatta actually caused the injury to Steven. *See People v. Cabrera*, 326 Ill. App. 3d 555, 565-65 (3d Dist. 2001) ("When the record is unclear concerning whether trial counsel's decision not to call exculpatory witnesses was a matter of counsel's trial strategy or counsel's incompetence, the defendant is entitled to a postconviction evidentiary hearing on that issue.").

C. Counsel was Ineffective for Failing to Present Exculpatory Evidence and Argument

Randy Liebich's trial counselors were not able to effectively present his defense because they apparently did not understand the medical evidence against him. As a result, they did not elicit exculpatory evidence which would have changed the outcome of the case. Counsel can be ineffective for failing to understand scientific issues in a case and failing to take steps to address those scientific issues at trial. *See Richey v. Bradshaw*, 498 F.3d 344, 364 (6th Cir. 2007) (holding that trial counsel was ineffective for failing to consult their own experts who would have challenged the State's scientific theories); *Dugas v. Coplan*, 428 F.3d 317, 328 (1st Cir. 2005) (holding that trial counsel was ineffective where he "did not consult an expert in arson investigation or learn how to effectively use the terminology and

techniques of arson investigation from his own research"). The decision in *Richey*, which the Second District Court of Appeals cited with approval in *Jacobazzi*, specifically held that it would be ineffective for counsel to fail to appropriately use and understand an expert in a case involving scientific issues, because "the mere hiring of an expert is meaningless if counsel does not consult with that expert to make an informed decision about whether a particular defense is viable." *Richey*, 498 F.3d at 362-63 (cited in *Jacobazzi*, 2009 WL 3968849, at *24).

There are two particular ways in which this lack of understanding and accompanying ineffectiveness manifest itself in counsel's performance. First, Dr. Teas' post-trial letter to the trial court, which the trial court did not allow to be made part of the record after trial counsel agreed with the State's contention that it was untrue, revealed significant ways in which counsel erred. In Dr. Teas' letter she expressed dismay to the Court that trial counsel had failed to elicit important exculpatory information, including (1) questioning Dr. Mileusnic about her conclusions in the autopsy findings that *all* of Steven's injuries were at least five days old at the time she died, (2) questioning Dr. Teas, Dr. Mileusnic and the testifying treating physicians about the present of Tylenol and Aspirin in his blood as evidence that he had not been feeling well prior to February 8; (3) questioning Dr. Teas, Dr. Mileusnic and the treating physicians about Steven's significant weight loss sometime in the four months prior to his death; (4) questioning Dr. Teas, Dr. Mileusnic and the treating physicians about Steven's sedation and the effect this sedation would have had on the presentation of his abdominal injuries at

the hospital; and (5) correcting the record at trial about the size of the hemorrhage and other facts from the medical records. (Ex. 21, Letter from Dr. Teas to Judge Jorgenson.)

Dr. Teas' affidavit filed before this Court as Exhibit 4 reveals further evidence of ways in which trial counsel's misunderstanding of the evidence prejudiced Randy Liebich. Most importantly, the trial court's finding that Randy Liebich was guilty was based in large part on the perceived concession by trial counsel that Dr. Teas had admitted Steven Quinn's injuries could have occurred on February 8. As. Dr. Teas explains in her affidavit, she was not present for closing arguments or the judge's ruling, and was dismayed to find that the Court had interpreted her testimony in this manner because of trial counsel's concession. This concession reflects either (1) trial counsel's lack of preparation and understanding of Dr. Teas' testimony, or (2) trial counsel's complete ineffectiveness in arguing the case at closing, by allowing the critical issue (and defense counsel's strategy for pursuing a defense) to be destroyed.

Each of the concerns expressed by Dr. Teas in her affidavit and letter represent a critical piece of exculpatory evidence that counsel either left on the table at trial, or that counsel inexplicably brushed away through concessions at closing. Trial counsel's strategy was clearly to present evidence that Steven's injuries occurred before February 8, yet counsel failed to elicit numerous pieces of uncontroverted medical evidence that would help prove just that. And, even more inexplicably, at closing counsel conceded that the timing of the injuries could have

been on February 8 even though Dr. Teas' testimony explicitly rejected that possibility. These decisions had no possible strategic basis, and both individually and cumulatively would have changed the outcome of Randy's trial. *Jacobazzi*, 398 Ill. App. 3d at 928 ("[I]neffectiveness may be found even where defense counsel does not completely abdicate her duty but rather presents a coherent defense theory that . . . nonetheless lacks an essential element that counsel [unreasonably] omitted.").

D. Trial Counsel Was Ineffective for Failing to File a Motion to Suppress Randy Liebich's Statements to Robert Liebich

Trial counsel was ineffective for failing to move to suppress Randy's statement to Robert Liebich concerning the hit he gave Steven in an effort get Steven to release his bite on Randy finger. Although Mr. Holman casts this, without explanation, as a decision of trial strategy, no reasonable strategy can be gleaned from eliciting a defendant's inculpatory statement at trial.

That Randy suffered prejudice as a result of this statement is clear from the record: the trial court stated as much in its ruling. *See* RA at 24 (The court stated, "I still can see Robert Liebich sitting in that chair testifying, is that the defendant looked me in the eye and said, 'I didn't hit that kid hard.' To me that speaks volumes; that is the defendant's admission that he hit Steven Quinn.")

Had a motion been filed, it would have prevailed for, even if the interrogation was not custodial at the outset, it quickly became so. *See, for example, People v. Gorman*, 207 Ill. App. 3d 461, 470 (4th Dist. 1991) (stating that "even if a suspect goes to the police station voluntarily or at the invitation of the police, the circumstances may eventually become custodial in nature"). Dion Liebich, who was

never contacted by the defense, noted that, in addition to occurring in an interrogation room, the questioning of Randy "involved a lot of raised voices," with "a lot of anger directed at Randy." (Ex. 12, Dion Liebich Aff. at ¶ 5.) For his part, Randy describes the questioning as "an hour of heated interrogation," during which the door was closed, despite Randy's expressed desire to remain outside the building (Ex. 11, Randy Liebich Supp. Aff. at ¶¶ 16, 25.) Even Robert Liebich noted that he asked Randy about the hit to Steven "several times," and that he asked him to "swear on his father's grave." (Ex. 28, Robert Liebich Handwritten Report.)

Despite this custodial environment, Randy was never read his *Miranda* rights. *See Gorman* 207 Ill. App. 3d at 471 (stating that "police stations are typically the location of custodial interrogations, thereby giving credibility to a suspect's claim that he believed himself to be in custody despite police testimony about how they treated the suspect"). Since Randy was subjected to a custodial interrogation without being Mirandized, a motion to suppress those statements would have been granted, and trial counsel was ineffective for not filing one.

E. Trial Counsel Was Ineffective for Failing to Communicate Plea Offers to Randy Liebich

Although Mr. Liebich brings this Petition primarily to assert his innocence, he also raises an alternative claim that trial counsel was ineffective for failing to pursue a plea agreement. Recently, the United States Supreme Court observed that "plea bargains have become so central to the administration of the criminal justice system that defense counsel have responsibilities in the plea bargain process, responsibilities that must be met to render the adequate assistance of counsel that

the Sixth Amendment requires in the criminal process at critical stages." *Missouri v. Frye*, 2012 WL 932020 (2012).

On the record, attorney Holman stated that he rebuffed the State's attempts to make an offer by stating that it was always his intention to go to trial. *See* RA at 2422 (Holman stating "[t]here were times when the state would come up to me and indicate that we haven't talked about an offer. And my only reply was in exchange for dismissing the charges he would testify against the mother in the case. And that was obviously met with nonapproval, and we left it at that. It was our, we have always maintained to go forward to trial on this case.").

Although the State never made a formal offer in this case, Attorney Holman essentially precluded them from doing so by insisting that any offer short of dismissal would be rejected. Indeed, according to Randy, Mr. Holman informed him that he told the State "straight out no, we want to go to trial. We don't want to know what an offer is." *Id.* at 2423. Randy also stated that, contrary to Mr. Holman's representations, he was interested in and would have accepted any reasonable offer. *See id.* at 2424 (Randy Liebich stating "[f]acing the kind of time that I'm facing and that I received I would have been willing to take an offer had it been reasonable").

By failing to even listen to what the State's offer might possibly be, Mr. Holman failed his client at a critical stage of the trial proceedings and provided ineffective assistance of counsel. *See Frye*, 2012 WL 932020 at *6 (stating that, in today's criminal justice system, "the negotiation of a plea bargain, rather than the unfolding of a trial, is almost always the critical point for a defendant").

The Court's decision in *Frye* also directs that a petitioner in Randy's situation may show prejudice, the second required prong of *Strickland*, by demonstrating a "reasonable probability that the end result of the criminal process would have been more favorable by reason of a plea to a lesser charge or a sentence of less prison time" *Id.* at *9 (citing *Glover v. United States*, 531 U.S. 198, 203 (2001) ("[A]ny amount of [additional] jail time has Sixth Amendment significance.")). Here, as Petitioner set forth in this Petition and in his affidavit, he never received any offers from the State because counsel did not allow the State to make them, despite evidence that the State wanted to convey an offer.

Given the amount of time Mr. Liebich received, it is likely that an offer from the State would have cut his prison sentence significantly. Although this issue was addressed briefly before the lower court on the record, with prosecutors confirming they never made an offer to Mr. Liebich before trial, Mr. Liebich lacks the necessary information to present evidence of the prejudice he suffered. The Post-Conviction Act, however, directs that Mr. Liebich may still sufficiently plead prejudice by stating why the evidence in support of prejudice cannot be attached to the petition. 725 ILCS 5/122-2. Here, evidence of his prejudice is in the possession of the States Attorney's Office, and Petitioner seeks leave of the Court to obtain discovery disclosing what offers the State intended to make to Mr. Liebich's counsel so that Mr. Liebich can adequately pursue this claim.

COUNT THREE: RANDY LIEBICH'S APPELLATE COUNSEL WAS INEFFECTIVE FOR FAILING TO RAISE MERITORIOUS ISSUES ON APPEAL

Petitioner re-alleges all of the earlier sections of this Petition and expressly incorporates them as if they were fully set forth herein.

A claim of ineffective assistance of appellate counsel is cognizable under the Post-Conviction Hearing Act. *People v. Mackiel*, 167 Ill.2d 525, 531 (1995). Like ineffective assistance of trial counsel claims, they are governed by the *Strickland* test. *People v. Caballero*, 126 Ill.2d 248, 269-70 (1989). Randy must show that his appellate counsel's failure to raise a sufficiency challenge was objectively unreasonable, and that it prejudiced Randy on appeal. *People v. Rogers*, 197 Ill.2d 216, 223 (2001). His counsel's performance is judged by an "objective standard of competence" under prevailing professional norms, *People v. Simms*, 192 Ill.2d 348, 361 (2000), and should find prejudice where there is a "reasonable probability that, but for counsel's unprofessional errors, the result of the proceeding would have been different." *People v. Richardson*, 189 Ill.2d 401, 411 (2000). A "reasonable probability" is a probability "sufficient to undermine confidence in the outcome." *People v. Enis*, 194 Ill.2d 361, 376-77 (2000).

Petitioner believes that the claims raised in this Petition could not have been raised by appellate counsel on direct appeal because, although some of them are claims that were before the Court in some fashion through Randy Liebich's *pro se* post-trial motions, they each rely on evidence outside of the record on appeal and involve new evidence the trial court and the appellate court did not have before

them and could not have considered in reaching a ruling. *See, e.g., People v. Smith*, 406 Ill. App.3d 879, 984 (1st Dist. 2010) ("[M]atters outside the record are not appropriate for our review on direct appeal.") (citing *People v. Manning*, 334 Ill.App.3d 882, 893–94 (1st Dist. 2002).) However, to the extent that any of the claims asserted herein are deemed waived for failure to present them on direct appeal, Petitioner received ineffective assistance of appellate counsel. Additionally, fundamental fairness requires that this Court review any claims that may have been waived for failure to raise them on direct appeal. S*ee People v. Pitsonbarger*, 205 Ill.2d 444, 458 (2002).

COUNT FOUR: RANDY LIEBICH'S PROSECUTION VIOLATED HIS RIGHT TO DUE PROCESS

Petitioner re-alleges all of the earlier sections of this Petition and expressly incorporates them as if they were fully set forth herein.

As described throughout this petition, Randy Liebich's prosecution for the death of Steven Quinn when the State had before it evidence that Randy Liebich could not have committed the crime because of the timing of Steven Quinn's injuries, when the State had ample evidence of an alternative suspect, and when the State had no clear evidence of his guilt, violated his federal right to due process. *See, e.g., Ex parte Clarence Lee Brandley*, 781 S.W.2d 886 (Tex. Ct. Crim. App. 1989), *reh. den'd*, and cases cited therein.

COUNT FIVE: CUMULATIVE ERROR

Petitioner re-alleges all of the earlier sections of this Petition and expressly incorporates them as if they were fully set forth herein.

Even if individually the errors and other matters alleged here are not found to be sufficiently prejudicial to grant Randy Liebich post-conviction relief, the cumulative effect of all of the matters alleged in this petition deprived Randy Liebich of his fundamental due process right to a fair trial. Thus, this Court should grant Randy Liebich post-conviction relief in the form of a new trial.

VI. CONCLUSION

When Steven Quinn arrived at both Mount Sinai Hospital and Rush Medical Center, medical staff focused on his head CT scan. However, at trial, the possibility that Steven's brain injury actually resulted from a days-old abdominal injury that Randy could not have caused, was not presented. New medical evidence suggests, however, that that is precisely what happened.

At trial, Dr. Munoz testified that he observed a hemorrhage that was diffused "throughout the brain." RA at 1674. He stated that this is significant because "that speaks of a trauma to the whole brain, to the whole head, not just localized force being applied to any particular area." *Id.* It is highly unlikely that in administering a beating, a person would administer blows to a victim's entire head. Instead, in a beating case, one would expect there to be obvious signs of trauma in one or two places on the victim's body. But that was not the case here. Dr. Green was "a little stunned" at the lack of traumatic marks on Steven's body. *Id* at 1043. Dr. Severin agreed that none of the bruises on Steven's body "stood out more than any other ones." *Id.* at 1410. Dr. Green's initial thought was that Steven's condition was metabolic, rather than traumatic. To Dr. Severin, Steven looked "toxic," instead of

abused. *Id* at 1374. The likely explanation for the unlocalized hemorrhage that was observed by Dr. Munoz was not a beating, but hypoxia, or lack of oxygen which can lead to, as Dr. Severin agreed on cross, "diffused hemorrhaging throughout the brain." *Id* at 1421.

In short, the initial impression of doctors Severin and Green was correct. Steven Quinn *was* toxic. His illness *was* metabolic. Steven collapsed because an infection had been raging inside his body for days. The infection was caused by an abdominal injury that resulted in an ischemic bowel and leakage of bacteria into Steven's body. The infection reached a tipping point when Randy was watching Steven, and it caused Steven to choke or seize when he was eating the hot dog that Randy had prepared for him. It eventually reduced his oxygen supply, causing abnormal breathing, hypoxia, and gathered momentum at the hospital, which is why Steven's labs worsened between Mount Sinai and Rush hospitals. As Dr. Severin put it, Steven's pancreatitis was "rapidly progressing" so that by the time of Dr. Munoz's craniectomy, the brain was being deprived of oxygen, causing it to swell massively. *Id* at 1395. In short, Steven may have died from an injured brain, but that injury was the result of an abdominal injury that Randy Liebich could not have caused.

In critical respects, this case is remarkably similar to the recent Seventh Circuit decision in *Aleman v. Village of Hanover Park*, 662 F.3d 897 (7th Cir. 2011), written by Judge Posner. In *Aleman*, the child had been "lethargic and feverish" for several days before Mr. Aleman cared for him. *Id. at* 901. The doctors (and the

courts) eventually concluded that the child's subdural hematoma and collapse (the same findings as in this case) could have been the "delayed effect of . . earlier trauma rather than of anything Aleman had done." *Id. at* 902. And the child's mother, much like Kenyatta Brown, was "a violent person with a criminal record." *Id.* Most importantly, the court recognized that "[a]lthough the medical profession once thought that there is no interim between trauma and collapse in shaken-baby syndrome, the medical profession now believes ... that there can be an interim in which the child would be conscious, but probably lethargic or fussy or feverish or have difficulty sleeping or eating." *Id. at* 902-03.

Like the defendant in *Aleman*, Randy was especially good with children, including Steven. Also like *Aleman*, the victim's mother, who had watched the child in the days prior to his death, was known to be violent. Most significantly, as was true in *Aleman*, Steven Quinn was fussy and showing signs of illness in the days before his collapse.

The medical profession now understands, in a way that it did not during Randy's trial, that an abdominal injury caused days earlier can lead to a hypoxic brain (brain swelling) and death. Randy Liebich simply had the misfortune of being present when the effects of an abdominal injury that Steven suffered days before he was left in Randy's care caused his collapse. Further, because of other errors of trial counsel the trial court received an incomplete view of the evidence, which also contributed to Randy's erroneous conviction.

For all of the reasons set forth in this petition, the Defendant respectfully requests that this Honorable Court allow the justice system to right a wrong by granting him a new trial.

VII. PRAYER FOR RELIEF

Wherefore, Petitioner Randy Liebich, though his attorneys, moves this Court to consider the prejudicial impact of each of the above-stated deprivations of his constitutional rights individually and in combination with one another.

Accordingly, Randy Liebich respectfully requests the following relief:

A. Outright reversal of his conviction;

B. Vacation of his conviction followed by a new trial; or

C. An evidentiary hearing in which proof may be offered concerning the allegations contained in his petition.

Respectfully Submitted,

BY: _____

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List of Exhibits

Affidavits from Medical Experts

Exhibit 1	Affidavit of Dr. Patrick Barnes
Exhibit 2	Affidavit of Dr. Michael Laposata
Exhibit 3	Affidavit of Dr. Darinka Mileusnic-Polchan
Exhibit 4	Affidavit of Dr. Shaku Teas
Exhibit 5	Affidavit of Dr. George Nichols
Exhibit 6	Affidavit of Dr. Peter Stephens
Exhibit 7	Affidavit of Dr. Waney Squier
Exhibit 8	Affidavit of Dr. Ronald Uscinski
Exhibit 9	Affidavit of Nathan Felix

Affidavits from Fact Witnesses

Exhibit 10	Affidavit of Randy Liebich (February 23, 2009)
Exhibit 11	Supplemental Affidavit of Randy Liebich (February 23, 2009)
Exhibit 12	Affidavit of Dion Liebich
Exhibit 13	Affidavit of Investigator Roger Lilly
Exhibit 14	Affidavit of Denise Foster (March 30, 2012)
Exhibit 15	Affidavit of Denise Foster (April 3, 2012)
Exhibit 16	Affidavit of Marlene Szafranski
Exhibit 17	Affidavit of Debra Minucciani
Exhibit 18	Affidavit of Ricky Holman
Exhibit 19	Affidavit of John Casey

Additional Exhibits

Exhibit 20	Rule 23 Order from the Illinois Appellate Court
Exhibit 21	Letter from Dr. Shaku Teas to Judge Jorgenson (August 26, 2004)
Exhibit 22	Postmortem Report
Exhibit 23	Microscopic Examination Notes of Dr. Darinka Mileusnic
Exhibit 24	Report of Mount Sinai Hospital
Exhibit 25	Transport Notes of Rush Medical Center
Exhibit 26	Rush Medical Center Medical Records
Exhibit 27	DuPage County Sheriff's Police Reports (from Mike Reidy Discovery Production)
Exhibit 28	Handwritten Report of Robert Liebich
Exhibit 29	March 3, 2009 *Pro Se* Post-Conviction Petition
Exhibit 30	April 1, 2009 Supplemental *Pro Se* Filing
Exhibit 31	Amended Petition for Post-Conviction Relief

STATE OF ILLINOIS
IN THE CIRCUIT COURT OF THE EIGHTEENTH JUDICIAL CIRCUIT
DUPAGE COUNTY

Randy Liebich, Petitioner	)	Cir. Ct. No. 02-CF-654
v.	)	Post Conviction No.
People of the State of Illinois	)	

AFFIDAVIT OF DR. PATRICK BARNES

1. My name is Patrick D. Barnes, M.D. I am a pediatric neuroradiologist and am board certified in Diagnostic Radiology and Neuroradiology. I am a Professor of Radiology at Stanford University Medical Center and Chief of Pediatric Neuroradiology and Medical Co-Director of the MRI/CT Center at Lucile Packard Children's Hospital at Stanford. I have practiced and taught on head injury in children for thirty years, and have published over a hundred articles, reviews and book chapters on this subject. My curriculum vita is attached. Att. 1.

2. I am a former member of the Child Abuse Task Force, Society for Pediatric Radiology and was Chair of the Task Force from 2007-2008. I am also a co-founder and member of the Child Abuse Task Force and SCAN team, Lucile Packard Children's Hospital, Stanford University Medical Center and Santa Clara Valley Medical Center.

3. I have reviewed the 2/8/02 CT scan for Steven Quinn as well as radiology reports for the 2/8 CT scan, babygram (skeletal survey) and chest x-ray.

4. I have received medical records, including records from Mt. Sinai Hospital (2/8), Rush Presbyterian Hospital (Rush) (2/8-2/11), pediatric records, the autopsy report and supporting documentation, and various state and defense consultations. I have also received the trial transcript and police reports.

Summary

5. The CT scan (brain) taken shortly after hospital admission shows thin hemorrhages (subdural, subarachnoid, intradural and/or intraparenchymal), the beginning of a hypoxic-ischemic brain (*i.e.,* a brain that lacks oxygen), sinus disease and suspicion of thrombosis (clotting) in the dural sinuses.

6. There are no fractures, soft tissue swelling or other abnormalities that would suggest head trauma.

7. Based on the clinical history and medical records, the CT findings are likely secondary to abdominal injury/infection, possibly aggravated by choking.

Radiology review

8. 2/8 CT scan (~ 6:25 pm). The CT scan (brain without contrast) taken shortly after admission to Mt. Sinai was read that evening and a report dictated the following morning. In this section, I provide the Mt. Sinai reports with comments based on my review of the images.

9. Mt. Sinai report (2/8). This handwritten report describes an intracranial hemorrhage with right subdural blood and a small right temporal intraparenchymal hemorrhage (bleeding within the brain), all concerning for trauma. There are areas of linear pattern in the right temporal lobe suspicious for subarachnoid hemorrhage. No fractures are seen.

10. Mt. Sinai report (dictated 2/9). The report on the same CT scan dictated the following morning describes a fine density in the right image, indicating intracerebral hemorrhage over the frontotemporoparietal lobes. There is an area of increased density in the subdural and subarachnoid space, indicating subdural and subarachnoid hemorrhage. There is no midline shift and no evidence of fracture or soft tissue swelling. The impression is of subarachnoid or subdural hematomas in the right hemisphere associated with intracerebral bleed in the right horn of the parietal and temporal lobes. There are also subarachnoid and subdural bleeds involving the posterior fossa.

    Comments. There are no fractures and no soft tissue swelling or other abnormalities in the contour of the scalp that would suggest impact or a large hematoma.

    The Mt. Sinai reports do not describe a subdural hemorrhage of significant size, and the images show thin bilateral subdural, intradural and/or subarachnoid hemorrhages that are not causing a mass effect or midline shift (compressing the brain or pushing it to one side). There may be some subpial hemorrhage in the right temporal lobe and some other right cerebral sites of hemorrhage or hemorrhagic strokes. Since the film quality is poor, it is difficult to identify precise size and location. The hemorrhages are acute on CT, which gives a timing range of 3 hours to 7-10 days. Given the size and location, these hemorrhages would not be subject to evacuation.

    There are bilateral maxillary sinus and ethmoid air cell opacities (sinusitis).

    The brain is beginning to show diffuse abnormality, more on the left, with possible loss of grey-white differentiation, though this cannot be precisely determined. This suggests hypoxia-ischemia, indicating that the brain is being deprived of oxygen, causing edema (brain swelling). This is a nonspecific finding that can reflect illness/infection, organ failure, sepsis, thrombosis (clotting/stroke) or trauma.

On CT, the major dural sinuses (superior sagittal and transverse) appear dense, suggesting thrombosis (abnormal clotting or stroke). Thrombosis may be secondary to a wide range of causes, including systemic illness. Ultimately, thrombosis of the major sinuses deprives the brain of oxygen and is sometimes accompanied by intracranial hemorrhage. An MRI and other supplemental radiology would have provided better information on these points.

It is not possible to determine on CT when the brain swelling began or what caused it. Brain swelling may be a slow process (as in lung or heart disease), a secondary consequence of infection/inflammation, or the result of a recent event, such as choking. An MRI would have provided more detailed information on timing.

To address cause and timing, the radiology must be correlated with the clinical history, laboratory tests and autopsy results. If the child is deceased, pathology (microscopic review of tissue slides) is the gold standard since far more detail can be seen by microscope than by CT scan or the naked eye.

11. Skeletal studies and chest x-rays. The x-rays show bilateral pulmonary (lung) disease. The radiology and autopsy reports confirm that there were no fractures or other skeletal injuries or abnormalities.

Differential diagnosis

12. Since 2000, the pediatric literature has identified many causes for medical findings previously viewed as diagnostic of non-accidental trauma. The differential diagnosis for subdural hemorrhage and other findings previously attributed to shaken baby syndrome or inflicted trauma in a 2002 article by leading forensic pediatricians includes trauma (accidental or non-accidental); medical or surgical interventions; metabolic, genetic, oncologic or infectious diseases; congenital malformations; autoimmune disorders; clotting disorders; and other miscellaneous conditions. Hymel K, Jenny C, Block R, *Intracranial Hemorrhage and Rebleeding in Suspected Victims of Abusive Head Trauma: Addressing the Forensic Controversies,* Child Maltreatment. 2002 Nov;7(4):329-348. My 2002 article is in accord. Barnes, PD, *Ethical Issues in Imaging Nonaccidental Injury: Child Abuse,* Topics in Magnetic Resonance Imaging. 2002 Apr;13(2):86-93 at 91.

13. A 2006 text on abusive head trauma in infants and children contains a more complete discussion of the alternative diagnoses. Sirotnak A, *Medical disorders that mimic abusive head trauma,* in Frasier et al, Abusive Head Trauma in Infants and Children: A Medical, Legal, and Forensic Reference, Ch. 14 at 191-226, St. Louis, MO: GW Medical Publishing 2006 (medical disorders that mimic abusive head trauma include prenatal, perinatal and pregnancy related conditions; accidental trauma; genetic and metabolic disorders; disorders of coagulation and clotting; infection, autoimmune conditions and vasculitis; oncological conditions; and nutritional deficiencies). My 2007 article contains a similar differential diagnosis and describes the pathophysiology, which includes increased intracranial pressure, systemic hypotension or hypertension, increased venous

pressure, vascular fragility, hematologic derangement and/or collagenopathy, superimposed on immature central nervous and other systems. Ex. 2 at 65-70. A more comprehensive discussion of the radiology is contained in my 2011 review. Ex. 3.

14. Over the past decade, many of the underlying precepts of shaken baby syndrome/abusive head trauma have been questioned. For example, in the early 2000s, it was widely believed that swollen brains were caused by traumatically torn axons (the nerve fibers that connect the brain cells) and that subdural hemorrhages were caused by traumatically ruptured bridging veins (the large veins that connect the brain to the sinuses). A corollary was that great force would be required to rupture axons and bridging veins and that the child would be immediately and devastatingly symptomatic.

15. In 2001, however, neuropathological studies found that the brain swelling seen in pediatric cases reflects lack of oxygen to the brain (hypoxia ischemia) rather than torn axons and is also seen in natural deaths. These studies found that thin subdural hemorrhages are also seen in natural deaths. Geddes JF, Hackshaw AK, Vowles GH, Nickols CD, Whitwell HL, *Neuropathology of Inflicted Head Injury in Children, I. Patterns of Brain Damage,* Brain. 2001 Jul;124(Pt 7):1290-8; Geddes JF, Vowles GH, Hackshaw AK, Nickols CD, Scott IS, Whitwell HL, *Neuropathology of Inflicted Head Injury in Children, II. Microscopic Brain Injury in Infants,* Brain. 2001 Jul;124(Pt 7):1299-306. Similar findings may also sometimes be seen in short falls. Plunkett J, *Fatal Pediatric Head Injuries Caused by Short-Distance Falls,* Am J Forensic Med Pathol. 2001 Mar;22(1):1-12.

16. More recently, we have learned that the anatomy of the infant dura is much more complex than previously recognized and that the findings previously attributed to trauma are closely linked to hypoxic ischemic encephalopathy, at least in neonates. Cohen MC, Scheimberg I, *Evidence of Occurrence of Intradural and Subdural Hemorrhage in the Perinatal and Neonatal Period in the Context of Hypoxic Ischemic Encephalopathy: An Observational Study from Two Referral Institutions in the United Kingdom,* Pediatr Dev Pathol. 2009 May-Jun;12(3):169-76. Reviews of the anatomy have further established that the thin subdurals found in children are too small to result from ruptured bridging veins and are more likely intradural in location. Squier W, Mack J, *The Neuropathology of Infant Subdural Haemorrhage*, Forensic Sci Int. 2009 May 30;187(1-3):6-13; Mack J, Squier J, Eastman JT, *Anatomy and Development of the Meninges: Implications for Subdural Collections and CSF Circulation*. Pediatr Radiol. 2009 Mar;39(3):200-10. These and other articles indicate that the findings previously attributed to shaking or abuse do not reflect traumatically torn axons or bridging veins (as previously believed), do not necessarily require great force (or in some instances any force at all), and may be secondary to a wide array of causes, including natural causes.

17. One of the factors now recognized as a cause of findings previously attributed to abuse is cerebral venous thrombosis (clotting in the veins that drain the brain). This diagnosis is easily missed since it presents with nonspecific symptoms and often requires specialized imaging techniques. *See, e.g.,* Kirkham, F, *Investigation and Management of Childhood Stroke,* Paediatrics Child Health. 2010 Sept;20(9):428-38 (stroke remains one of the

commonest causes of death and disability in childhood, with advances in recent years due to improvements in noninvasive methods of imaging); Mallick AA, O'Callaghan FJ, *The Epidemiology of Childhood Stroke,* Eur J Paediatric Neurol. 2010 May;14(3):197-205. (stroke diagnosed more often given increased medical awareness and better imaging techniques); Bousser MG, Barnett HJM, *Cerebral Venous Thrombosis* (Ch. 12 in Stroke Pathophysiology, Diagnosis, and Management, 4th Ed. 2004) (CVT far more common than previously assumed; challenging diagnosis for clinicians due to wide spectrum of clinical presentations, unpredictable course and multifactorial causes; risk factors in young children include acute systemic illness); deVeber G, Andrew M, Adams C, Bjornson B, Booth F, Buckley DJ, *Cerebral Sinovenous Thrombosis in Children,* N Engl J Med. 2001 Aug 9;345(6):417-23 (risk factors include acute systemic illness and dehydration; extraparenchymal hemorrhages seen in 9% of cases). In this case, the high densities along the dural venous sinuses seen on CT suggest thrombosis of the major sinuses, which would explain the CT findings.

18. This case is further complicated by the abdominal injury/infection. Abdominal injuries and infections are difficult to diagnose and may progress slowly. *See, e.g.,* Moser L et al, *Prolonged Survival Time Following Duodenal Transection in a Child With Abdominal Trauma,* Am Acad For Sciences Abstract G42 (Feb. 2009) (24 hour period between fall from bicycle and collapse; symptoms of serious abdominal injury may be subtle; timeline requires correlation with microscopic sections of the injury); Herr S, *Abdominal and Chest Injuries in Abused Children,* Chapter 36 in Jenny C et al Child Abuse and Neglect Diagnosis, Treatment and Evidence (Elsevier Saunders 2011) (commonly injured structures include the small bowel, liver and pancreas, with injuries often similar to those seen in bicycle handlebar injuries; symptoms may be subtle, nonspecific and delayed; since the bowel content and inflammatory responses are initially walled off within the retroperitoneum; it may take several days for diffuse peritonitis and the associated symptoms to develop, with patients often presenting for medical care hours, days or even weeks after the injuries occurred); Alexander R, *Associated Injuries,* Chapter 12 in Frasier et al, Abusive Head Trauma in Infants and Children, a Medical, Legal, and Forensic Reference (G.W. Medical Publishing 2006) (abdominal trauma symptoms are a developing phenomenon that occurs as the bleeding progresses or infection begins; treatment is often delayed since symptoms of abdominal trauma may develop late or the person who abused the child may delay seeking medical care). The child abuse literature also recognizes that abdominal injuries in children may be natural rather than traumatic. *See, e.g.,* Ludwig S, *Visceral Injury Manifestations of Child Abuse,* in Reece R and Ludwig S, Child Abuse, Medical Diagnosis and Management (Lippincott, Williams and Wilkins 2d ed. 2001) (perforated bowel secondary to inflammatory bowel disease may mimic child abuse; signs and symptoms of nontraumatic bowel diseases may not be apparent, particularly in the young child).

19. Head injuries may similarly result in deterioration and/or death after a period of normality or relative normality. *See, e.g.,* Arbogast KB, Margulies SS, Christian CW, *Initial Neurologic Presentation in Young Children Sustaining Inflicted and Unintentional Fatal Head Injuries,* Pediatrics. 2005 Jul;116(1):180-4 (on rare occasions, infants or toddlers may sustain a fatal head injury yet present to hospital clinicians as lucid before

death); Denton S, Mileusnic D, *Delayed Sudden Death in an Infant Following an Accidental Fall, A Case Report with Review of the Literature,* Am J Forensic Med Pathol. 2003 Dec;24(4):371-6 (9-month-old acted normally for 72 hours after fall before fatal collapse); Huntington R, Letter, *Symptoms Following Head Injury*, Am J Forensic Med Pathol. 2002 Mar;23(1):105-06 (13-month-old had severe intracranial injury with symptoms delayed for several hours, during which time she was under hospital care); Gilliland MG, *Interval Duration Between Injury and Severe Symptoms in Nonaccidental Head Trauma in Infants and Young Children,* J Forensic Sci. 1998 May;43(3):723-5. (finding intervals of more than 24 hours and sometimes up to 72 hours between the trauma and the collapse in approximately 25% of alleged shaking, shaking/impact or impact cases). When the findings result from a natural disease process, the concept of a "lucid interval" is virtually inapplicable since, like any disease process, the natural conditions that mimic abusive head trauma – ranging from stroke to metabolic or genetic disorders – may result in sudden collapse or may be slowly progressive, sometimes presenting with nonspecific symptoms for days or weeks before collapse.

20. Given these major shifts in the literature, I recently published a major invited review of the medical issues in this area, including the implications of particular radiological findings. Barnes PD, *Imaging of Nonaccidental Injury and the Mimics: Issues and Controversies in the Era of Evidence-Based Medicine*, Radiol Clin North Am. 2011 Jan;49(1):205-29. Att. 2. Similar reviews have been published in neuropathology and emergency medicine. Squier W, *The "Shaken Baby" Syndrome: Pathology and Mechanisms*. Acta Neuropathol. 2011 Nov;122(5):519-42; Gabaeff SC, Challenging *the Pathophysiologic Connection between Subdural Hematoma, Retinal Hemorrhage and Shaken Baby Syndrome*, West J Emerg Med. 2011 May;12(2):144-58. A recent presentation by a leading child abuse pediatrician similarly stated that no trained pediatrician thinks that subdural hemorrhage, retinal hemorrhage and encephalopathy equals abuse. Instead, each physical finding must be evaluated separately, and the case must then be reconstructed in its entirety. Jenny C, Presentation, *The Mechanics: Distinguishing AHT/SBS from Accidents and Other Medical Conditions*, slide 11, New York City Abusive Head Trauma/Shaken Baby Syndrome Training Conference (Sept. 23, 2011).

21. For many years, physicians routinely diagnosed shaken baby syndrome/abusive head trauma based primarily or solely on a triad of findings (subdural hemorrhage, retinal hemorrhage and brain swelling). Today, it is well understood that there are many alternative causes for these findings. In distinguishing between natural, accidental and nonaccidental causes, the physician must consider the full range of possibilities, as well as the combined or synergistic effects of two or more conditions. The role of the pediatric neuroradiologist is to identify possibilities and to point out whether particular diagnoses are consistent or inconsistent with the radiology. Any diagnosis must be coordinated with the clinical history and medical records. If the child has died, the diagnosis must also be coordinated with the pathology.

Clinical history

22. The pediatric records appear normal with the exception of possibly abnormal weight gain as an infant (MED 227) and a possible weight loss of nearly 5 pounds between his last checkup on 11/6/01 and hospital admission on 2/8/02. MED 261, 46.

23. The remainder of the history has been provided to me by counsel. This history indicates that the child, who was nearly 3 years old, may have had nonspecific symptoms in the days before his collapse (lethargy, possible cold symptoms) on 2/8. The evening before his collapse, he was crying and refused to eat dinner, causing the mother to physically discipline him. There is disagreement on whether the child ate that evening.

24. On 2/8, the child slept until approximately 10 a.m, when his mother left for work, leaving him with Mr. Liebich, who was also caring for their 11 day old baby. According to Mr. Liebich, the child ate some cereal after his mother left but left the milk.

25. According to Mr. Liebich, the child drank a glass of orange juice, ate part of a cut-up hot dog and drank water at approximately 3 p.m. When he appeared to choke, Mr. Liebich patted him on the back and attempted to clear the airway with his finger in case the hot dog had lodged in the child's throat. When he did so, the child bit down on his finger, and Mr. Liebich slapped or tapped him on the side of his face to get him to release the finger. The child may have then vomited. Mr. Liebich described the child as dizzy after this incident but walking on his own. Mr. Liebich and the child then fell asleep while watching a video.

26. When the mother returned ~4:30 p.m., she found that the child was breathing abnormally and seemed to need to vomit. When the child did not respond normally, Mr. Liebich and the mother took him to the hospital, stopping en route at Mr. Liebich's work. The manager described the child as blinking and appearing ill but with no notable signs of trauma.

Mt. Sinai: medical records and trial testimony

27. Hospital records. At Mt. Sinai, the labs results from a 6:22 p.m. blood draw confirmed pancreatitis (amylase 3025 v. reference range 20-120; lipase 2368 v. reference range 22-51). MED 24. The blood and urine tests confirmed high glucose. MED 24, 28.

28. Although there are no references to size in the medical records, the CT scan was interpreted as showing a large subdural hemorrhage requiring immediate evacuation. The abdominal CT and other orders were cancelled, and the child was sent to Rush for evacuation of the subdural. MED 14.

29. The Mt. Sinai records and transport notes describe numerous lines and other marks that appeared on the child's body after arrival in the hospital. MED 9, 42, 47-48. These included a bruise in the right temporal region and "red small marks on

abdomen/back/buttocks". MED 42. Some of the lines are described as horizontal rope marks. MED 47.

30. Dr. Green. Dr. Paula Green, the attending E.R. doctor, testified that her initial exam showed no signs of trauma and that she thought Steven was suffering from an illness or metabolic disorder. Green 19. However, the child began posturing and she called Dr. Boykin, a trauma doctor. Green 29-30. A radiology technician was alarmed by the CT and called to say that there was a bleed, resulting in transport to Rush. Green 31.

31. Dr. Green testified that severe brain damage and bleeding can be caused by ischemia (lack of oxygen) and that there was no midline shift. Green 47, 48, 50. She testified that Dr. Munoz, the Rush neurosurgeon, cancelled the abdominal CT since the focus was now on the head. Green 60, 61.

32. Dr. Green described Mr. Liebich as calm, restrained and respectful. Green 60.

33. Dr. Boykin (Rush). Dr. Tracy Boykin, a trauma doctor, testified that from the initial presentation, she assumed that Steven was suffering from a medical condition or febrile seizure rather than trauma. Boykin 83-84. The initial high glucose suggested a metabolic disorder. Boykin 88.

34. Subsequently, Dr. Boykin saw Steven posturing, which is an early sign of severe brain injury. Boykin 86. She was called to the CT scan machine by a radiology technician who told her that the child's head was "full of blood." Boykin 89. Dr. Boykin stated that while she sees CT scans every day, she doesn't usually read them. Boykin 93. Dr. Boykin looked at the CT scan with the technician and agreed that "it was really bad." Boykin 90.

35. Dr. Boykin testified that she became angry with Mr. Liebich when he yawned when Steven was posturing and when she looked at the CT scan. Boykin 90, 94-95. When she returned to the emergency department after reviewing the CT scan, she told Mr. Liebich and the child's mother that the child had obviously suffered a severe brain injury secondary to trauma and that it looked as if Mr. Liebich had been "sitting at home beating on him all day." Boykin 90-91. She became angrier when Mr. Liebich, who was holding the new baby, "didn't really say much at all" and "shrugged as if he didn't know" when she told him that the child's injuries were inconsistent with choking on a hot dog. Boykin 91-92.

36. Dr. Boykin testified that it is unlikely that a person with a subdural hemorrhage would die suddenly since this is a venous bleed and can be slow, as in nursing home deaths. Boykin 102. She confirmed that severe abdominal injuries would cause problems for the brain and other organs, leading to hemorrhagic shock and a hypoxic brain and heart. Boykin 105. Dr. Boykin did not know if much attention was paid to the abdomen as she was "more of a consult". Boykin 105. Dr. Boykin did not talk to Dr. Munoz after his post-operative report and did not know what he found during surgery. Boykin 112.

37. Tammy Smith, R.N. Ms. Smith, a member of the Rush transport team, testified to the marks that appeared before, during and after transport. Ms. Smith recalled turning to the transport resident at Mt. Sinai and saying, "why are we taking him to Rush? He's dead." Smith 25. The transport notes refer to a large subdural hemorrhage. MED 46.

Rush: medical records and testimony

38. February 8. The Rush admission entry, physician notes and anesthesia reports describe a large subdural hemorrhage. The admitting diagnosis is head trauma. MED 49, 67, 76, 79.

39. Labs taken at Rush at 9 pm confirmed pancreatitis and liver dysfunction (lipase 9598; amylase 1131; SGOT 5429; SGPT 3130). MED 181.

40. The child had neurosurgery for evacuation of the subdural hemorrhage at approximately 10 p.m. However, the postoperative diagnosis states "No subdural hematoma found," and the notes confirm that "there was no large subdural blood accumulation." Instead, there was "a severely swollen brain with a large amount of subarachnoid hemorrhage and a small thin subdural hematoma." MED 77-78.

41. After the surgery, the neurosurgeon cancelled the abdominal evaluation since the child was very close to brain death. MED 86.

42. References to a large subdural hemorrhage continue throughout the hospital records, with virtually no references to pancreatitis other than a reference to "pancreatic disease" at Mt. Sinai and "rule out pancreatic injury" in the transport notes. MED 6, 48.

43. February 9. The morning after surgery, the abdomen was "impressively swollen" and firm with "erythema perumbilically." MED 95A. A peritoneal (penrose) drain was placed and a large amount (500 cc) of serosanguinous pink fluid was drained. MED 84 A-B. It does not appear that this fluid was cultured. An attending physician noted the Cullen's sign (erythema or redness around the umbilicus), which is a clinical sign of pancreatitis. MED 99.

44. At that point, the diagnosis was (1) traumatic brain injury (child abuse) rule out shaken baby syndrome; and (2) SIRS shock (a systemic inflammatory response that affects the entire body) with MODS (multiple organ dysfunction syndrome) involving the central nervous system, hematology, pulmonary, bowel and CV (cardiovascular), rule out sepsis/septic shock (widespread infection). MED 99.

45. By then, the child had failed a brain death test and was being transfused for thrombocytopenia (low platelet count; side effects include bruising and bleeding). MED 91, 103, 188, 101, 212. Antibiotics were ordered. MED 135.

46. There are continued references to new lines and marks that appear on the child's body during hospitalization. These are often characterized as bruises, whip marks or lash marks. MED 70, 75, 98, 99, 105, 145.

47. February 10. On February 10, the attending physician diagnosed traumatic brain injury secondary to nonaccidental trauma/shaken baby syndrome. MED 109. A subsequent note indicates a diagnosis of brain death, respiratory failure and SIRS shock with multiple organ dysfunction syndrome. MED 111.

48. February 11. On February 11, progress notes (portions missing) diagnose traumatic brain injury secondary to child abuse/shaken baby syndrome with right subdural and intraparenchymal bleeds, SIRS and multiple organ dysfunction syndrome. MED 117.

49. A child abuse pediatrician's notes state that the child was found to have large subdural and intraparenchymal hemorrhages, extensive cutaneous injuries and intra-abdominal injuries without a history of trauma, and that these findings collectively are diagnostic of child abuse. MED 118-119.

50. Later that morning, the neurosurgeon told the family that the child had been physically abused and was brain dead. MED 121-122.

51. Discharge report. The discharge report states that the initial CT showed a large right subdural hemorrhage and intraparenchymal hemorrhage, with nonaccidental head and abdominal trauma strongly suspected. It repeats the child abuse pediatrician's statements that the findings were collectively diagnostic of child abuse. MED 60-61. The medical diagnosis is "SIRS shock with multi-organ dysfunction...involving CNS, pulmonary, GI, and cardiovascular systems." MED 62.

52. Dr. Severin, pediatric critical care. Dr. Paul Severin testified that he had treated approximately 20 victims of child abuse but that this might be the first time that he had been asked to time injuries. Severin 3, 60-61.

53. Dr. Severin testified that when Steven arrived at Rush, he was critically ill, appeared to be in shock and had bruises on his head, body and legs, all of which appeared to be about the same age. Severin 34-35, 41. He felt that bowel injuries were unlikely since the bowel sounds were good. Severin 44. However, the lab tests showed elevated pancreatic enzymes and poor liver function, raising concern for abdominal injuries. Severin 46-47. It appears that Dr. Severin may not have received the lab results until after the surgery. Severin 49.

54. Dr. Severin testified that before surgery, the major concern was with the head since by clinical exam and CT scan it appeared that the "brain had a lot of blood in it" that had to be taken out immediately or Steven would die. Severin 49.

55. The following morning, Dr. Severin found a distended stomach with "redness around the belly button," which he identified as the Cullen's sign (a sign of pancreatitis). Severin 50-51. Dr. Severin had received the autopsy report confirming pancreatitis and other abdominal findings. Severin 51-52. He explained that the disruption of the blood vessels can cause bleeding and poor blood supply to the intestines, causing the tissue to break

down and perforate, resulting in leakage of the contents into the abdomen. Severin 53. The hematomas in the liver and gallbladder were consistent with this process. Severin 53.

56. Dr. Severin testified that the pancreatitis was acute (recent), that the child would have had immediate symptoms, and that the process must have just started based on the progression of the bowel sounds. Severin 54-55. He testified that he believed that these findings were caused by a force "such as falling from a large height, like 20 or 30 feet, rapid deceleration to an unyielding force. In like a motor vehicle crash, blunt trauma such as a fist, foot, blunt object like a [brick]". Severin 55-56.

57. Based on the head injuries, Dr. Severin testified that the injuries likely occurred 4-6 hours before hospital admission. Severin 57. He did not believe that the trauma could have occurred the day before admission since the child would not have survived until admission. Severin 57-58. This estimate was based on the extent of the injury, the intracranial pressure at surgery, the injury to the pancreas and liver, the bruising (particularly to the scrotum and head), and the lack of any history of trauma. Severin 58.

58. Dr. Severin testified that he was told that the CT showed a large subdural hemorrhage and intraparenchymal hemorrhage. Severin 66-67. However, after surgery, he learned that the subdural hemorrhage was small and not as serious as the CT scan suggested and that the surgery instead found diffuse subarachnoid hemorrhage. Severin 67-68. He agreed that the absence of a midline shift on the initial CT indicated that the massive increase in intracranial pressure had not occurred at the time of the CT scan. Severin 69-70.

59. Dr. Severin had not reviewed the pathology slides and was not aware that Dr. Mileusnic had dated the head injuries at approximately five days. Severin 71-72, 93-94. He testified that even if the pathology showed injuries that were at least five days old, he would stand firm that the injuries occurred within 48 hours of his examination [*i.e.*, no earlier than 9 p.m. on February 6] based on the physical examination and acuteness of the illness. Severin 74. The presence or absence of macrophages and fibroblasts would not affect his opinion. Severin 74. Dr. Severin testified the bruises also occurred in this time period. Severin 70-71.

60. Dr. Severn testified that axonal injury comes from shearing force in shaken baby cases but can also come from poor blood flow (*i.e.*, hypoxia-ischemia). Severin 81-82.

61. Dr. Severin had no doubt that Steven had severe abdominal injuries that could have caused death. Severin 76. He did not agree that abdominal injuries can take a couple days to become symptomatic. Severin 76.

62. Dr. Munoz, Rush neurosurgeon. Dr. Lorenzo Munoz, the neurosurgeon who operated on Steven, testified that Steven was transferred to Rush because Mt. Sinai determined that he had a severe head injury and that surgical intervention was required. Munoz 20. He was told that Mt. Sinai had seen "a lot of blood" in Steven's CT scan. Munoz 22. Dr. Munoz

looked at the CT scan before performing surgery and reviewed the CT scan in court, stating that the pronounced white haziness under the bone causes one to think about subdural hemorrhage. Munoz 25, 27-28.

63. Dr. Munoz testified that the CT showed a subdural hemorrhage in a location highly suggestive of severe head trauma caused by a mismatch between the movement of the brain and skull, causing shearing of the bridging veins and a large space-occupying bleed that creates pressure on the brain. Munoz 29. He testified that he thought there was a right frontal temporal parietal and posterior interhemispheric subdural hemorrhage but that the haziness was subarachnoid hemorrhage. Munoz 29-30.

64. Dr. Munoz testified that the CT showed a loss of grey-white differentiation due to massive brain swelling. Munoz 31, 32. He testified that these findings were caused by acceleration and deceleration equivalent to a car accident, causing shearing injury. Munoz 33-34. Dr. Munoz testified that the child's prospects were very dismal based on the CT but that he elected to take heroic measures by decompressing the blood clot since the child's pupils were still reactive. Munoz 35.

65. Dr. Munoz testified that at surgery, he saw a very swollen brain that started to come out of the skull opening created at surgery, with massive amounts of red fresh blood. Munoz 40-41. Based on the CT, he thought there would be a larger subdural hemorrhage but most of the blood was subarachnoid rather than subdural. Munoz 42. Dr. Munoz testified that the presence of subdural and subarachnoid blood indicates a major trauma and that the color of the blood gives a very good idea of the timing. Munoz 42-43.

66. Dr. Munoz testified that one sees this type of brain swelling in children who fall out of 3-4 story buildings or who are struck by cars or hit windshields while unrestrained. Munoz 43. He testified that the amount of force and trauma was similar to being in a horrible car accident and that the amount of blood in the brain told him that the injury must be fairly recent. Munoz 81, 44. Dr. Munoz testified that it is not possible to evacuate subarachnoid blood but that he had evacuated a thin subdural hemorrhage. Munoz 45-46.

67. After surgery, the child's eyes were fixed and nonreactive, indicating that there had been a spiraling or ongoing process that could not be stopped in spite of best efforts. Munoz 47. After the surgery, Dr. Munoz told Dr. Bass, the pediatric surgeon, that the child was brain dead and that there was no use doing exploratory abdominal surgery. Munoz 51-52.

68. Dr. Munoz testified that the brain findings resulted from nonaccidental trauma and that his best estimate was that the injury occurred within six hours of arriving at Mt. Sinai. Munoz 49-50. He testified that it was impossible that the injury could have occurred on the evening of February 7 and that the child could not have walked unassisted, talked, ate or drank after the injury. Munoz 49-51. Dr. Munoz testified that estimating the timing of injury is not something that one learns in pathology and that he extrapolated his opinion from seeing hundreds of children like Steven. Munoz 59.

69. Dr. Munoz indicated that lucid intervals are limited to epidural hemorrhages and that while it is possible for a heart attack or bowel perforation to kill a person, this is "so unlikely" that he had "never heard of it happening and certainly not in...[his] experience." Munoz 62-64. He testified that it is not possible to remain conscious with the head injury and subarachnoid hemorrhage seen in this case. Munoz 69.

70. Dr. Munoz disagreed that the best means of dating intracranial injuries is through examining pathological material obtained in neurosurgical intervention or at autopsy. Munoz 74. Instead, he testified that x-rays, perpetrator admissions and "looking at living tissue" are the gold standard in timing. Munoz 74-75.

Autopsy

71. The medical examiner's investigative report states that the child was admitted with a diagnosis of head trauma and was found to have a "massive" subdural hemorrhage that was evacuated on 2/8. There is no mention that the subdural hemorrhage was minimal and/or not present at surgery or that the child had pancreatitis. MED 297.

72. At autopsy, Dr. Mileusnic, the Cook County medical examiner who performed the autopsy, found major abdominal injury/infection, including a large segment of necrotic (dead) bowel with a small (.1 inch) perforation, peritonitis, peripancreatitis, pancreatitis and liver injury. She also noted the severely damaged brain and numerous contusions and lines on the body. The death was ruled a homicide. MED 273-284. Based on her review of the microscopic slides (pathology), Dr. Mileusnic indicated that key findings were subacute and/or approximately five days old. MED 289.

73. Dr. Mileusnic. Dr. Mileusnic testified that the child had a hemorrhagic necrotic bowel with a perforation, peritonitis, pancreatitis, and earlier injury manifested by fibrin deposits. Mileusnic 55-58. Some of the hemorrhages, including the swollen scrotum, were caused by the tracking of blood from the abdomen into the scrotum. Mileusnic 59. There was also deep bruising "that was not consistent with normal corporal punishment." Mileusnic 67-68. Dr. Mileusnic testified that this case was more complex because of the abdominal trauma.

74. Dr. Mileusnic testified that the child had numerous marks and bruises. Some of these marks were consistent with tape-related or other artifacts, and some changed between the hospital and autopsy photographs. Mileusnic 22-23, 27-30, 35-36, 40, 43.

75. Dr. Mileusnic testified that there can be a lucid interval following head trauma, with symptoms and deterioration occurring 24-48 hours after injury. Mileusnic 75-77. She referenced one of her own case studies, in which the child deteriorated and died 72 hours after an accidental fall off a bed, with tremendous brain swelling appearing as a late manifestation of the earlier impact. Mileusnic 77. She also testified that some head injured people are released from the hospital and go home and die. Mileusnic 115.

76. Dr. Mileusnic testified that the pathology indicated that the head injuries were approximately 5 days old, plus or minus 1-2 days. She testified that it is difficult to give intervals on abdominal injuries since these can deteriorate rapidly. Mileusnic 86. Abdominal injuries also notorious for slowly developing manifestations. Mileusnic 94.

77. Dr. Mileusnic testified that Dr. Severin's estimate of 24-48 hours for the bruises was consistent with her findings since it can take days for deep bruises to reach the surface. Mileusnic 90-91. However, she was uncomfortable dating external bruises based on appearance. Mileusnic 92.

78. Dr. Mileusnic testified that Steven had early traumatic pancreatitis, which can occur from a natural disease process or from the breaking of the cell membranes from impact in the area of the pancreas. Mileusnic 95. The first symptoms would not be instantaneous. Mileusnic 95.

79. Dr. Mileusnic explained that the body responds to injury by sending neutrophils, followed by macrophages and lymphocytes, with fibroblasts appearing at 5-7 days to heal the process. Mileusnic 98. In this case, Dr. Mileusnic saw fibroblasts in some of the head findings, placing them in the 5-7 day range. Mileusnic 98-99.

80. Dr. Mileusnic did not think she took slides of the abdomen or bowel and had not reviewed the histology slides before trial. She testified that she had not been provided with information on the child's symptoms before death though she had heard a bit that morning. Mileusnic 102. She testified that inconsolable crying, finicky eating, loss of appetite, lethargy and sleeping for a long period can be symptoms of abdominal or head trauma. Mileusnic 101-102.

81. Dr. Mileusnic testified that what she saw at autopsy was a combination of the original injury and a complex cascade of events that occurs after injury, complicated by hypoxia. As a result of such cascades, even seemingly mild injury can lead to subsequent deterioration. Mileusnic 103-104. She testified that subdural hemorrhages may be secondary to hypoxia rather than trauma, particularly if they are thin and bilateral. MED 104, 106. This case was, however, complicated by the abdominal injuries and a healing subgaleal hemorrhage (bleeding below the scalp) on the left that appeared to be about five days old. Mileusnic 106.

82. Dr. Mileusnic testified that progressive abdominal injuries will lead to compromised circulation, shock and brain edema. Mileusnic 106. Loss of consciousness may occur quickly or may take a long period of time since the anatomy of the bowel sequesters it from other parts of the body. Mileusnic 108-109.

83. A more precise determination of timing would require a detailed history, including family interviews, medical records and police reports. Mileusnic 117. On these issues, she would defer to a board certified pediatric intensivist who had talked to the family, listened to the bowel and read the lab reports. Mileusnic 120. She would also defer to

the neurosurgeon on timing based on what he saw on the right side of the brain. Mileusnic 120-121.

84. Dr. Mileusnic testified that the child died because the brain failed, which can be the result of many different processes. Mileusnic 122. A child with a perforated bowel would have more symptoms than finicky eating, and she found it hard to believe that a child with such injuries would eat a whole breakfast without complaining. Mileusnic 126-128. She confirmed that the head injuries appeared to be approximately five days old at the time of death. Mileusnic 131.

85. Dr. Teas. Dr. Shaku Teas, the defense forensic pathologist, agreed that the autopsy photographs showed peritonitis and an ulcerated bowel with a small perforation. She testified that this appeared to be a crush injury that could be caused by hitting, punching or pushing from the back. Teas 38. Crush injuries may damage the mucosa and blood vessels, and it may take some time before the area becomes necrotic and the bowel contents spill into the peritoneal cavity. Teas 37-38. There may be some initial pain but the child may appear to be fine until the wall becomes ulcerated and peritonitis sets in. At that point, there would be more pain, and the child may become septic and/or lose consciousness. Teas 39.

86. Dr. Teas explained that pathologists time injuries by looking at the stage of healing as seen under the microscope. Teas 39, 50-51. When there is a continuum of infection, it is important to look at the oldest area of injury. In this case, Dr. Teas saw early granulation tissue, several layers of spindle cells, and capillaries that were nearly visible, which takes 7-10 days. Teas 56-57. Dr. Teas agreed that the injuries appeared to be approximately five days old, though they might be a little older. Teas 57. She testified that they could be four days old but she would put it more towards 5-6 days and certainly would not put it at 3 days or less based on the healing, granulation tissues and chronic changes. Teas 56-57, 118-121. She also identified an older injury in the dura. Teas 62.

87. Dr. Teas testified that a subdural hemorrhage is caused by bridging veins that are torn by the movement of the brain in the skull but that there can be lucid intervals. Teas 21, 72. Given the pathology, it was important to get a detailed history for 72 hours before the collapse. Teas 83. The history of crying, finicky eating and sleeping for a long period were important if abnormal. Teas 85-87. Dr. Teas testified that a person could suffer a minor injury, remain conscious and experience brain swelling days later. In this case, the brain swelling rapidly progressed throughout the hospital stay. Teas 90-91.

88. Dr. Teas agreed that Steven died from a severe beating and that some of the marks in the photographs were consistent with a belt or hanger. Teas II 6-7. Other marks were hospital artifacts reflecting the blood pressure cuff and tubing. Teas II 57.

89. Closing arguments. In closing arguments, the state claimed that "[t]here was blood everywhere" on the initial CT and that the brain was "mangled" by force. Closing Argument (CA) 79-80. The State relied on Dr. Munoz's assertion that there are no lucid intervals in head injuries. CA 86.

90. The State interpreted the Cullen's sign as evidence of a kick or punch, and rejected Dr. Mileusnic's testimony that the swollen scrotum was caused by tracking of blood from the inflamed peritoneal cavity. CA 164, 168. The state rejected the testimony of Dr. Mileusnic and Dr. Teas, who testified that the marks on the thighs appeared to be artifacts from tape and hospital interventions, suggesting that these confirmed a whipping that was akin to a flogging. CA 82. The State argued that Mr. Liebich's "hotdog" story was "one of the dumbest explanations for why the kid would be unconscious with bruises all over his body." CA 105.

91. In the defense closing, Mr. Liebich's attorney stated that Dr. Teas testified that it was not impossible that the injuries occurred on February 8. CA 152. He stated that the pathologists had severe doubts on this timing but were "not sure" if there were older injuries. CA 152.

92. In rebuttal, the State suggested that pathology is a "voodoo" science. CA 169.

93. Verdict. The court found Mr. Liebich guilty of beating Steven to death on February 8. In the Verdict, the Court stated that Dr. Teas timed the injuries somewhere between Feb. 4 and Feb. 9, which she characterized as "a great guess." Verdict 5-7.

94. The Court concluded that the force required to create the abdominal and head injuries would be the equivalent of falling from 20-30 feet, or from blunt trauma with a bat, brick, foot or fist to the abdomen, and that the child could not have eaten after receiving these injuries. Verdict 14.

95. The Court held that a doctor who opens the skull shortly after hospital arrival is in the best forensic position to determine the cause and timing of head injuries based on the amount and color of the blood, and that there is no lucid interval concept or theory with abdominal issues. Verdict 16-20.

Comments

96. The trial testimony and conviction appeared to rest on some combination of the initial misinterpretation of the CT scan, poor communication between the doctors, and a great deal of outdated medical literature, some of which was outdated by the time of trial and much of which has become outdated in the decade since Steven's death.

97. Steven died less than a year after publication of the Geddes research, which found that the swollen brains in infants reflected hypoxia ischemia (lack of oxygen to the brain) rather than traumatically torn axons, and that thin subdural hemorrhages are also found in natural deaths. By the time of trial in 2004, several of the prosecution witnesses, including Dr. Mileusnic, Dr. Green and Dr. Boykin, were aware of this shift in the literature, but other prosecution witnesses continued to rely on the basic tenets of shaken baby syndrome, which held that the subdural hemorrhages and brain swelling can only be caused by tremendous force (often described as equivalent to multistory falls or motor

vehicle accidents) and are immediately symptomatic. Since 2004, the child abuse literature has increasingly recognized that there are many natural and accidental causes for these findings and that there can be lucid intervals of up to 72 hours.

98. In this case, the lab reports, discharge diagnosis and autopsy report establish that the child had abdominal injury/infection, with hospital diagnoses of systemic inflammatory response syndrome (SIRS) and multiple organ dysfunction syndrome (MODS). The CT findings are consistent with these diagnoses and do not suggest head trauma.

99. The dense dural sinuses seen on CT are suspicious for thrombosis (abnormal clotting), which would not be unexpected in the presence of abdominal injury/infection. This case also presents multiple risk factors for thrombosis, including sinusitis, inflammatory bowel disease, poor appetite, vomiting, lethargy, unsteady gait and seizures.

100. There is no medical or scientific basis for the testimony that it is possible to time an injury based on the color of the blood as perceived by the naked eye. It is relatively common to find evidence of older injuries or processes under the microscope that cannot be seen on radiology or by the naked eye.

101. There is also no medical or scientific basis for the testimony that there can be no lucid interval following abdominal or head injuries, and that the force to create Steven's injuries would be the equivalent of falling from 20-30 feet or of blunt trauma with a bat, brick, foot or fist to the abdomen. Abdominal injuries may be caused by relatively minor trauma and may not become seriously symptomatic for days. Head injuries may also be slow to develop.

102. I agree with Dr. Munoz that the CT findings, particularly the hypoxic brain, were recent developments, likely occurring around the time of collapse. These findings were, however, likely secondary to pre-existing abdominal injuries/infection rather than head trauma. There is no radiological evidence of head trauma occurring on the day of admission or earlier.

103. I also agree with Dr. Severin that the child would have been unable to walk, eat or behave normally once he developed pancreatitis and a hypoxic-ischemic brain. However, these developments were likely secondary to pre-existing abdominal injuries/infection which, according to the pathology, began some days earlier. There is nothing in the records reviewed suggesting that the abdominal injuries/infection began on the day of collapse. Any attempt at dating should be coordinated with the clinical symptoms and pathology.

104. I have not reviewed the hospital and autopsy photographs so cannot comment specifically on the marks and lines that appeared during the hospitalization. However, the fact that these marks were not evident on arrival suggests that they may reflect a secondary coagulopathy such as disseminated intravascular coagulation rather than trauma occurring shortly before admission. This would be consistent with venous sinus thrombosis suggested on the CT scan.

105. In this case, the CT scan is consistent with thrombosis and hypoxia-ischemia secondary to the abdominal injuries/infection identified in the lab tests and at autopsy. The choking episode was likely a symptom of the abdominal injury/infection and may have triggered or accelerated the collapse. There is also a possibility of aspiration.

Conclusion

106. The CT findings are consistent with and likely secondary to the abdominal findings, possibly triggered or accelerated by choking. There are no radiological findings suggesting head trauma.

I swear under penalty of perjury that the foregoing is true and correct.

_____

Patrick David Barnes, M.D.

Date: _____

105. In this case, the CT scan is consistent with thrombosis and hypoxia-ischemia secondary to the abdominal injuries/infection identified in the lab tests and at autopsy. The choking episode was likely a symptom of the abdominal injury/infection and may have triggered or accelerated the collapse. There is also a possibility of aspiration.

Conclusion

106. The CT findings are consistent with and likely secondary to the abdominal findings, possibly triggered or accelerated by choking. There are no radiological findings suggesting head trauma.

I swear under penalty of perjury that the foregoing is true and correct.

Patrick David Barnes, M.D.

Date: March 31, 2012

State of California, County of San Mateo
Subscribed and sworn to (or affirmed) before me on this 31st day of March 2012, by Patrick David Barnes proved to me on the basis of satisfactory evidence to be the person(s) who appeared before me.

(Signature of Notary)

SHRIDHAR GORE
COMM. # 1926557
NOTARY PUBLIC - CALIFORNIA
SAN MATEO COUNTY
MY COMM. EXP. MAR. 22, 2015

Name: Patrick D. Barnes, M.D. **Mar 2012**
Office Address: Department of Radiology
Lucile Salter Packard Children's Hospital
Stanford University Medical Center
725 Welch Road
Palo Alto, CA 94304
E-Mail: pbarnes@stanford.edu **Phone:** 650-497-8601
Place of Birth: Oklahoma City, Oklahoma, USA **Fax:** 650-497-8745

Education:

1965-1969	Letters / Pre-Medicine	University of Oklahoma, Norman, OK
1969-1973	Doctor of Medicine	University of Oklahoma College of Medicine, Oklahoma City, OK

Postdoctoral Training:

Residency:

1973-1976 Diagnostic Radiology, University of Oklahoma College of Medicine, Oklahoma City, Oklahoma

Fellowship:

1976-1977 Fellow in Pediatric Neuroradiology and Cardiovascular Radiology, Children's Hospital and Harvard Medical School, Boston, MA

Licensure and Certification:

1973	Federal Licensure Examination Certificate
1974	Oklahoma State Board of Medical Examiners
1977	American Board of Radiology Certificate in Diagnostic Radiology
1986	Commonwealth of Massachusetts Board of Registration in Medicine
2000	Medical Board of California C50437
1995	American Board of Radiology Certificate of Added Qualifications in Neuroradiology
2008	American Board of Radiology Maintenance of Certification in Neuroradiology

Academic Appointments:

1976-1977	Instructor in Radiology, University of Oklahoma College of Medicine
1977-1986	Lecturer in Radiologic Technology, University of Oklahoma College of Health
1977-1982	Assistant Professor of Radiology, University of Oklahoma College of Medicine
1980-1986	Adjunct Faculty, Radiologic Technology, Oscar Rose Junior College
1980-1986	Clinical Assistant Professor of Neurosurgery, University of Oklahoma College of Medicine
1982-1986	Associate Professor of Radiology, University of Oklahoma College of Medicine
1987-1992	Assistant Professor Radiology, Harvard Medical School
1992-2000	Associate Professor of Radiology, Harvard Medical School

2000-	Clinical Associate Professor of Radiology, Stanford University Medical Center
2002-	Associate Professor of Radiology, Stanford University Medical Center
2007-	Professor of Radiology, Stanford University Medical Center

Hospital and Affiliated Institution Appointments:

1977-1986	Pediatric Radiologist, Neuroradiology and Cardiovascular Radiology, Oklahoma Children's Memorial Hospital, Oklahoma City, Oklahoma
1977-1986	Consulting Radiologist, Oklahoma Memorial Hospital and Veterans Administration Hospital, Oklahoma City, Oklahoma
1984-1986	Consulting Radiologist, Oklahoma Diagnostic Imaging Center, Oklahoma City, Oklahoma
1987-1991	Associate Radiologist, Neuroradiology, The Children's Hospital, Boston, MA
1987-2000	Consulting Radiologist, Brigham and Women's Hospital, Beth Israel Hospital, New England Deaconess Hospital, Dana Farber Cancer Institute, Boston, MA
1990-1997	Clinical Coordinator, Magnetic Resonance Imaging, Children's Hospital, Boston, MA
1992-1995	Chief, Section of Neuroradiology, Department of Radiology, Children's Hospital, Boston, MA
1995-1999	Chief, Division of Neuroradiology, Department of Radiology, Children's Hospital, Boston, MA
1995-2000	Board of Directors, Children's Hospital Radiology Foundation, Inc.
1996-2000	Clinical Executive Committee, Department of Radiology, Children's Hospital, Boston, MA
1997-1998	Associate Director of CT, Department of Radiology, Children's Hospital, Boston, MA
1997-1999	Director of MRI, Department of Radiology, Children's Hospital, Boston, MA
1999-2000	Director, Division of Neuroradiology, Department of Radiology, Children's Hospital, Boston, MA
1999-2000	Treasurer, Children's Hospital Radiology Foundation, Inc.
1999-2000	Associate Chief for Clinical Operations, Department of Radiology, Children's Hospital, Boston, MA
2000	Senior Associate Neuroradiologist, Department of Radiology, Beth Israel Deaconess Medical Center, and Harvard Medical Faculty Physicians, Inc.
2000-	Staff Physician, Pediatric Neuroradiologist, Lucile Salter Packard Children's Hospital and Stanford University Medical Center
2001-	Interim Director, Pediatric Radiology, Lucile Salter Packard Children's Hospital (Jun-Aug./ JCAHO Survey)
2002-	Chief, Section of Pediatric Neuroradiology, Lucile Salter Packard Children's Hospital, Stanford University Medical Center, Palo Alto, CA
2002-	Co-Medical Director, MRI/CT Center, Lucile Salter Packard Children's

Hospital

Other Professional Positions and Major Visiting Appointments:

1988 Visiting Professor, The Western Pennsylvania Hospital, Pittsburg, PA

1989 Visiting Professor, New England Medical Center and Tufts University Medical School, Boston, MA

1989 Visiting Professor, Akron Children's Hospital, Akron General Hospital, and Northeastern Ohio Universities College of Medicine, Akron, Ohio

1990 Visiting Professor, Rhode Island Hospital and Brown University College of Medicine, Providence, R.I.

1991 Visiting Professor, University of Massachusetts Medical Center and Medical School, Worcester, MA

1993 Visiting Professor, Columbus Children's Hospital and the Ohio State University Hospitals, Columbus, OH

1993 Visiting Professor, Christchurch Hospital, University of Otago, Christchurch, New Zealand

1993 Visiting Professor, Royal Children's Hospital, University of Melbourne, Melbourne, Australia

1993 Visiting Professor, Royal Alexandra Hospital for Children, University of Sydney, Sydney, Australia

1993 Visiting Professor, Prince of Wales Children's Hospital, University of New South Wales, Sydney, Australia

1997 Visiting Professor, Montreal Children's Hospital, Montreal General Hospital, Montreal Neurologic Institute, McGill University, Montreal, Quebec, Canada

1998 Visiting Professor, Children's Hospital of Pittsburgh, University of Pittsburgh, Pittsburgh, PA

1998 Visiting Professor, William Beaumont Hospital, Royal Oak, MI

2000 Visiting Professor, Rhode Island Hospital and the Hasbro Children's HospitalBrown University School of Medicine, Providence, RI

2000 Visiting Professor, Massachusetts General Hospital, The Mass General Hospital for Children, and Harvard Medical School, Boston, MA

2008 Visting Professor, Department of Radiology, Duke University Medical Center, Durham NC.

2009 Visiting Professor, Department of Radiology, Hospital for Sick Children, University of Toronto, Toronto Ontario Canada.

2010 Visiting Professor, Department of Radiology, University of Arizona Medical Center, Tucson AZ.

2010 Visiting Professor, Department of Radiology, Vancouver General Hospital, BC Children's Hospital, University of British Columbia, Vancouver BC, Canada.

Hospital and Health Care Organization Service Responsibilities:

1977-1986 Staff Pediatric Radiologist and Section Chief, Pediatric Neuroradiology and Cardiovascular Radiology, Oklahoma Children's Memorial Hospital

1987-1992	Associate Radiologist, Neuroradiology, The Children's Hospital, Harvard Medical School, Boston, MA
1992-1995	Chief, Section of Neuroradiology, Department of Radiology, Children's Hospital, Boston, MA
1995-2000	Chief, Division of Neuroradiology, Department of Radiology, Children's Hospital, Boston, MA
1997-1998	Associate Director of CT, Department of Radiology, Children's Hospital, Boston, MA
1997-1999	Director of MRI, Department of Radiology, Children's Hospital, Boston, MA
1999-2000	Director, Division of Neuroradiology, Department of Radiology, Children's Hospital, Boston, MA
1999-2000	Associate Chief for Clinical Operations, Department of Radiology, Children's Hospital, Boston, MA
2000-	Pediatric Neuroradiologist, Lucile Salter Packard Children's Hospital and Stanford University Medical Center
2001-	Section Chief, Pediatric Neuroradiology, Lucile Salter Packard Children's Hospital, Stanford University Medical Center
2001-	Interim Director, MRI/CT Center, Lucile Salter Packard Children's Hospital, Stanford University Medical Center
2002-	Interim Director, Pediatric Radiology, Lucile Salter Packard Children's Hospital (Jun-Aug./ JCAHO Survey)
2002-	Chief, Section of Pediatric Neuroradiology, Lucile Salter Packard Children's Hospital, Stanford University Medical Center, Palo Alto, CA
2002-	Medical Co-Director, MRI/CT Center, Lucile Salter Packard Children's Hospital

Major Administrative Responsibilities:

1984-1986	Clinical Project/Program Consultant, Oklahoma Diagnostic Imaging Center, University of Oklahoma Health Sciences Center, Oklahoma City, Oklahoma
1985-1986	Clinical Project/Program Director, Oklahoma Teaching Hospitals, Magnetic Resonance Center
1987-1990	Clinical Coordinator, The Children's Hospital MRI Determination-Of-Need Process, Department of Public Health, The Commonwealth of Massachusetts, DON Certification, Jan. 1988.
1987-1990	Clinical Coordinator for MRI, The Children's Hospital and The Joint Center for Magnetic Resonance Imaging
1990-1997	Clinical Coordinator, Children's Hospital MRI Service.
1992-1995	Chief, Section of Neuroradiology, Department of Radiology, Children's Hospital, Boston, MA

1992-1999	Co-Director, Combined Neuroradiology Fellowship Program, Brigham & Women's Hospital, Beth Israel Hospital, Children's Hospital, New England Deaconess Hospital, Boston, MA
1992-1999	Director, Pediatric Neuroradiology Fellowship Program, Department of Radiology, Children's Hospital, Boston, MA
1995-2000	Chief, Division of Neuroradiology, Department of Radiology, Children's Hospital, Boston, MA
1996-2000	Board of Directors, Children's Hospital Radiology Foundation, Inc (CHRFI), Children's Hospital, Boston, MA
1996-2000	Clinical Executive Committee, Department of Radiology, Children's Hospital, Boston, MA
1997-1998	Associate Director of CT, Department of Radiology, Children's Hospital, Boston, MA
1997-1999	Director of MRI, Department of Radiology, Children's Hospital, Boston, MA
1998-1999	Chair, Bylaws Committee, Children's Hospital Radiology Foundation, Inc (CHRFI), Children's Hospital, Boston, MA
1999-2000	Treasurer, Children's Hospital Radiology Foundation, Inc.
1999-2000	Director, Division of Neuroradiology, Department of Radiology, Children's Hospital, Boston, MA
1999-2000	Associate Chief for Clinical Operations, Department of Radiology, Children's Hospital, Boston, MA
2000-	Pediatric Neuroradiologist, Lucile Salter Packard Children's Hospital and Stanford University Medical Center
2001-	Interim Director, Pediatric Radiology, Lucile Salter Packard Children's Hospital (Jun-Aug./ JCAHO Survey)
2002-	Chief, Section of Pediatric Neuroradiology, Lucile Salter Packard Children's Hospital, Stanford University Medical Center, Palo Alto, CA
2002-	Medical Co-Director, MRI/CT Center, Lucile Salter Packard Children's Hospital

Major Committee Assignments:

Hospital and Medical School:

1977-1981	Safety Committee, Oklahoma Children's Memorial Hospital
1977-1986	Neonatal Care Committee, Oklahoma Children's Memorial Hospital
1977-1986	Utilization Review Committee, Oklahoma Children's Memorial Hospital

1979-1986	Education and Research Committee, Oklahoma Children's Memorial Hospital
1984-1985	Chairman, State of Oklahoma Teaching Hospitals Task Force on Magnetic Resonance, Oklahoma City, OK
1985-1986	Quality Assurance Committee, Oklahoma Children's Memorial Hospital
1988-1990	Chairman, Joint Center for Magnetic Resonance Imaging, Consortium Clinical and Research Committee, Boston, MA

1988-2000	Pediatric Brain Tumor Working Group, The Children's Hospital and Dana-Farber Cancer Institute, Boston
1988	Steering Committee, Magnetic Resonance Imaging, Department of Radiology, The Children's Hospital, Boston
1989-1991	Chair, Radiology Quality Assurance/Quality Improvement Audit Committee, Children's Hospital, Boston
106.	Radiology Quality Improvement/Risk Management Committee, Children's Hospital, Boston
1992-	Neuroradiology Consultant, Child Protection Service, Children's Hospital, Boston
1992-2000	Department of Radiology Sedation & Contrast Media Committee, Children's Hospital, Boston
1996	Review of the Department of Neurology, Ad Hoc Review Committee, Children's Hospital, Boston
1998-1999	Neuroscience Business Planning Steering Committee and Marketing Team, Children's Hospital, Boston
1998-1999	Harvard Medical School Information Technology Initiative, Hospital and Clinical Linkages Committee, Harvard Medical School and Children's Hospital, Boston
1991-1999	Representative, Department of Radiology, Physician's Leadership Council of the Physician's Organization, Children's Hospital, Boston
2000-	Sedation Committee, Lucile Salter Packard Childrens Hospital at Stanford, Palo Alto, CA
2000-	MR / CT Imaging Facility Planning Committee, Lucile Salter Packard Childrens Hospital at Stanford, Palo Alto, CA
2000-	6-Sigma GEMS MR Capacity Committee, Stanford University Medical Center, Palo Alto, CA.
2005-	Phases I, II LPCH Expansion Committee, Imaging.

Regional:

1985-1986	Consultant on MRI, Oklahoma Health Planning Commission, Technical Advisory Committee, Oklahoma City, OK
2008-	Member, Child Abuse Task Force, SCAN Team, Lucile Packard Children's Hospital, Stanford University Medical Center, and Santa Clara Valley Medical Center.

National:

1987-1999	Quality Assurance Review Center, National Brain Tumor Committee, and Diagnostic Imaging Committee, Pediatric Oncology Group - High-risk Medulloblastomas, Providence RI
1991-1993	Pediatric Medical Advisory Board for MRI, General Electric Medical Systems.
1991-2000	Member, Neurology Major Test Committee, American Board of Psychiatry and Neurology, National Board of Medical Examiners, Philadelphia, PA

1998 Expert Panel Participant, Evidence-Based Guideline Development for the Management of Children Younger than Two Years of Age with Minor Head Trauma, Packard Foundation.

2000- Expert Panel Participant, Evidence-Based Neuroimaging in the Neonate-Practice Parameter Development Committee, American Academy of Neurology.

2005- Neuroradiologic Consultant / Central Reviewer, Neuroimaging and Neurodevelopmental Outcome, SUPPORT Multicenter Project, Neonatal Research Network, National Institute of Child Health and Human Development (NICHD).

2006- Neuroradiologic Consultant / Central Reviewer, Intervention Trial of Hypothermia for Term HIE Multicenter Project, Neonatal Research Network, National Institute of Child Health and Human Development (NICHD).

2007-2008 Chair, Child Abuse Task Force, Society for Pediatric Radiology.

Professional Societies and Offices:

1977-1986 Oklahoma County Medical Society
1977-1986 Oklahoma State Medical Association
1977-1986 Central Oklahoma Radiological Society
1977-1986 Oklahoma State Radiological Association
1977-1986 Central Oklahoma Pediatric Society
1977-1986 Oklahoma City Clinical Society
1977-1986 Oklahoma Neurological Society
1977- American Medical Association
1977- Radiologic Society of North America
1977- American College of Radiology
1980-1986 Rocky Mountain Neurosurgical Society
1980- Society for Pediatric Radiology
1980- American Society of Neuroradiology
1980- American Roentgen Ray Society
1987- New England Roentgen Ray Society
1987- Boston Neuroradiology Club
1987- Boston Pediatric Radiology Club
1987- Massachusetts Radiological Society
1988-1998 Society of Magnetic Resonance Imaging
1991-1992 Member, Pediatric Neuroradiology Subcommittee on Training and Practice Standards, American Society of Neuroradiology
1991- The Kirkpatrick Society
1992-1996 Chair, Pediatric Neuroradiology Committee, Society for Pediatric Radiology
1992-1998 Chair, Pediatric Neuroradiology Subcommittee on Training and Standards, American Society of Neuroradiology

1992-1993	Co-Founder and member-at-large, Steering Committee, Pediatric Neuroradiology Section of the American Society of Neuroradiology - the American Society of Pediatric Neuroradiology
1993-1995	Member-at-Large, Executive Committee, American Society of Pediatric Neuroradiology, and alternate Representative to Subspecialty Council, American Society of Neuroradiology
1995-1996	Treasurer, American Society of Pediatric Neuroradiology
1996-1997	Secretary and Chair, Membership Committee, American Society of Pediatric Neuroradiology
1996	Chair, Subcommittee "Standard for Cranial Computed Tomography in Infants and Children", The Society for Pediatric Radiology and American College of Radiology

1997	Chair, Subcommittee "Standard for Cranial Magnetic Resonance Imaging in Infants and Children", The Society for Pediatric Radiology and American College of Radiology
1996	Member, Subcommittee "Standard for Sedation/Analgesia in Pediatric Radiology" (M. Cohen, Chair), The Society for Pediatric Radiology and American College of Radiology
1997-1998	Vice President, President-Elect, and Chair, Nominating/Award Committee, American Society of Pediatric Neuroradiology
1998	Member, Caffey Awards Committee, Society for Pediatric Radiology 41st Annual Meeting, Tucson, AZ, May 7-9
1998	Chair, Derek Harwood-Nash Award Committee, American Society of Pediatric Neuroradiology, American Society of Neuroradiology 36th Annual Meeting, Philadelphia, PA, May 17-21
1998-1999	President and Chair, Program/Education Committee, American Society of Pediatric Neuroradiology
1998-1999	Member, Executive Committee, Program Committee, Clinical Practice Committee, Clinical Outcomes Research Committee, American Society of Neuroradiology
1999-2000	Chair, Board of Directors, American Society of Pediatric Neuroradiology
2000-	Chair, Standards and Guidelines Committee, American Society of Pediatric Neuroradiology
2000-	Member, Child Abuse Committee, Society for Pediatric Radiology
2007	Chair, Child Abuse Task Force, Society for Pediatric Radiology
2008-	Member, Child Abuse Task Force, Society for Pediatric Radiology
2008-	Member, Neuroradiology Committee, Society for Pediatric Radiology

Editorial Boards:

1988-	Reviewer, Radiology (journal of the Radiological Society of North America)
1988-	Reviewer, American Journal of Neuroradiology (journal of the American Society of Neuroradiology)
1991-	Editorial Board, Reviewer, Journal of Child Neurology

1991-	Reviewer, American Journal of Roentgenology (American Roentgen Ray Society)
1993-	Reviewer, Neuroradiology
1993-	Reviewer, Pediatrics
1993-	Reviewer, Journal of Pediatrics
1994-	Editorial Board, Reviewer, Pediatric Radiology (Journal of The Society for Pediatric Radiology and the European Society for Pediatric Radiology)
1995-1997	Associate Editor for Pediatric Neuroradiology, International Medical Image Registry
1995-	Reviewer, Journal of Computed Assisted Tomography
1997-	Reviewer, Neurology

Awards and Honors:

1969	Letzeiser Honor List, University Of Oklahoma
1972	Alpha Omega Alpha
1973	Graduation with Honors, Doctor of Medicine, University of Oklahoma College of Medicine

1995	Derek Harwood-Nash Outstanding Pediatric Neuroradiology Paper: **Tzika AA**, Barnes PD (mentor), Tarbell NJ, Nelson SJ, Scott RM. "Multivoxel proton spectroscopy of childhood brain tumors", presentation at ASNR 33rd Annual Meeting, Chicago, IL.
1996	Spirit Award, Children's Hospital, Boston, MA.
1996	Honorary Member, Australasian Society of Pediatric Imaging
1997	Kirkpatrick Young Investigator Award: **Alberico RA**, Barnes PD (mentor), Robertson RL, Burrows PE. "Dynamic cerebrovascular imaging in pediatric patients with use of helical CT angiography", paper presentation at the Society for Pediatric Radiology 40th Annual Meeting, St. Louis, MO.
1997	Cum Laude Citation (Scientific Exhibit): **Levine D**, Barnes PD (mentor), Madsen JR, Hulka CA, Li W, Edelman RR. "HASTE MR imaging improves sonographic diagnosis of fetal central nervous system anomalies", scientific exhibit and paper presentation at Radiological Society of North America 83rd Scientific Assembly and Annual Meeting, Chicago, IL.
1998	John A. Kirkpatrick Jr. Teaching Award, Pediatric Radiology Fellowship Program, Department of Radiology, Children's Hospital and Harvard Medical School, Boston, MA.
1999	Derek Harwood-Nash for Outstanding Pediatric Neuroradiology Paper: **Robertson RL**, Ben-Sira L, Schlaug G, Maier SE, Mulkern RV, Duplessis A, Barnes PD (mentor), Robson CD. Line scan diffusion imaging of the brain in neonatal cerebral infarction, paper presented at the ASNR/ASPNR Annual Meeting, San Diego, CA.

2000 Medical Intelligence Corporation Scientific Achievement Award for Outstanding Contributions to Neuroimaging in Enhancing Understanding of Timing of Fetal Injury, Las Vegas, Nevada, October 19, 2000.

2000 Outstanding Head & Neck Radiology Paper: **Robson CD**, Mulliken JB, Robertson RL, Proctor MR, Barnes PD (mentor). Prominent basal emissary foramina in syndromic craniosysnostosis – correlation with phenotype and molecular diagnosis, paper presented at the ASNR/ASPNR/ASHNR Annual Meeting, Atlanta, GA, May 2000.

2001 Award of Appreciation for Service & Leadership as Past President 1998-1999, The American Society of Pediatric Neuroradiology, American Society of Neuroradiology 39th Annual Meeting, Boston, MA, April 23, 2001.

2003 Stanford B. Rossiter Senior Faculty of the Year 2002-2003. Outstanding Contributions to Resident Education, Compassionate Patient Care, and Research, Department of Radiology, Stanford University Medical Center.

2005 Senior Faculty of the Year 2004-2005. Outstanding Contributions to Resident Education, Compassionate Patient Care, and Research, Department of Radiology, Stanford University Medical Center.

2006 Senior Faculty of the Year 2005-2006. Outstanding Contributions to Resident Education, Compassionate Patient Care, and Research, Department of Radiology, Stanford University Medical Center.

2008 The Herman Grossman Lecturer, Department of Radiology, Duke University Medical Center, In Appreciation for Your Contributions to Pediatric Radiology and the Eleventh Annual Herman Grossman Lecturer, April 10, 2008.

2010 Caffey Award Scientific Paper. Bammer R, Holdsworth S, Skare S, Yeom K, Barnes P. Clinical evaluation of readout-segmented-EPI for diffusion-weighted imaging. Scientific Paper Presentation Society for Pediatric Radiology Annual Meeting, Boston MA April 2010.

2010 Caffey Award Scientific Paper. Skare S, Holdsworth S, Yeom K, Barnes P, Bammer R. High-resolution motion-corrected diffusion-tensor imaging (DTI) in infants. Scientific Paper Presentation Society for Pediatric Radiology Annual Meeting, Boston MA April 2010.

2010 Caffey Award Scientific Paper. Bammer R, Holdsworth S, Skare S, Yeom K, Barnes P. 3D SAP-EPI in motion-corrected fast susceptibility weighted imaging (SWI). Scientific Paper Presentation Society for Pediatric Radiology Annual Meeting, Boston MA April 2010.

2010 Caffey Award Scientific Paper. Bammer R, Holdsworth S, Skare S, Yeom K, Barnes P. T1-weighted 3D SAP-EPI for use in pediatric imaging. Scientific Paper Presentation Society for Pediatric Radiology Annual Meeting, Boston MA April 2010.

2011 An America's Top Doctor - US News & World Report (Top 1% of neuroradiologists in the nation for 5 years, Castle Connolly Medical Ltd.) <health.usnews.com/top-doctors>.

RESEARCH, TEACHING, AND CLINICAL CONTRIBUTIONS

Research Activities:

1985	Surface Coil Magnetic Resonance Imaging Clinical Research and Development Project, Dan Galloway, M.D., Patrick Barnes, M.D., and John Prince, Ph.D., Principal Co-Investigators, Oklahoma Diagnostic Imaging Center, University of Oklahoma Health Sciences Center and General Electric Medical Systems, Inc. (IRB#02926).
1986	Magnetic Resonance Imaging and the Evaluation of Morphologic and Biochemical Abnormalities. Patrick Barnes, M.D., and John Prince, Ph.D., Radiology, Principal Co-Investigators, University of Oklahoma Health Sciences Center (IRB#02958), Oklahoma Teaching Hospitals and Philips Medical Systems, Inc. (FDA-PMA-#P840063A).
1987-1991	Pre-Radiation Chemotherapy in the Treatment of Children with Brain Stem Neoplasia, Evaluation with CT and MRI, Pediatric Oncology Group, Cynthia Kretschmer, M.D., The Massachusetts General Hospital, Coordinator (POG8833); Neuroradiologic consultant.

1988-1997	Infant Heart Surgery: CNS Sequelae of Circulatory Arrest, evaluation including Magnetic Resonance Imaging, Jane Newburger, M.D., Principal
1988-1998	Investigator, Department of Cardiology, The Children's Hospital (NIH 1R01HL4178601); Neuroradiologic consultant.
1990-1991	Fast Spin Echo Magnetic Resonance Neuroimaging Project, Patrick Barnes, M.D. and Robert Mulkern, Ph.D., Principal Investigators, Children's Hospital, General Electric Medical Systems, Inc. (CH90-10-099).
1990-1997	Chemotherapy and Radiation Therapy in the Treatment of Seeding Tumors of the CNS in Children, Amy Billett, M.D. and Nancy Tarbell, M.D., Study Chairpersons (DFCI 90-114); Neuroradiologic consultant.
1990-1997	Radiosensitizer Chemotherapy (Etanidazole-SR 2508) and Radiotherapy in Children with Brain Stem Gliomas, Nancy Tarbell, M.D., Study Chairperson (DFCI 90-080); Neuroradiologic consultant.
1991-1999	High Stage Medulloblastomas, Quality Assurance Review Center, Pediatric Oncology Group, Nancy Tarbell, M.D. and Patrick D. Barnes, M.D., Co-Principal Investigators
1992-1997	Stereotactic Radiotherapy for Pediatric Brain Tumors, Nancy Tarbell, M.D., Study Chairperson (DFCI 92-077); Neuroradiologic consultant.
1992-1997	Stereotactic Radiation Therapy for Recurrent or Metastatic CNS Tumors, J. Fontanesi, M.D., J. Loeffler, M.D., P. Barnes, M.D., et al, Coordinators, Pediatric Oncology Group SRS #9373 Protocol.
1994-2000	MR-Techniques in the Assessment of the Newborn Brain, Steven A. Ringer, M.D., Ph.D., Petra S. Huppi, M.D., Co-Principal Investigators, JPN Clinical Research Initiative and Reynolds-Rich-Smith Fellowship; Neuroradiologic Consultant.
1996	Efficacy And Cost-Effectiveness of Fast-Screening Brain MRI Versus Conventional MRI in Children Suspected of Having a Brain Tumor L.

Santiago Medina, M.D., Patrick D. Barnes, M.D., A.D. Paltiel, M.D., David Zurakowski, The Society for Pediatric Radiology Research and Education Fund Grant.

1996-2000 Metabolic and Hemodynamic MR Characterization of Pediatric Brain Tumors, A. Aria Tzika, Principal Investigator, Patrick Barnes, M.D., et al, Co-Investigator, American Cancer Society (EDT-80188)

1996-2000 Rehabilitation, Brain Lesions, and Movement in Infants, Edward E. Tronick, Ph.D., Linda Fetter, Ph.D., Alan Leviton, M.D., Co-Principal Investigators (NIH RO1); Neuroradiologic Consultant.

1996-2000 Ultrafast MRI of the Fetal Brain, D. Levine, M.D., Principal Investigator (NIH R29 NS37945-01), Beth Israel Deaconess Medical Center; Neuroradiologic Consultant.

Page 10

1999-2000 Pediatric Brain Tumor Consortium, M. Kieran, M.D., Nancy J. Tarbell, M.D. Co-Principal Investigators (NIH/NCI 1 U01 CA 81452-01), Children's Hospital, Massachusetts General Hospital, and Dana Farber Cancer Center; Member, Neuroradiology Committee and Senior Site Neuroradiologic Consultant.

1999-2000 Pediatric Centers for MRI Study of Normal Brain Development, NIH-NINDS-98-13, Michael Rivkin, M.D., principal investigator; Co-investigator and Consultant.

2001- PAR-98-017 (Reiss) NIMH Longitudinal MRI Study of Brain Development in Fragile X (7.5% effort funded).

2001- 2 R01 MH50047 (Reiss) NIMH Longitudinal Outcomes and Neuroimaging of Fragile X Syndrome (5% effort funded).

2001- Barth R, MRI of Fetal Ventriculomegaly.

2001 Arriagno R (NIH) Neonatal Diagnosis of Possible Brain Injury in Very Low Birth Weight Preterm Infants.

2001- Reiss et al. Velocardiofacial syndrome – neuroimaging.

2001- Reiss et al. Bipolar disorder – neuroimaging.

2001- Reiss et al. Coffin-Lowry syndrome – neuroimaging.

2002- Barnes P, et al. Stanford University Certification of Human Subjects Approval IRB Protocal ID 78050: Magnetic Resonance Imaging (MRI) of the Developing Central Nervous System (CNS), March 5, 2002.

2002- Diabetic Ketoacidosis Cerebral Edema Multicenter Study (N. Glaser et al [1% effort funded].

2006- 2U HD 27880-16 Van Meurs (PI). Project period: 04/01/06–03/31/11 NIH/NICHD *Multicenter Network of Neonatal Intensive Care Units Intervention Trial of Hypothermia for Term Hypoxic Ischemic Encephalopathy.* Role: Central MRI reader/Neuroimaging consultant

2006- 2U HD 27880-16 Van Meurs (PI). Project period: 04/01/06–03/31/11 NIH/NICHD *Multicenter Network of Neonatal Intensive Care Units Neuroimaging and Neurodevelopmental Outcome, SUPPORT Multi-Center Project* This project investigates the value of brain magnetic imaging (MRI) in predicting neurodevelopmental outcome in extremely low birthweight (ELBW) infants. Role: Central MRI reader / Neuroimaging consultant

2008 The Well-Nourished and Sleeping Preterm Infant Will Have Improved Brain (Ariagno). Development and Neurodevelopmental Outcome. The Gerber Foundation. Consultant. 08/01/2005-07/31/2008

2008- NIH 1R01 EB008706 Bammer (PI) Project period: 09/01/08 – 08/31/13 Effort: 4.5% ADC: $414,692 "Short Axis EPI MRI at High Field"

2008- Neuroradiologic Consultant / Central Reviewer, National Holoprosencephaly Project, The Carter Center.

2009- NIH 1R01 EB008706 Roland Bammer (PI); Project period: 09/01/08-08/31/13; Effort: 4.5% ; ADC: $414,692 ; "Short Axis EPI for Diffusion Tensor MRI at High Field."

2009- NIH 1R01 MH083972 Antonio Hardan (PI); Project period: 3/1/09 – 12-31-13; Effort: 4.5%; ADC: $391,595 ; "A Neuroimaging Study of Twin Pairs with Autism"

2009- LPCH Center for Brain Behavior Awards in Pediatric Neurosciences K. Yeom (PI); Project Period: 2009-2011; Effort: 1%; ADC: $145,000 "MR Imaging Correlates for Cognitive Dysfunction in Pediatric Medulloblastoma Treated with Cranial Irradiation."

Teaching:

Local Contributions:

1976-1979 Course Director and Conference Leader, Pediatric House Staff Core Lecture Series, Pediatric Radiology, Oklahoma Children's Memorial Hospital

1976-1980 Conference Co-leader, Monthly Orthopaedic Radiology-Pathology Conference, Oklahoma Teaching Hospitals

1977-1979 Physician Associates Radiology Lecture Series, College of Allied Health, University of Oklahoma

1977-1982 Conference Co-Leader, Weekly Pediatric Cardiology and Cardiac Surgery Conference

1977-1982 Conference Co-Leader - "Sickle Cell Anemia", Annual Clinical Demonstration for First Year Medical Students, College of Medicine, University of Oklahoma.

1977-1982 Pediatric Cardiac Cine-Angiocardiographic case review and consultation weekly with Pediatric, Pediatric Cardiology, Thoracic Surgery Staff, Residents and Fellows

1977-1985 Pediatric Grand Rounds, Oklahoma Children's Memorial Hospital.

1977-1986	Attending Physician and Conference Leader, Daily and Weekly Clinical Teaching Rounds, Children's Memorial Hospital, University of Oklahoma College of Medicine; Pediatric Radiology Film and Fluoroscopy Review with Radiology, Pediatric, Family Medicine Residents and Medical Students.
1977-1986	Pediatric Neuroradiology Case Review and Consultation daily with Neurosurgery, Neurology, Pediatric, and Adolescent Medicine Staff, Residents, Fellows and Medical Students
1977-1986	Pediatric Computed Tomography, Conventional Tomography, and Special Procedures case review and consultation daily with Pediatric, Pediatric Surgery, Adolescent Medicine, and Orthopedic Staff, Residents, Fellows and Medical Students
1977-1986	Elective Tutorials in Pediatric Neuroradiology and Cardiovascular Radiology for Pediatric, Radiology, Neurosurgery, Neurology and Pediatric Surgery Residents, Fellows, and Students
1977-1986	Weekly Diagnostic Radiology Residency Lecture Series, University of Oklahoma College of Medicine

1977-1986	Quarterly Radiologic Technology Inservice in Pediatric Neuroradiology and Cardiovascular Radiology Special Procedures
1977-1986	Co-Leader, Weekly Neurosurgery/Neurology Grand Rounds, Oklahoma Teaching Hospitals and St. Anthony Hospital, Oklahoma City, Oklahoma
1978-1982	Course Lecturer, Annual Department of Radiological Sciences Continuing Medical Education Courses, University of Oklahoma Health Sciences Center
1978-1985	Lecturer, Annual Graduate Physics Seminar, College of Allied Health, University of Oklahoma Health Sciences Center
1979-1981	Lecturer, Annual Radiology Grand Rounds, Oklahoma Teaching Hospitals
1980-1985	Lecturer, Pediatric Surgery Core Lecture Series in Pediatric Radiology, Oklahoma Children's Memorial Hospital
1981-1986	Lecturer, Neurology/Pediatric Neuroradiology Lecture Series, Oklahoma Teaching Hospitals
1982-1985	Participant, Senior Radiology Resident Pre-Board Examinations, University of Oklahoma College of Medicine
1982-1986	Lecturer, Pediatric House Staff Core Lecture Series in Pediatric Radiology, Oklahoma Children's Memorial Hospital
1983-1986	Course Developer and Director, Resident Final Examination in Pediatric Radiology, University of Oklahoma College of Medicine
1985-1986	Oklahoma Diagnostic Imaging Center Lecture Series, Course Co-Developer and Co-Director
1985-1986	Oklahoma Teaching Hospitals Department of Radiological Sciences, Magnetic Resonance Imaging Lecture Series (Course Developer and Director)

1986	"Magnetic Resonance Imaging for the Referring Physician", Continuing Medical Education Seminar, Program Co-Director, Session Moderator, and Lecturer, Oklahoma Teaching Hospitals and the University of Oklahoma College of Medicine
1987-	Daily Neuroradiology Case Review and Consultation with Pediatric and Adolescent Medicine, Neurology, Neurosurgery, Radiology, Oncology, Radiation Therapy, Orthopedic, ORL/Head and Neck Surgery, Ophthalmology, Plastic Surgery, Oral Surgery, and Neuropathology Staff, Fellows, Residents, Medical Students, and visitors, Children's Hospital, Boston, MA
1987-	Weekly Pediatric Neurology-Neuroradiology Rounds with Staff, Fellows, Residents, Medical Students, and visitors, Conference Co-Leader, Children's Hospital, Boston, MA
1987-	Weekly Pediatric Neurosurgery-Neuroradiology Rounds with Staff, Fellows, Residents, Medical Students, and visitors, Conference Co-Leader, Children's Hospital, Boston, MA

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1987-	Weekly Pediatric Neuroncology-Neuroradiology Rounds with Pediatric Oncology, Radiation Oncology, and Neurosurgery Staff, Fellows, Residents, Medical Students, and visitors (The Children's Hospital and Dana-Farber Cancer Institute), Conference Co-Leader, Children's Hospital, Boston, MA
1987-	Weekly Longwood Medical Area Neuroradiology Conference with Staff, Fellows, Residents, Medical Students, and visitors (The Children's Hospital, Brigham & Women's Hospital, Beth Israel Hospital, New England Deaconess Hospital, Dana-Farber Cancer Institute), Conference Co-Leader, Children's Hospital, Boston, MA
1987-	Monthly Pediatric ORL/Head & Neck Radiology Rounds with Staff, Fellows, Residents, Medical Students, and visitors, Conference Co-Leader, Children's Hospital, Boston, MA
1987-	Monthly Pediatric Radiology Difficult Case Conference (Risk Management and Quality Improvement) with Staff, Fellows, Residents, Medical Students, and visitors, Children's Hospital, Boston, MA
1987-	Monthly Boston Area Neuroradiology Club Case Conference with Staff, Fellows, Residents, Medical Students, and visitors (Massachusetts General Hospital)
1987-	Pediatric Neuroradiology Annual Lecture Series, Course Co-Director and Lecturer, for Staff, Fellows, Residents, Medical Students, and visitors.
1987-	Pediatric Neuroradiology Introductory Lectures for Harvard Medical Students and Rotating Radiology Residents, Radiology, Children's Hospital, Boston, MA
1987-1988	Cardiac Radiology Lecture Series, Course Developer and Lecturer, Radiology, Children's Hospital, Boston, MA

1987-1990	Magnetic Resonance Imaging Lecture Series, Course Developer, Director, and Lecturer, Radiology, Children's Hospital, Boston, MA
1987	Invited Lecturer, MRI in Pediatric Neuroradiology, Radiology Grand Rounds, Brigham and Women's Hospital, Boston, MA
1987	Lecturer, "Scoliosis and the Neuroradiologist", "The Impact of MR on Central Nervous System Imaging in Childhood", and "Magnetic Resonance-Diagnostic Imaging Principles", The Children's Hospital and Harvard Medical School Post- Graduate Course, Pediatric Imaging, Boston, MA
1987	Lecturer, "Pediatric Central Nervous System Imaging, The Brigham & Women's Hospital and Harvard Medical School Post-graduate Course, CT and MRI Update, Cambridge, MA
1988	Invited Lecturer, "MRI in Pediatric Neuroncology", Joint Center for Radiation Therapy Grand Rounds, Children's Hospital, Boston, MA, June 8, 1988
1988	Invited Lecturer, "Magnetic Resonance in Pediatric Imaging", The Children's Hospital and Harvard Medical School Post-graduate Course, Pediatric Medicine

1988	Lecturer, "Magnetic Resonance Imaging of the Pediatric Central Nervous System, Part I - Brain" ; "Magnetic Resonance Imaging of the Pediatric Central Nervous System, Part II – Spine", & Case Review Panel, The Brigham & Women's Hospital and Harvard Medical School Post-graduate Course, CT and MRI Update, Cambridge, MA
1988	Invited Lecturer, "Magnetic Resonance Imaging", The Children's Hospital, Massachusetts General Hospital, and Harvard Medical School Post-graduate Course, Child Neurology
1989	Lecturer, "Magnetic Resonance in Pediatric Neuroimaging" ; "Magnetic Resonance Imaging in Spinal Dysraphism", The Brigham & Women's Hospital and Harvard Medical School Post-graduate Course, CT and MRI Update, Boston, MA
1989	Invited Lecturer, "Magnetic Resonance in Pediatric and Adolescent Neuroimaging", The Children's Hospital, Massachusetts General Hospital, and Harvard Medical School Post-graduate Course, Child Neurology
1990	Lecturer, "MR Imaging of the Pediatric Central Nervous System", The Brigham & Women's Hospital and Harvard Medical School Post-graduate Course, CT and MRI Update, Cambridge, MA
1991	Invited Lecturer, "MRI Signal Patterns-I", & "MRI Signal Patterns-II", Radiology Resident Lecture Series, University of Massachusetts Medical Center and Medical School, Worcester, MA, March 8, 1991
1991	Invited Lecturer, "Pediatric Spine Imaging", Radiology Grand Rounds, University of Massachusetts Medical Center and Medical School, Worcester, MA, March 8, 1991

1991 Invited Lecturer, "MRI of Congenital Spine Lesions", Neurology Grand Rounds, University of Massachusetts Medical Center and Medical School, Worcester, MA, March 9, 1991

1991 Invited Lecturer, "MRI of the Pediatric Central Nervous System", Western Massachusetts Radiological Society, Holyoke, MA, Sept. 24, 1991

1991 Lecturer, "MR Imaging of the Pediatric Central Nervous System", The Brigham & Women's Hospital and Harvard Medical School Post-graduate Course, CT and MRI Update, Cambridge, MA

1991 Invited Lecturer, "MRI in the Pediatric CNS", Harvard Longwood Neurological Training Program Post-graduate Course, Intensive Review of Neurology

1991 Invited Lecturer, "MRI in Pediatrics", Anesthesiology Grand Rounds, Children's Hospital, Boston, MA, Dec. 18, 1991

1992 Invited Lecturer, "Pediatric Brain Tumors", Radiology Grand Rounds, Boston City Hospital, University Hospital, and Boston University Medical School, Boston, MA, Feb. 25, 1992

1991 Invited Lecturer, "Cerebral Dysgenetic Syndromes, Clinical and MRI Correlates", Child Neurology Course, Massachusetts General Hospital, Children's Hospital, and Harvard Medical School Post-Graduate Course, September 1992, Boston, MA

1992 Invited Lecturer, "Pediatric CNS Tumor Imaging", The Harvard Medical School Post-Graduate Course in Neurosurgery-Brain Tumors, November 30, Boston, MA

1993 Invited Lecturer, Massachusetts General Hospital and Harvard Medical School Radiology Review Course, "Congenital CNS Abnormalities". April, Cambridge, MA

1993 Lecturer, "Neuroimaging Techniques in Pediatrics", Child Psychiatry Lecture, Children's Hospital, Boston, MA, June 8,1993

1993 Lecturer, "Neuroimaging in Pediatrics", Radiologic Technologist Inservice Lecture, Children's Hospital, Boston, MA, June 23, 1993

1993 Lecturer, "Neuroimaging-The Pediatric Brain", The Children's Hospital and Harvard Medical School Post-Graduate Course in Practical Pediatric Radiology, July 29, Brewster, MA.

1993 Invited Lecturer, "Malformations of the Brain", "Posterior Fossa and Craniocervical Junction Anomalies", The Massachusetts General Hospital and Harvard Medical School Post-Graduate Course in Neuroradiology, September 21 and 22, Boston, MA

1994 Lecturer, "Pediatric Neuroimaging: The Brain", The Children's Hospital and Harvard Medical School Post-Graduate Course in Practical Pediatric Imaging: Update '94, August 4, New Seabury, MA

1994 Presenter, "Brain Tumors in Children", The Massachusetts General Hospital and Harvard Medical School Post-Graduate Course in Neuroradiology, October 3-7, Boston, MA

1994 Lecturer, "Pediatric Brain Imaging", The Brigham and Women's Hospital and Harvard Medical School Post-Graduate Course, Pediatric Brain Imaging, MRI and CT Update, October 27 and 28, Cambridge, MA

1995 Invited Lecturer, "Congenital CNS Abnormalities", Massachusetts General Hospital, Brigham and Women's Hospital, and Harvard Medical School Radiology Review Course, April, Cambridge, MA

1995 Lecturer, "Pediatric Brain Imaging- Protocols and Pitfalls", The Children's Hospital and Harvard Medical School Post-Graduate Course in Practical Pediatric Imaging: Update '95, July 26, New Seabury, MA

1995 Invited Lecturer, ""Inflammatory CNS Conditions in Childhood", "Spine and Spinal Cord Anomalies in Childhood", The Massachusetts General Hospital and Harvard Medical School Post-Graduate Course, Basic and Current Concepts in Neuroradiology, Head & Neck Radiology, and Neuro MRI, September 19 and 20, Boston, MA

1995 Moderator, Pediatric Neuroradiology Session, The Brigham and Women's Hospital and Harvard Medical School Post-Graduate Course, MRI and CT Update, October 12 and 13, Cambridge, MA

1995 Lecturer, "Pediatric CNS Imaging: Protocols & Pitfalls", "Developmental Brain Abnormalities", The Brigham and Women's Hospital and Harvard Medical School Post-Graduate Course, MRI and CT Update, October 12 and 13, Cambridge, MA

1996 Invited Lecturer, "Pediatric Neuroradiology", Massachusetts General Hospital, Brigham and Women's Hospital, and Harvard Medical School Radiology Review Course, April, Cambridge, MA

1996 Moderator, Pediatric Neuroradiology Session, The Children's Hospital and Harvard Medical School Post-Graduate Course in Practical Pediatric Imaging: Update 1996, July 22, Boston, MA

1996 Invited Lecturer, "Imaging of the Orbits and Sinuses: Part I", "Imaging of the Orbits and Sinuses: Part II", The Children's Hospital and Harvard Medical School Post-Graduate Course in Practical Pediatric Imaging: Update 1996, July 22, Boston, MA

1996 Invited Lecturer, "Congenital Brain Anomalies" and "Brain Tumors in Children", The Massachusetts General Hospital and Harvard Medical School Post-Graduate Course, Basic and Current Concepts in Neuroradiology, Head & Neck Radiology, and Neuro MRI, October 8, Boston, MA

1996 Moderator, Pediatric Neuroradiology Session, The Brigham and Women's Hospital and Harvard Medical School Post-Graduate Course, MRI & CT Update, October 25, Cambridge, MA

1996 Lecturer, "Hydrocephalus", The Brigham and Women's Hospital and Harvard Medical School Post-Graduate Course, MRI & CT Update, October 25, Cambridge, MA

1996 Invited Lecturer, "Imaging of Cranial and Intracranial Tumors of Childhood", The Brain Tumor Center, Brigham and Women's Hospital,

Children's Hospital, Joint Center of Radiation Therapy, and Dana Farber Cancer Institute, Tumors of the Central Nervous System Post-Graduate Course, November 25, Boston, MA

1997 Invited Lecturer, "Potential Problems and Pitfalls in Pediatric Neuroradiology", Boston University Medical Center, Department of Radiology Grand Rounds, March 20, Boston, MA

1997 Lecturer, "Imaging of Macrocephaly, Parts I and II", The Children's Hospital and Harvard Medical School Post-Graduate Course in Practical Pediatric Imaging: Update 1997, July 21, Boston, MA

1997 Invited Lecturer, "Brain Tumors in the Pediatric Age", and "Congenital and Developmental Conditions of the Spine and Spinal Cord", The Massachusetts General Hospital and Harvard Medical School Post-Graduate Course, Basic and Current Concepts in Neuroradiology, Head & Neck Radiology, and Neuro MRI, September 15 and 16, Boston, MA

1997 Moderator, Pediatric Neuroradiology Session, The Brigham and Women's Hospital and Harvard Medical School Post-Graduate Course, MRI & CT Update 1997, October 31, Boston, MA

1997 Lecturer, "Congenital Brain Anomalies--A Problem-Oriented Approach", The Brigham and Women's Hospital and Harvard Medical School Post-Graduate Course, MRI & CT Update 1997, October 31, Boston, MA

1997 Invited Lecturer, "Radiologic Diagnosis of Brain Tumors in Children", Joint Venture Neuroncology, The Partners Health Care System, Dana Farber Cancer Institute, and Harvard Medical School and Brain Tumor Management, November 24, Boston, MA

1997 Moderator, Pediatric Neuroradiology Session, Joint Venture Neuroncology The Partners Health Care System, Dana Farber Cancer Institute, and Harvard Medical School Post-Graduate Course, Tumors of the Central Nervous System and Brain Tumor Management, November 24, Boston, MA

1998 Invited Lecturer, The Brigham & Women's Hospital and Massachusetts General Hospital Radiology Review Post-Graduate Course, "Pediatric Neuroradiology", April 6, Cambridge, MA

1998 Invited Lecturer, "Congenital and Developmental Conditions of the Spine and Spinal Cord", The Massachusetts General Hospital and Harvard Medical School Post-Graduate Course, Basic and Current Concepts in Neuroradiology, Head & Neck Radiology, and Clinical Functional MRI and Spectroscopy, September 16, Boston, MA

1998 Moderator, Pediatric Neuroradiology Session, The Brigham and Women's Hospital and Harvard Medical School Post-Graduate Course, MRI/CT Update 1998,October 30, Boston, MA

1998 Lecturer, "Major Congenital Brain Anomalies", The Brigham and Women's Hospital and Harvard Medical School Post-Graduate Course, MRI/CT Update 1998, October 30, Boston, MA

1999 Invited Lecturer, "Neonatal MRI: New Techniques", Division of Newborn Medicine Clinical Conferences, Children's Hospital, January 4, Boston, MA

1999 Invited Speaker, Imaging of Brain Tumors in Children, Parents Workshop, Jimmy Fund Clinic, Dana-Faerber Cancer Institute, May 1, Boston, MA.

1999 Invited Speaker, Radiologic Diagnosis of Brain Tumors in Children, Tumors of the Central Nervous System: Management of Brain Tumors Post-graduate Course, Brigham and Women's Hospital, Massachusetts General Hospital, Children's Hospital, Dana-Faerber Cancer Institute, Harvard Medical School, September 13, Boston, MA

1999 Invited Speaker, Congenital and Developmental Conditions of the Spine and Spinal Cord, Neuroradiology, Head & Neck Radiology, Clinical Functional MRI and Spectroscopy Post-graduate Course, Massachusetts General Hospital, Massachusetts Eye & Ear Infirmary, Harvard Medical School, October 6, Boston, MA

1999 Invited Speaker, Potential Pitfalls in Pediatric Neuroradiology, and Session Moderator, Pediatric Neuroradiology Session, MRI/CT Update Post-graduate Course, Brigham & Women's Hospital, Harvard Medical School, October 29, Boston, MA

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2000 Invited Discussant, Pediatric Neuroncology, Neurosurgery, and Neurology Conferences, Department of Radiology, Massachusetts General Hospital, Jan.-Feb., Boston, MA

2000 Basic Technical and Biological Principles of Magnetic Resonance Imaging Lecture Series, Department of Radiology, Beth Israel Deaconess Medical Center, Feb.-May, Boston, MA

2000 Pediatric Neuroradiology Resident Pre-Board Review, Department of Radiology, Beth Israel Deaconess Medical Center, May, Boston, MA

2000- Daily Pediatric Neuroradiology and Head & Neck CT and MRI Case Review / Consultations with Fellows, Residents, Medical Students, and Visiting Physicians, Lucile Salter Packard Children's Hospital and Stanford University Medical Center, Palo Alto, CA

2000- Conference Co-Leader, Weekly Pediatric Neuroncology Conference, Lucile Salter Packard Children's Hospital at Stanford, Palo Alto,

2000- Conference Leader, Weekly Pediatric Neuroradiology, Neurology, and Neurosurgery Conference, Lucile Salter Packard Children's Hospital at Stanford, Palo Alto, CA

2000- Pediatric Neuroradiology Lectures, Neuroradiology Lecture Series, Department of Radiology, Stanford University Medical Center, Palo Alto, CA

2000- Faculty Participant, Weekly Neuroradiology Case Review / QI Conference Department of Radiology, Stanford University Medical Center, Palo Alto, CA

2000- Faculty Participant, Weekly Neurology Case Conference, Stanford University Medical Center, Palo Alto, CA

2000- Faculty Participant, Weekly Perinatal Conference, Lucile Salter Packard Children's Hospital at Stanford, Palo, Alto, CA

2000 Invited Lecturer, Pitfalls in Pediatric Neuroradiology, Neurosurgery Grand Rounds, Stanford University Medical Center, Palo Alto, CA Sept. 1, 2000

2000- Faculty Participant, International Perinatal Teleconferences (Hong Kong), Lucile Salter Packard Children's Hospital at Stanford, Palo Alto, CA

2000 Medical Student Clerkship Lecture, Pediatric Neuroradiology, Department of Radiology, Stanford University Medical Center, Palo, Alto, CA, Oct. 12, 2000

2000 Invited Lecturer, Imaging of Neonatal Encephalopathy, Neonatal Intensive Care Clinical Research Conference, Lucile Salter Packard Children's Hospital at Stanford, Palo Alto, CA, Oct. 16, 2000.

2001 Invited Lecturer, Potential Pitfalls in Pediatric Neuroradiology-The Impact of Advancing Neuroimaging Techniques, Department of Radiology, Stanford University Medical Center, Palo Alto, CA, Feb. 13, 2001.

2001 Faculty participant, Weekly Epilepsy Conference, Stanford University Medical Center, Palo Alto, CA.

2001- Monthly Pediatric Neuroradiology Lecture Series for Neurology Residents & Fellows, Stanford University Medical Center, Palo Alto, CA.

2001- Monthly Pediatric Neuroradiology Lecture Series for Neurosurgery Residents and Fellows Stanford University Medical Center, Palo Alto, CA.

2001- Monthly Pediatic Head & Neck Imaging Lecture Series for ORL/Head & Neck Residents and Fellows, Stanford University Medical Center, Palo Alto, CA.

2001- Pediatric Neuroradiology Lectures, Pediatric Radiology Lecture Series, Department of Radiology, Stanford University Medical Center, Palo Alto, CA.

<u>Regional, national, or international contributions</u>:

1988 Invited Lecturer, "Neurocutaneous Syndromes", & "Pediatric Spine Imaging-Spinal dysraphism", Western Pennsylvania Hospital, Pittsburg, PA, Nov. 3, 1988

1989 Invited Lecturer, "Pediatric Spine Imaging", New England Medical Center and Tufts Medical School, Feb. 9, 1989

1989 Invited Lecturer, "MRI-Basic Principles and Pediatric Applications", Akron Children's Hospital, Akron, OH, May 3, 1989

1989 Invited Lecturer, "MRI in Pediatric Spine Imaging", Northeast Ohio University Medical Center, Akron, OH, May 3, 1989

1989 Invited Lecturer, "MRI in Pediatric and Adolescent Neuroimaging", Akron Radiological Society, Akron, OH, May 3, 1989

1989 Invited Discussant, Neuroimaging-Neuropathology Correlation Conference, Akron Children's Hospital, Akron, OH, May 4, 1989

1989 Invited Lecturer, "Imaging of the Neurocutaneous Syndromes", Akron Children's Hospital, Akron, OH, May 4, 1989

1990 Invited Lecturer, "MRI in Pediatric Neuroimaging-Guidelines", & "Pediatric Spine Imaging", Rhode Island Hospital and Brown University Medical School, April 2, 1990

1990 Invited Lecturer, " Neuroimaging of the Neurocutaneous Syndromes", Radiology Grand Rounds, Rhode Island Hospital and Brown University Medical School, April 2, 1990

1991 Moderator, Pediatric Neuroradiology, Special Scientific Session, American Society of Neuroradiology, 29th Annual Meeting, Washington, D.C.

1991 Moderator and Discussant, Pediatric Neuroradiology Scientific Session, Radiological Society of North America 77th Annual Meeting, Chicago

1992 Invited Lecturer, "Signal Intensity Patterns in MRI of the Pediatric CNS", Radiology Resident Lecture, Ohio State University Health Sciences Center, Columbus, OH, April 8, 1992

1992 Invited Lecturer, "MRI in Pediatric CNS Imaging", Columbus Radiological Society, Columbus, OH, April 8, 1992

1991 Invited Lecturer, "Pediatric Spine Imaging", Radiology Grand Rounds, Columbus Children's Hospital, Columbus, OH, April 9, 1992

1992 Co-Moderator and Discussant, Scientific Session on Pediatric Neuroradiology, Society for Pediatric Radiology 35th Annual Meeting, May 17, Orlando, FL

1992 Invited Lecturer and Panelist, "Sedation in Pediatric Neuroradiology", American Society of Neuroradiology 30th Annual Meeting, June 3, St. Louis, MO

1992 Panelist, Scientific Session on Pediatric Neuroradiology, American Society of Neuroradiology 30th Annual Meeting, June 3, St. Louis, MO

1993 Co-Moderator and Co-Discussant, Neuroradiology Long Papers Session, Society for Pediatric Radiology, 36th Annual Meeting, Seattle, Washington, May 13, 1993

1993 Co-Discussant, Pediatric Scientific Session, American Society of Neuroradiology, 31st Annual Meeting, Vancouver, B.C., Canada, May 19, 1993

1993 Discussant, Pediatric Specialties Scientific Session, American Society of Neuroradiology, 31st Annual Meeting, Vancouver, B.C., Canada, May 19, 1993

1993 Invited Lecturer, "MRI in Pediatric Imaging", Christchurch Hospital, University of Otago, Christchurch, New Zealand, Oct. 4, 1993

1993 Invited Lecturer, "Basics of MRI", & "Signal Intensity Patterns in MRI of the Pediatric CNS", and Discussant, Epilepsy Conference, Royal Children's Hospital, University of Melbourne, Melbourne, Australia, Oct. 11, 1993

1993 Invited Lecturer, "MRI in Pediatric Cerebrovascular Disease", and Discussant, Pediatric Neurology and Neurosurgery Conference, Prince of Wales Hospital, University of Sydney, Sydney, New South Wales, Australia, Oct. 13, 1993

1993 Invited Discussant, Radiology Resident Case Review Lecture, Royal Alexandra Hospital for Children, University of Sydney, Sydney, New South Wales, Australia, Oct. 13, 1993

1993 Invited Lecturer, " Imaging in Pediatric Neuroncology", "Neurocutaneous Syndromes", "Pediatric Neurovascular Diseases", Australasian Society for Paediatric Imaging (ASPI), October 15-17, Leura, New South Wales, Australia.

1993 Invited Lecturer, "Congenital & Developmental Brain Abnormalities", "Intracranial Inflammatory Processes", "Metabolic and Neurodegenerative Disorders", "Vascular Diseases and Trauma", "Cranial and Intracranial Tumors", "Neurocutaneous Syndromes", "Developmental and Acquired Abnormalities of the Spine and Spinal Neuraxis". ASPI MRI Symposium, October 18, Leura, New South Wales, Australia

1994 Invited Lecturer, "Imaging of the Pediatric Central Nervous System: Current Concepts", The Denby Bowdler Lecture, The Annual Post-Graduate Meeting, The Royal Alexandra Hospital for Children, Sydney, New South Wales, Australia, Oct. 21, 1993

1994 Moderator and Invited Lecturer, Update Course in Pediatric Radiology-Neuroradiology, Radiologic Society of North America, November 28, Chicago, IL.

1995 Invited Lecturer, Current Concepts in Pediatric Imaging-Neuroradiology, The Society for Pediatric Radiology, April 27, Colorado Springs, CO.

1995 Invited Lecturer, Society of Magnetic Resonance Technologists, Pediatric MRI-Sedation and Monitoring, 1994 Annual Regional Meeting, October 8, Boston, MA

1995 Moderator and Invited Lecturer, Update Course in Pediatric Radiology-Neuroradiology, Radiological Society of North America, November 27, Chicago, IL

1995 Co-Moderator and Co-Discussant, Pediatric Scientific Session, American Society of Neuroradiology 33rd Annual Meeting, April 23, Chicago, IL

1995 Co-Moderator and Co-Discussant, Neuroradiology Scientific Session, Society for Pediatric Radiology, 38th Annual Meeting, April 29, Washington, D.C.

1995 Invited Lecturer, Emergency Pediatric Radiology Categorical Course- "Increased Intracranial Pressure"-American Roentgen Ray Society 95th Annual Meeting, April 30, Washington, D.C.

1995 Invited Lecturer, Update Course in Clinical Neuroradiology: Pediatric Neurovascular Imaging, Refresher Course, Radiological Society of North America, 81st Annual Meeting, November 29, Chicago, IL

1995 Invited Lecturer, Special Focus Session: Pediatric Sedation. Radiological Society of North America, 81st Annual Meeting, November 30, Chicago, IL

1996 Co-Moderator, and Co-Director, Pediatric Neuroradiology Session, IPR '96 Pediatric Neuroimaging Symposium, International Pediatric Radiology 3rd Conjoint Meeting, SPR, ESPNR, ASPI, May 25, Boston, MA

1996 Invited Lecturer, "Current and New Concepts in Imaging of the Pediatric Spine" IPR 96 Pediatric Neuroimaging Symposium., International Pediatric Radiology 3rd Conjoint Meeting, SPR, ESPNR, ASPI, May 25, Boston, MA

1997 Invited Lecturer, "Imaging of Head and Neck Masses in Childhood", McGill University, Department of Diagnostic Radiology Grand Rounds, January 20, Montreal, Quebec, Canada

1997 Invited Lecturer, "Cranial and Intracranial Tumors of Childhood: An Overview", Montreal Children's Hospital, Department of Diagnostic Imaging, January 21, Montreal, Quebec, Canada

1997 The Dr. Bernadette Nogrady Lecturer, "Imaging of the Neurocutaneous Syndromes in Childhood", Medical Grand Rounds, Montreal Children's Hospital, McGill University, Jan. 21, Montreal, Quebec, Canada.

1997 Invited Lecturer, "Congenital Malformations of the Brain", Practical MRI Categorical Course, American Roentgen Ray Society, 97th Annual Meeting, May 4, Boston, MA.

1997 Invited Lecturer, "MRI and Other Advanced Imaging Techniques", Spinal Dysraphism Workshop, Society for Pediatric Radiology, May 15, St.Louis, MO.

1997 Invited Lecturer, "Advanced Techniques in Pediatric Neuroradiology", New England Conference of Radiologic Technologists and New England Chapter of the American Radiology Nurses Association 39th Annual Fall Symposium, September 26, Sturbridge, MA

1998 Invited Lecturer, "Imaging of the Pediatric Spine, Part I", Department of Radiology, Children's Hospital of Pittsburgh and University of Pittsburgh Medical Center, February 9, Pittsburgh, PA

1998 Invited Lecturer, "Potential Pitfalls in Imaging of the Pediatric CNS", Department of Radiology, Children's Hospital of Pittsburgh and University of Pittsburgh Medical Center, February 9, Pittsburgh, PA

1998 Invited Lecturer, Department of Radiology, Children's Hospital of Pittsburgh and University of Pittsburgh Medical Center, Teaching Session with Residents and Fellows, February 9, Pittsburgh, PA

1998 Invited Lecturer, "Imaging of the Pediatric Spine, Part II", Department of Radiology, Children's Hospital of Pittsburgh and University of Pittsburgh Medical Center, February 10, Pittsburgh, PA

1998 Invited Lecturer, "Imaging of CNS Injury in Child Abuse", Department of Radiology, Children's Hospital of Pittsburgh and University of Pittsburgh Medical Center, February 10, Pittsburgh, PA

1998 Invited Lecturer, Department of Radiology, Children's Hospital of Pittsburgh and University of Pittsburgh Medical Center, Teaching Session with Residents and Fellows, February 10, Pittsburgh, PA

1998 Invited Lecturer, "Potential Pitfalls in Imaging of the Pediatric CNS", Department of Radiology, William Beaumont Hospital, March 18, Royal Oak, MI

1998 Invited Lecturer, "Imaging of CNS Injury in Child Abuse", Department of Radiology, William Beaumont Hospital, March 18, Royal Oak, MI

1998 Course Director and Moderator, Multimodality Imaging of Head & Neck Lesions in Childhood -- The Oral Cavity, Jaw, and Neck; The Eye and Orbit; The Ear and Temporal bone; The Nose, Paranasal Sinuses, and Craniofacial Structures; Sunrise Sessions, The Society for Pediatric Radiology, 41st Annual Meeting, May 7-9, Tucson, AZ

1998 Co-Moderator, Scientific Session VI--Neuroradiology, The Society for Pediatric Radiology, 41st Annual Meeting, May 9, Tucson, AZ

1998 Invited Lecturer, Focus Session: Scoliosis "Imaging the Spine in Scoliosis", the American Society of Neuroradiology, 36th Annual Meeting, May 17-21, Philadelphia, PA

1998 Course Director and Moderator, Minicourse in Pediatric Neuroradiology: Session I: "Pediatric Neurovascular Diseases"; Session II: "Pediatric CNS Tumors"; Session III: "Congenital and Developmental Abnormalities"; Session IV: "Traumatic, Inflammatory, and Neurodegenerative Diseases", Radiological Society of North America, 84th Scientific Assembly and Annual Meeting, November 29-December 1, Chicago, IL

1998 Invited Speaker, Minicourse in Pediatric Neuroradiology, "Tumors about the Third Ventricle", Radiological Society of North America, 84th Scientific Assembly and Annual Meeting, November 30, Chicago, IL

1998 Invited Speaker, Special Focus Session--Child Abuse Revisited, Radiological Society of North America, 84th Scientific Assembly and Annual Meeting, December 1, Chicago, IL

1998 Invited Lecturer, "Potential Pitfalls in Imaging of the Pediatric CNS", The Roger A. Hyman Memorial Lecture, Long Island Radiological Society and Winthrop-University Hospital , Dec. 8, Long Island, NY

1999 Invited Speaker, "Shaken Baby Syndrome", Current Issues in Emergency Practice, Seventh Annual Massachusetts Emergency Nurses Association and Massachusetts College of Emergency Physicians Course, April 13, Marlboro, MA

1999 Invited Speaker, "The Pediatric Radiologist as Expert Witness: How I do it", Society for Pediatric Radiology, Postgraduate Course, May 12, Vancouver, B.C., Canada

1999 Pediatric Focus Sessions Director and Moderator, Session I: "Diagnosis and Management of Head and Neck Vascular Anomalies of Childhood"; Session II: "Diagnosis and Management of Craniofacial Anomalies"; Session III: "Diagnosis and Management of Craniocervical Anomalies"; Session IV: Basic Science/Applications – Watershed Patterns: Anatomy and Pathology; Session V: Diagnosis and Management of Pediatric Neuroendocrine Disorders"; Session VI: "Diagnosis and Management of Pediatric Epilepsy", American Society of Neuroradiology/American Society of Pediatric Neuroradiology Annual Meeting, May 22-23, San Diego, CA

1999 Invited Speaker, Neuroncologic Imaging in Children, Neuroimaging Session, Frontiers of Hope, A Brain Tumor Symposium for Patients, Survivors, Family, Friends, and Professionals, The Brain Tumor Society, November 13, Providence, RI

2000 Invited Speaker, Potential Pitfalls in Pediatric Neuroradiology, Parts I & II, Department of Diagnostic Imaging Grand Rounds, Brown University School of Medicine, Rhode Island Hospital, and the Hasbro Children's Hospital, Providence RI.

2000 Invited Speaker, Neuroradiology of Pediatric Scoliosis, Practical Spine Imaging & Image Guided Therapy Symposium, The American Society of Spine Radiology, February 23, Marco Island, FL

2000 Invited Speaker, Diffusion Imaging in Children, ASNR 2000: Advanced Imaging Symposium, American Society of Neuroradiology, April 2, Atlanta, GA

2000 Moderator, Pediatric Scientific Session, American Society of Pediatric Neuroradiology, American Society of Neuroradiology Annual Meeting, April 2-8, Atlanta, GA

2000 Invited Speaker, Pediatric Neuroradiology, Advanced Medical Malpractice Seminar, Office of Legal Education, Executive Office for U.S. Attorneys, United States Department of Justice, May 2, Columbia, SC.

2000 Invited Speaker, Course Director, Syllabus Editor / Co-author, & Session Moderator, Problem-Focused Strategies in Pediatric Neuroradiology: An Interactive Symposium, Society for Pediatric Radiology and American Society of Pediatric Neuroradiology Joint Post-graduate Course, May 4-6,

Naples, FL.

2000 Invited Speaker and Participant, Fetal & Neonatal Neurologic Injury, Part I - Neuroimaging Patterns and the Timing of Fetal Brain Injury – Medical Intelligence Corporation Keynote Addrress; Part II - The Neuroimaging Expert, Birth Injury and the Law VII, Oct. 19, Las Vegas, NV

2001 Invited Speaker and Participant, Imaging of Fetal & Neonatal CNS Injury Parts I-III, 17th Annual Conference on Obstretics, Gynecology, Perinatal Medicine, Neonatalogy, and the Law, Jan. 2-5, San Juan, PR

2001 Invited Speaker, Pediatric Spine Imaging, Fetal and Infant Neuro-MR, Pediatric Brain Imaging I-II, MR Update 2001, Neuroradiology and Musculoskeletal Imaging Advances, Stanford Radiology, Feb. 16, Las Vegas, Nevada

2001 Invited Speaker and Participant, Sam Hersch Cerebral Palsy Symposium at the Salk Institute, Feb. 27-28, La Jolla, CA.

2001 Invited Speaker & Session Co-coordinator, RSNA Oncodiagnosis Panel-Pediatric Brain Tumors, Radiologic Society of North America 87th Scientific Assembly and Annual Meeting, Chicago, IL, Dec. 28, 2001.

2002 Barnes PD. Invited Speaker. Current and Advanced Techniques in Imaging of the Pediatric Central Nervous System. Department of Neurology Grand Rounds. Stanford University Medical Center, Palo Alto, CA, Jan. 30, 2002.

2002 Invited Speaker. Current and Advanced Techniques in Pediatric Otolaryngology / Head & Neck Imaging – A Problem-focused Approach, Western Society of Pediatric Otolaryngology Annual Meeting, Lucile Packard Children's Hospital at Stanford, Palo Alto, CA, Mar. 16, 2002

2002 Invited Speaker. Neuroimaging of congenital and neonatal Infections. Postgraduate Course: Perinatal and neonatal imaging, Society for Pediatric Radiology, Philadelphia, PA, May 2, 2002.

2002 Session Co-Moderator. White Matter Symposium. American Society of Neuroradiology / American Society of Pediatric Neuroradiology, Vancouver, B.C., May 16, 2002.

2003 Barnes PD. Current and Advanced Imaging of the Fetal and Neonatal CNS. Mid-Coastal California Perinatal Outreach Program, 23rd Annual Meeting, Stanford University Schoolof Medicine, Monterey, CA, Jan. 2003.

2003 Barnes PD. Neuroimaging: a medical perspective. Litigating catastrophically injured infant cases, Association of Trial Lawyers of America, Feb.22, 2003, Atlanta, GA.

2003 Barnes PD. Trauma, including Child Abuse. CT & MRI: State of the Art & Unanswered Questions, SPR Postgraduate Course, San Francisco, CA, May 6, 2003.

2004 Barnes PD. Nonaccidental Head Injury in Children. Neurosciences Grand Rounds. Santa Clara Valley Medical Center. San Jose, CA, Feb. 5, 2004.

2004 Barnes PD. Forensic Science, Evidence-based Medicine, and the "Shaken Baby Syndrome": Radiographic Imaging and Findings. American

Academy of Forensic Sciences Annual Meeting, Dallas,Tx, Feb. 16, 2004.

2004 Barnes PD. Nonaccidental Injury of the Developing Brain: Issues, Controversies, and the Mimics. Moderator and Speaker. Neuroimaging Aspects. Focus Session, American Society of Pediatric Neuroradiology. American Society of Neuroradiology Annual Meeting, Seattle, WA, June 7, 2004.

2004 Barnes PD. Co-Moderator, Pediatric scientific session, American Society of Pediatric Neuroradiology, American Society of Neuroradiology Annual Meeting, Seattle, WA, June 8, 2004.

2004 Barnes PD. Moderator, Pediatric Session and Speaker. MDCT applications in Pediatric Neuroradiology (Brain, Spine, Head & Neck). 6th Annual International Symposium on Multidetector-Row CT. Stanford University Medical Center, San Francisco CA, June 23, 2004.

2004 Barnes PD. Child abuse: the role of neuroimaging in the clinical and forensic evaluation of suspected nonaccidental injury including its mimics. 12th Annual Pediatric Update, Lucille Packard Children's Hospital and Stanford University Medical Center, July 16, 2004.

2005 Barnes PD. Neuroimaging of the pediatric spine – scoliosis. Neuroscience Grand Rounds. Santa Clara Valley Medical Center. San Jose, CA, March 3, 2005.

2005 Barnes PD. Diagnostic imaging of neonatal brain injury. California Association of Neonatologists (CAN) and American Academy of Pediatrics (AAP) District IX Section on Perinatal Pediatrics, 11th Annual Conference, Current Topics and Controversies in Perinatal and Neonatal Medicine, Coronado CA, March 6, 2005.

2005 Barnes PD. Co-moderator, Neuroradiology scientific session, Society for Pediatric Radiology Annual Meeting, New Orleans, LA, May 7, 2005.

2005 Barnes PD. Moderator, CAQ Review Sessions, Pediatric Brain, Head & Neck, and Spine Imaging, American Society of Pediatric Neuroradiology, American Society of Neuroradiology Annual Meeting, Toronto, Ontario, Canada, May 26-27, 2005.

2005 Barnes PD. Co-Moderator, Pediatric scientific session, American Society of Pediatric Neuroradiology, American Society of Neuroradiology Annual Meeting, Toronto, Ontario, Canada, May 26, 2005.

2005 Barnes P.Child abuse: the role of neuroimaging in the clinical and forensic evaluation of suspected nonaccidental injury including its mimics. 13th Annual Pediatric Update, Lucile Packard Children's Hospital and Stanford University Medical Center, July 8, 2005.

2005 Barnes P.Child abuse: the role of neuroimaging in the clinical and forensic evaluation of suspected nonaccidental injury including its mimics. Neurosurgery Grand Rounds, Stanford University Medical Center, July 15, 2005.

2006 Barnes P. Imaging of the Pediatric Central Nervous System and Head & Neck: MRI, CT, US, Nuclear Medicine – Which to do? 14th Annual Pediatric Update, Lucile Packard Children's Hospital and Stanford University Medical Center, July 21, 2006.

2006 Barnes P. Child Abuse: Issues and Controversies in the Era of Evidence-Based Medicine. Pediatric Grand Rounds, Lucile Packard Children's Hospital and Stanford University Medical Center, October 13, 2006.

2006 Hahn J, Barnes P. Prenatal Neurologic Consultations and Management of Brain Malformations. Pediatric Grand Rounds, Lucile Packard Children's Hospital and Stanford University Medical Center, Nov. 3, 2006.

2007. Barnes PD. Co-Director and Co-Moderator. Brain, Head & Neck, and Spine Imaging. Advances in Pediatric CT and MRI. Department of Radiology, Stanford School of Medicine Postgraduate Course. Las Vegas, Nevada, March 17, 2007.

2007 Barnes PD. Lecturer. Advances in Pediatric CT and MRI: Head & Neck Imaging I (Orbit, Sinus, Ear), Head & Neck Imaging II (Face & Neck), Spine Imaging I (Developmental Anomalies), Spine Imaging II (Acquired Conditions), Brain Imaging III (Acute neurologic conditions – Trauma [including child abuse], hemorrhage, vascular disease), Brain Imaging V (Subacute neurologic conditions – Tumors, epilepsy). Department of Radiology, Stanford School of Medicine Postgraduate Course. Las Vegas, Nevada, March 17, 2007. Course Syllabus.

2007 Barnes PD. Lecturer. How I do it – Advanced Neuro-MRI of Nonaccidental CNS injury and its Mimics. Society for Pediatric Radiology 50th Annual Meeting and Postgraduate Course. Miami FL. April 20, 2007.

2007 Barnes P. Lecturer. Child Abuse: Pitfalls in Pediatric Neuroimaging. EBMS Symposium: An Evidence-based Analysis of Infant Brain and Skeletal Injury. Chicago IL, May 10, 2007.

2007 Barnes P. Lecturer. Child Abuse: Issues and Controversies in the Era of Evidence-Based Medicine. Department of Social Services and Child Protection, Lucile Packard Children's Hospital and Stanford University Medical Center, June 21, 2007.

2007 Barnes P. Lecturer. Child Abuse: Issues & Controversies. Pediatrics CME Program. Salinas Valley Memorial Healthcare System, Salinas CA, Nov. 16, 2007.

2008 Barnes P. Lecturer. Child Abuse and the Mimics. Imaging of Brain, Blood, & Bones. Death of a Child Symposium. The Center for American and International Law. Plano TX, March 4, 2008.

2008 Barnes P. Imaging of Child Abuse: Controversies in the Era of Evidence-Based Medicine. Herman Grossman Visiting Lecturer. Radiology & Pediatrics Grand Rounds. Duke University Medical Center, Durham NC, April 10, 2008.

2008 Barnes P. Update on Brain Imaging in Nonaccidental Trauma. Neuroimaging I Session, Pediatric Radiology Series. Radiologic Society of North America, Chicago IL, Nov. 30, 2008.

2008 Barnes P. Co-Moderator & Discussant, Neuroimaging I Scientific Paper Session, Pediatric Radiology Series , Radiologic Society of North America, Chicago, IL Nov. 30, 2008

2008 Barnes P. Neuroimaging in the Evaluation of Pattern and Timing of Fetal and Neonatal Brain Injury. Fetal & Neonatal Annual Care Conference.

Santa Clara Valley Medical Center. San Jose CA, November 7, 2008.

2009 Barnes P. Medical Imaging in Brain Trauma; Intracranial Hemorrhage and Thrombosis (Krasnokutsky M): Imaging & Pitfalls. An Evidence-based Analysis of Infant Brain & Skeletal Trauma. EBMS Symposium, Denver CO, February 22, 2009.

2009 Barnes P. Imaging of Child Abuse and the Mimics: Controversies in the Era of Evidence-Based Medicine. Innocence Network Conference. South Texas College of Law, Houston TX, March 21. 2009.

2009 Barnes P. Neuroimaging in the Evaluation of Pattern and Timing of Fetal and Neonatal Brain Abnormalities. The Latest Tools and Science to Determine the Origin and Timing of Irreversible Brain Damage. Obstetric Malpractice West Coast Conference & Workshop. San Francisco CA, April 28, 2009.

2009 Child Abuse and the Mimics: Controversies in the Era of Evidence-Based Medicine.Visiting Professor, Department of Radiology, Hospital for Sick Children, University of Toronto, Toronto Ontario Canada, Sept. 24, 2009.

2009 Child Abuse, NAI, and the Mimics: Controversies in the Era of Evidence-Based Medicine Seminar. Shaken Baby Death Review Team (Gouge Inquiry). Ministry of the Attorney General, Province of Ontario. Toronto Ontario, Canada, Sept. 24, 2009.

2009 Child Abuse – Nonaccidental Injury (NAI): Controversies in the Era of Evidence-Based Medicine. Controversies in Forensic Science and Medicine: Towards Resolution in the 21st Century. Centre for Forensic Science and Medicine, University of Toronto, Toronto Ontario, Canada, Sept. 25, 2009.

2010 Neuroimaging in the Evaluation of pattern and timing of fetal and neonatal brain abnormalities. The 26th Annual Conference on Obstetrics, Gynecology, Perinatal Medicine, Neonatology, and the Law. Boston University Continuing Medical Education Course, San Jose del Cabo, Mexico, Jan. 2, 2010 (Program and Website material only).

2010 Evidence-based Update: Imaging in Nonaccidental Injury and the Mimics: Blood, Brain, & Bones. National Association of Criminal Defense Attorneys and the Innocence Network (Bureau of Justice Assistance Grant), April 15, 2010, Atlanta GA.

2010 Imaging of the Pediatric Head & Neck (Resident & Fellow Lecture), Department of Radiology, University of Arizona Medical Center, Tucson AZ, June 9, 2010.

2010 Child abuse and the mimics. Update on issues & controversies in the era of evidence-based medicine. Pediatric Grand Rounds. Department of Pediatrics. University of Arizona Medical Center, Tucson AZ, June 9, 2010.

2010 Imaging of fetal and neonatal brain abnormalities. Birth Injury Group. American Association of Justice, Vancouver, BC, Canada, July 11, 2010.

2010 Invited Lecturer & Panelist. Child abuse and the mimics. Update on issues & controversies in the era of evidence-based medicine. National Child Abuse DRC Conference, Las Vegas NV, August 26, 2010.

2010 Expert Testimony (Baumer Case). Child abuse and the mimics. Update on issues & controversies in the era of evidence-based medicine. Michigan Innocence Project. University of Michigan Law School, Detroit, Michigan September 30, 2010.

2010 Child abuse and the mimics. Update on issues & controversies in the era of evidence-based medicine. Neuroscience Grand Rounds, Vancouver General Hospital, BC Children's Hospital, University of British Columbia, Vancouver BC, Canada, October 13, 2010.

2010 Pediatric Head & Neck Imaging I, II; Pediatric Spine Imaging (Resident & Fellow Lecture Series), Department of Radiology, Vancouver General Hospital, BC Children's Hospital, University of British Columbia, Vancouver BC, Canada, October 13-14, 2010.

2010 Imaging of Pediatric CNS Malformations (Neuroradiology and Pediatric Radiology Fellow Lecture), Department of Radiology, Vancouver General Hospital, BC Children's Hospital, University of British Columbia, Vancouver BC, Canada, October 14, 2010.

2011 Neuroimaging in the Evaluation of pattern and timing of fetal and neonatal brain abnormalities (3 lectures). The 27th Annual Conference on Obstetrics, Gynecology, Perinatal Medicine, Neonatology, and the Law. Boston University Continuing Medical Education Course, Maui, Hawaii, January 2-6, 2011.

2011 Imaging of the Pediatric Head & Neck; Imaging of Pediatric CNS Malformations; Imaging of Pediatric CNS Tumors. Radiology Board Review Course. Las Vegas NV, January 12, 2011.

2011 Imaging of child abuse and the mimics. Issues & controversies in the era of evidence-based medicine. California Public Defenders Association Annual Meeting, Monterey, CA, January 21,2011.

2011 Invited Participant & Discussant, Pediatric Abusive Head Trauma. Medical, Forensic, and Scientific Advances and Prevention. Third International Conference. PennState Hershey College of Medicine. San Francisco, CA, July 7-8, 2011.

2011 Imaging of child abuse and the mimics. 2nd Biennial International Conference on Brain Injury in Children, SickKids Centre for Brain & Behavior, The Hospital for Sick Children, July 13, 2011, Toronto, Canada.

2011 Imaging of child abuse and the mimics. Evidence Based Medicine and Social Investigation (EBMSI) Conference, Vancouver, Canada, August 5, 2011.

2011 Child abuse and the mimics: controversies in the era of evidence-based medicine. Cook County Public Defenders' Conference, Oak Brook IL September 8-9, 2011.

2011 Findley K, Barnes P, Moran D, Sperling C. Challenging shaken baby syndrome convictions in the light of new medical and scientific research. *Integris Health* Law & Medicine Lecture Series. Innocence Project. Oklahoma City University School of Law, Oklahoma City, OK, Sep. 21, 2011.

Teaching Awards:

1998	John A. Kirkpatrick Jr. Teaching Award, Pediatric Radiology Fellowship Program, Department of Radiology, Children's Hospital and Harvard Medical School, Boston, MA.
2003	Stanford B. Rossiter Senior Faculty of the Year 2002-2003. Outstanding Contributions to Resident Education, Compassionate Patient Care, and Research, Department of Radiology, Stanford University Medical Center.
2005	Senior Faculty of the Year 2004-2005 Outstanding Contributions to Resident Education, Compassionate Patient Care, and Research, Department of Radiology, Stanford University Medical Center.
2006	Senior Faculty of the Year 2005-2006. Outstanding Contributions to Resident Education, Compassionate Patient Care, and Research, Department of Radiology, Stanford University Medical Center.

Major Curriculum and Educational Programs Developed:

1976-1979	Course Director and Conference Leader, Pediatric House Staff Core Lecture Series, Pediatric Radiology, Oklahoma Children's Memorial Hospital
1976-1980	Conference Co-leader, Monthly Orthopaedic Radiology-Pathology Conference, Oklahoma Teaching Hospitals
1977-1979	Physician Associates Radiology Lecture Series, College of Allied Health, University of Oklahoma
1977-1982	Conference Co-Leader, Weekly Pediatric Cardiology and Cardiac Surgery Conference
1977-1982	Conference Co-Leader - "Sickle Cell Anemia", Annual Clinical Demonstration for First Year Medical Students, College of Medicine, University of Oklahoma.
1977-1982	Pediatric Cardiac Cine-Angiocardiographic case review and consultation weekly with Pediatric, Pediatric Cardiology, Thoracic Surgery Staff, Residents and Fellows
1977-1985	Pediatric Grand Rounds, Oklahoma Children's Memorial Hospital.

1977-1986	Attending Physician and Conference Leader, Daily and Weekly Clinical Teaching Rounds, Children's Memorial Hospital, University of Oklahoma College of Medicine; Pediatric Radiology Film and Fluoroscopy Review with Radiology, Pediatric, Family Medicine Residents and Medical Students.
1977-1986	Pediatric Neuroradiology Case Review and Consultation daily with Neurosurgery, Neurology, Pediatric, and Adolescent Medicine Staff, Residents, Fellows and Medical Students
1977-1986	Pediatric Computed Tomography, Conventional Tomography, and Special Procedures case review and consultation daily with Pediatric, Pediatric Surgery, Adolescent Medicine, and Orthopedic Staff, Residents, Fellows and Medical Students

1977-1986	Elective Tutorials in Pediatric Neuroradiology and Cardiovascular Radiology for Pediatric, Radiology, Neurosurgery, Neurology and Pediatric Surgery Residents, Fellows, and Students
1977-1986	Weekly Diagnostic Radiology Residency Lecture Series, University of Oklahoma College of Medicine
1977-1986	Quarterly Radiologic Technology Inservice in Pediatric Neuroradiology and Cardiovascular Radiology Special Procedures
1977-1986	Co-Leader, Weekly Neurosurgery/Neurology Grand Rounds, Oklahoma Teaching Hospitals and St. Anthony Hospital, Oklahoma City, Oklahoma
1978-1982	Course Lecturer, Annual Department of Radiological Sciences Continuing Medical Education Courses, University of Oklahoma Health Sciences Center
1978-1985	Lecturer, Annual Graduate Physics Seminar, College of Allied Health, University of Oklahoma Health Sciences Center
1979-1981	Lecturer, Annual Radiology Grand Rounds, Oklahoma Teaching Hospitals
1980-1985	Lecturer, Pediatric Surgery Core Lecture Series in Pediatric Radiology, Oklahoma Children's Memorial Hospital
1981-1986	Lecturer, Neurology/Pediatric Neuroradiology Lecture Series, Oklahoma Teaching Hospitals
1982-1985	Participant, Senior Radiology Resident Pre-Board Examinations, University of Oklahoma College of Medicine
1982-1986	Lecturer, Pediatric House Staff Core Lecture Series in Pediatric Radiology, Oklahoma Children's Memorial Hospital
1983-1986	Course Developer and Director, Resident Final Examination in Pediatric Radiology, University of Oklahoma College of Medicine
1985-1986	Oklahoma Diagnostic Imaging Center Lecture Series, Course Co-Developer and Co-Director
1985-1986	Oklahoma Teaching Hospitals Department of Radiological Sciences, Magnetic Resonance Imaging Lecture Series (Course Developer and Director)

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1986	"Magnetic Resonance Imaging for the Referring Physician", Continuing Medical Education Seminar, Program Co-Director, Session Moderator, and Lecturer, Oklahoma Teaching Hospitals and the University of Oklahoma College of Medicine
1986-2000	Daily Neuroradiology Case Review and Consultation with Pediatric and Adolescent Medicine, Neurology, Neurosurgery, Radiology, Oncology, Radiation Therapy, Orthopedic, ORL/Head and Neck Surgery, Ophthalmology, Plastic Surgery, Oral Surgery, and Neuropathology Staff, Fellows, Residents, Medical Students, and visitors, Children's Hospital, Boston, MA

1986-2000 Weekly Pediatric Neurology-Neuroradiology Rounds with Staff, Fellows, Residents, Medical Students, and visitors, Conference Co-Leader, Children's Hospital, Boston, MA

1986-2000 Weekly Pediatric Neurosurgery-Neuroradiology Rounds with Staff, Fellows, Residents, Medical Students, and visitors, Conference Co-Leader, Children's Hospital, Boston, MA

1986-2000 Weekly Pediatric Neuroncology-Neuroradiology Rounds with Pediatric Oncology, Radiation Oncology, and Neurosurgery Staff, Fellows, Residents, Medical Students, and visitors (The Children's Hospital and Dana-Farber Cancer Institute), Conference Co-Leader, Children's Hospital, Boston, MA

1986-2000 Weekly Longwood Medical Area Neuroradiology Conference with Staff, Fellows, Residents, Medical Students, and visitors (The Children's Hospital, Brigham & Women's Hospital, Beth Israel Hospital, New England Deaconess Hospital, Dana-Farber Cancer Institute), Conference Co-Leader, Children's Hospital, Boston, MA

1986-2000 Monthly Pediatric ORL/Head & Neck Radiology Rounds with Staff, Fellows, Residents, Medical Students, and visitors, Conference Co-Leader, Children's Hospital, Boston, MA

1986-2000 Monthly Pediatric Radiology Difficult Case Conference (Risk Management and Quality Improvement) with Staff, Fellows, Residents, Medical Students, and visitors, Children's Hospital, Boston, MA

1986-2000 Monthly Boston Area Neuroradiology Club Case Conference with Staff, Fellows, Residents, Medical Students, and visitors (Massachusetts General Hospital)

1986-2000 Pediatric Neuroradiology Annual Lecture Series, Course Co-Director and Lecturer, for Staff, Fellows, Residents, Medical Students, and visitors.

1986-2000 Pediatric Neuroradiology Introductory Lectures for Harvard Medical Students and Rotating Radiology Residents, Radiology, Children's Hospital, Boston, MA

1986-1988 Cardiac Radiology Lecture Series, Course Developer and Lecturer, Radiology, Children's Hospital, Boston, MA

1986-1990 Magnetic Resonance Imaging Lecture Series, Course Developer, Director, and Lecturer, Radiology, Children's Hospital, Boston, MA

2000 Basic Technical and Biological Principles of Magnetic Resonance Imaging Lecture Series, Department of Radiology, Beth Israel Deaconess Medical Center, Boston, MA

2000 Pediatric Neuroradiology Resident Pre-Board Review, Department of Radiology, Beth Israel Deaconess Medical Center, Boston, MA

2000- Pediatric Neuroradiology Lectures, Neuroradiology Lecture Series, Stanford University Medical Center, Palo Alto, CA.

2000- Annual Pediatric Neuroradiology Lecture Series for Neurology Residents & Fellows, Stanford University Medical Center, Palo Alto, CA.

2001- Annual Pediatric Neuroradiology Lecture Series for Neurosurgery

Residents and Fellows Stanford University Medical Center, Palo Alto, CA.

2001- Pediatic Head & Neck Imaging Lecture Series for ORL/Head & Neck Residents and Fellows, Stanford University Medical Center, Palo Alto, CA.

2001- Pediatric Neuroradiology Lectures, Pediatric Radiology Lecture Series, Department of Radiology, Stanford University Medical Center, Palo Alto, CA.

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Original Articles:

1. Gilsanz V, Strand R, Barnes P, Nealis J. Results of presumed cryptogenic epilepsy in childhood by CT scanning. Annals of Radiology 1979;22:184-187.
2. Carson J, Tunell W, Barnes P, Altshuler G. Hepatoportal sclerosis in childhood. Journal of Pediatric Surgery 1981;16:291-296.
3. Leonard J, Barnes P, Keyes J, Huff D, Strange D, Vanhoutte J, Galloway D. Digital radiography: utilization of a nuclear medicine computer system. Computerized Radiology 1983;7:85-90.
4. Barnes P, Reynolds A, Galloway D, Pollay M, Leonard J, Prince J. Digital myelography of spinal dysraphism in infancy. American Journal of Neuroradiology 1984; 5:208-211; American Journal of Roentgenology 1984; 142:1247-1252.
5. Carson J, Barnes P, Tunell W, Smith E, Jolley S. Imperforate anus, the neurologic implication of sacral abnormalities. Journal of Pediatric Surgery 1984; 19:838-842.
6. Barnes P, Lester P, Yamanashi W, Woosley R, Wheatley K. Magnetic resonance imaging in childhood intracranial masses. Magn Reson Imaging 1986;4:41-49.
7. Barnes P, Lester P, Yamanashi W, Prince J. Magnetic resonance imaging in infants and children with spinal dysraphism. American Journal of Neuroradiology 1986; 7:465-472; American Journal of Roentgenology 1986; 147:339-346.
8. Tunell W, Barnes P, Austin J, Reynolds A. Neuroradiologic evaluation of sacral abnormalities in imperforate anus complex. Journal of Pediatric Surgery 1986;22:58-61.
9. Hamza M, Noorani P, Bodensteiner J, Barnes P. Benign extracerebral fluid collections: a cause of macrocrania in infancy. Pediatric Neurology 1987; 3:218-221.
10. Noorani P, Bodensteiner J, Barnes P. Colpocephaly: frequency and associated findings. Journal of Child Neurology 1988;3:100-104.
11. Bartynski W, Barnes P, Wallman J. Cranial CT of autosomal recessive osteopetrosis. American Journal of Neuroradiology 1989;10:543-550.
12. Schick R, Jolesz F, Macklis J, Barnes P. Magnetic resonance diagnosis of dural venous sinus thrombosis complicating L-asparaginase therapy. J Computerized Medical Imaging and Graphics 1989;13:319-327.

13. Pierce S, Barnes P, Loeffler J, McGinn C, Tarbell N. Definitive radiation therapy in the management of symptomatic patients with optic glioma: survival and long-term effects. Cancer 1990;69:45-52.
14. Healey EA, Barnes PD, Kupsky WJ, Scott RM, Sallan SE, Black PM, Tarbell NJ. The prognostic significance of postoperative residual tumor in ependymoma. Neurosurgery 1991;28:666-671.

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15. Mattle HP, Wentz KU, Edelman RR, Wallner B, Finn JP, Barnes PD, Atkinson DJ, Kleefield J, Hoogewoud HM. Cerebral venography with MR. Radiology 1991;178:453-458.
16. O'Tuama LA, Janicek MJ, Barnes PD, Scott RM, Black PM, Sallen SE, Tarbell NJ, Kupsky WJ, Wagenaar D, Ulanski JS, Davis R, Treves S. Functional imaging of treated childhood brain tumors: SPECT imaging with 201-T1 and 99m-Tc-HMPAO. Pediatr Neurology 1991;7:249-257.
17. Meyer JS, Hoffer FA, Barnes PD, Mulliken JB. Biological classification of soft tissue vascular anomalies, MRI correlation. AJR 1991;157:559-564.
18. Scott RM, Barnes P, Kupsky W, Adelman L. Cavernous angiomas of the central nervous system in children. J Neurosurg 1992;76:38-46.
19. Jones KM, Mulkern RV, Mantello MT, Melki PS, Ahn SS, Barnes PD, Jolesz FA. Brain hemorrhage: evaluation with fast spin-echo and conventional dual spin-echo images. Radiology 1992;182:53-58.
20. Ahn SS, Mantello MT, Jones KM, Mulkern RV, Melki PS, Higuchi N, Barnes PD. Rapid MR imaging of the pediatric brain using a fast spin-echo (FSE) technique. AJNR 1992;13:1169-1177.
21. Warf BC, Scott RM, Barnes PD, Hendren WH. Tethered spinal cord in patients with anorectal and urogenital malformations. Pediatr Neurosurg 1993;19(1):25-30.
22. Schutzman SA, Barnes PD, Mantello MT, Scott RM. Epidural hematomas in children. Annals of Emergency Medicine 1993;22:535-541.
23. Barnes PD, Brody JD, Jaramillo D, Akbar JU, Emans JB. Atypical idiopathic scoliosis: MRI evaluation. Radiology 1993;186:247-253.
24. Tice H, Barnes P, Goumnerova L, Scott RM, Tarbell NJ. Pediatric and adolescent oligodendrogliomas. AJNR 1993;14:1293-1300.
25. Zerbini C, Gelber R, Weinberg D, Sallan S, Barnes P, Kupsky W, Scott RM, Tarbell N. Prognostic factors in medulloblastoma including DNA ploidy. J Clin Oncol 1993;11(4):616-622.
26. Dunbar SF, Barnes PD, Tarbell NJ. Radiologic determination of the caudal border of the spinal field in craniospinal irradiation. Int J Radiation Oncology Biol Phys 1993;26:669-673.
27. Kretschmar CS, Tarbell NJ, Barnes PD, Krischer JP, Burger PC, Kun L. Pre-irradiation chemotherapy and hyperfractionated radiation therapy 66 Gy for children with brain stem tumors. Cancer 1993;72(4):1404-1413.

28. Tice H, Jones K, Mulkern R, Schwartz R, Kalina P, Ahn S, Barnes P, Jolesz F. Evaluation of intracranial neoplasia with fast spin-echo and conventional dual spin-echo images. JCAT 1993;17:425-431.
29. Hetelekidis S, Barnes P, Tao M, Fischer E, Schneider L, Scott RM, Tarbell N. 20-year experience in childhood craniopharyngioma. Int J Radiation Oncology Biol Phys 1993;27:189-195.

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30. O'Tuama LA, Treves ST, Larar JN, Packard AB, Kwan AJ, Barnes PD, Scott RM, Black PM, Madsen JR, Goumnerova LC, Sallan SE, Tarbell NJ. Thallium-201 versus technetium-MIBI SPECT in evaluation of childhood brain tumors: a within-subject comparison. J Nucl Med 1993;34:1045-1051.
31. Strand RD, Barnes PD, Young Poussaint T, Estroff JA, Burrows PE. Cystic retrocerebellar malformations: unification of the Dandy-Walker complex and Blake's pouch cyst. Pediatr Radiol 1993;23:258-260.
32. Vera M, Fleisher GR, Barnes PD, Bjornson BH, Allred EN, Goldmann DA. Computed tomography imaging in children with head trauma: utilization and appropriateness from a quality improvement perspective. Infect Control Hosp Epidemiol 1993;14:491-499.
33. Kooy HM, van Herk M, Barnes PD, Alexander E, Dunbar SF, Tarbell NJ, Mulkern RV, Holupka E, Loeffler JS. Image fusion for stereotactic radiotherapy and radiosurgery treatment planning. Int J Radiation Oncology Biol Phys 1994;28:1229-1234.
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36. Dunbar SF, Tarbell NJ, Kooy HM, Alexander E, Black PM, Barnes PD, Goumnerova L, et al. Stereotactic radiotherapy for pediatric and adult brain tumors: preliminary report. Int J Radiation Oncology Biol Phys 1994;30:531-539.
37. Scott RM, Hetelekidis S, Barnes PD, Goumnerova L, Tarbell NJ. Surgery, radiation, and combination therapy in the treatment of childhood craniopharyngioma -- a 20 year experience. Pediatr Neurosurg 1994;21:75-81.
38. Tarbell NJ, Barnes P, Scott RM, Goumnerova L, Pomeroy SL, Black P McL, Sallan SE, Billett A, LaValley B, Helmus A, Kooy HM, Loeffler JS. Advances in radiation therapy for craniopharyngiomas. Pediatr Neurosurg 1994;21:101-107.

39. Klufas RA, Hsu L, Barnes PD, Patel MR, Schwartz RB. Dissection of the carotid and vertebral arteries: imaging with MR angiography. AJR 1995;164:673-677.
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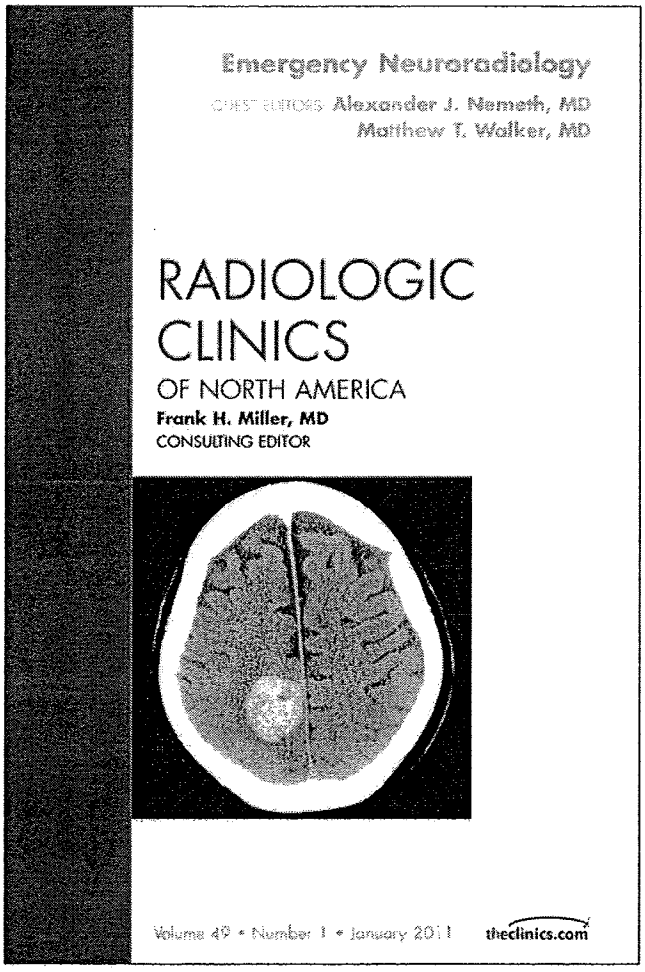

Imaging of Nonaccidental Injury and the Mimics: Issues and Controversies in the Era of Evidence-Based Medicine

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KEYWORDS

• Evidence-based medicine • Nonaccidental injury
• Nonaccidental trauma • Nonaccidental head injury
• Child abuse

Nonaccidental injury (NAI) is reportedly the most frequent cause of traumatic injury in infants (peak incidence age 6 months; 80% of traumatic brain injury deaths under the age of 2 years).[1–4] NAI, nonaccidental trauma (NAT), and nonaccidental head injury are more recently used terms instead of the traditional labels, child abuse, battered child syndrome, and shaken baby syndrome (SBS). The traditional definition of NAI/SBS is intentional or inflicted physical injury to infants characterized by the triad of (1) subdural hemorrhage (SDH), (2) retinal hemorrhage (RH), and (3) encephalopathy (ie, diffuse axonal injury [DAI]) occurring in the context of inappropriate or inconsistent history (particularly when unwitnessed) and commonly accompanied by other apparently inflicted injuries (eg, skeletal).[1–4] This empirical formula is under challenge by evidence-based medical and legal principals.[4–14]

TRAUMATIC BRAIN INJURY

Traumatic brain injury has been categorized in several ways.[1,4] Primary injury directly results from the initial traumatic force and is immediate and irreversible (eg, contusion or shear injury). Secondary injury arises from or is associated with the primary injury and is potentially reversible (eg, swelling, hypoxia-ischemia, seizures, or herniation). Traditional biomechanics describes impact loading as linear forces that produce localized cranial deformation and focal injury (eg, fracture, contusion, or epidural hematoma). Accidental injury (AI) is considered typically associated with impact and, with the exception of epidural hematoma, is usually not life threatening. Impulsive loading refers to angular acceleration/deceleration forces resulting from sudden nonimpact motion of the head on the neck (ie, whiplash) and produces diffuse injury with tissue disruption (eg, bridging vein rupture with SDH and white matter shear with DAI). Young infants are thought particularly vulnerable to the latter mechanism (ie, SBS) because of weak neck muscles, a relatively large head, and an immature brain. SBS is traditionally postulated to result in the triad of primary traumatic injury (ie, SDH, RH, and DAI), which has been reportedly associated with the most severe and fatal CNS injuries. Stated assault mechanisms

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in NAI include battering, shaking, impact, shaking-impact, strangulation, suffocation, and combined assaults (shake-bang-choke).[1–4] Although the spectrum of injury in NAI overlaps that of AI, certain patterns have been previously reported as characteristic of or highly suspicious for NAI.[1–4] These include multiple or complex cranial fractures (**Fig. 1**), acute interhemispheric SDH (**Fig. 2**), acute-hyperacute SDH (**Fig. 3**), DAI, chronic SDH, and the combination of chronic and acute SDH (**Fig. 4**). The latter combination is thought indicative of more than one abusive event. Imaging evidence of brain injury may occur with or without other clinical findings of trauma (eg, bruising) or other traditionally higher-specificity imaging findings of abuse (eg, classic metaphyseal lesions or rib fractures) (**Fig. 5**).[1–4] Therefore, clinical and imaging findings of injury out of proportion to the history of trauma and injuries of different ages have been the basis of making a medical diagnosis and offer expert testimony that such "forensic" findings are "proof" of NAI/SBS, particularly when encountered in premobile, young infants.

EVIDENCE-BASED MEDICINE

Evidence-based medicine (EBM) is now the guiding principle as medicine moves from an authoritarian to an authoritative era to overcome bias and ideology.[4,15–20] EBM quality-of-evidence ratings of the literature (eg, classes I–IV) are based on levels of accepted scientific methodology and biostatistical significance (eg, *P* values) and apply to the formulation of standards and guidelines for every aspect of medicine, including diagnostics, therapeutics, and forensics. EBM analysis reveals that few published reports in the traditional NAI/SBS literature merit a quality-of-evidence rating above class IV (eg, expert opinion alone).[5] Such low ratings do not meet EBM recommendations for standards (eg, level A) or for guidelines (eg, level B). Difficulties exist in the rational formulation of a medical diagnosis or forensic determination of NAI/SBS based on an alleged event (eg, shaking) that is inferred from clinical, imaging, or pathology findings in the subjective context of (1) an unwitnessed event, (2) a noncredible history, or (3) an admission or confession under dubious circumstances.[6] This problem is further confounded by the lack of consistent and reliable criteria for the diagnosis of NAI/SBS and because much of the traditional literature on child abuse consists of anecdotal case series, case reports, reviews, opinions, and position papers.[5,6,10,11,21,22] Many reports include cases having impact injury, which

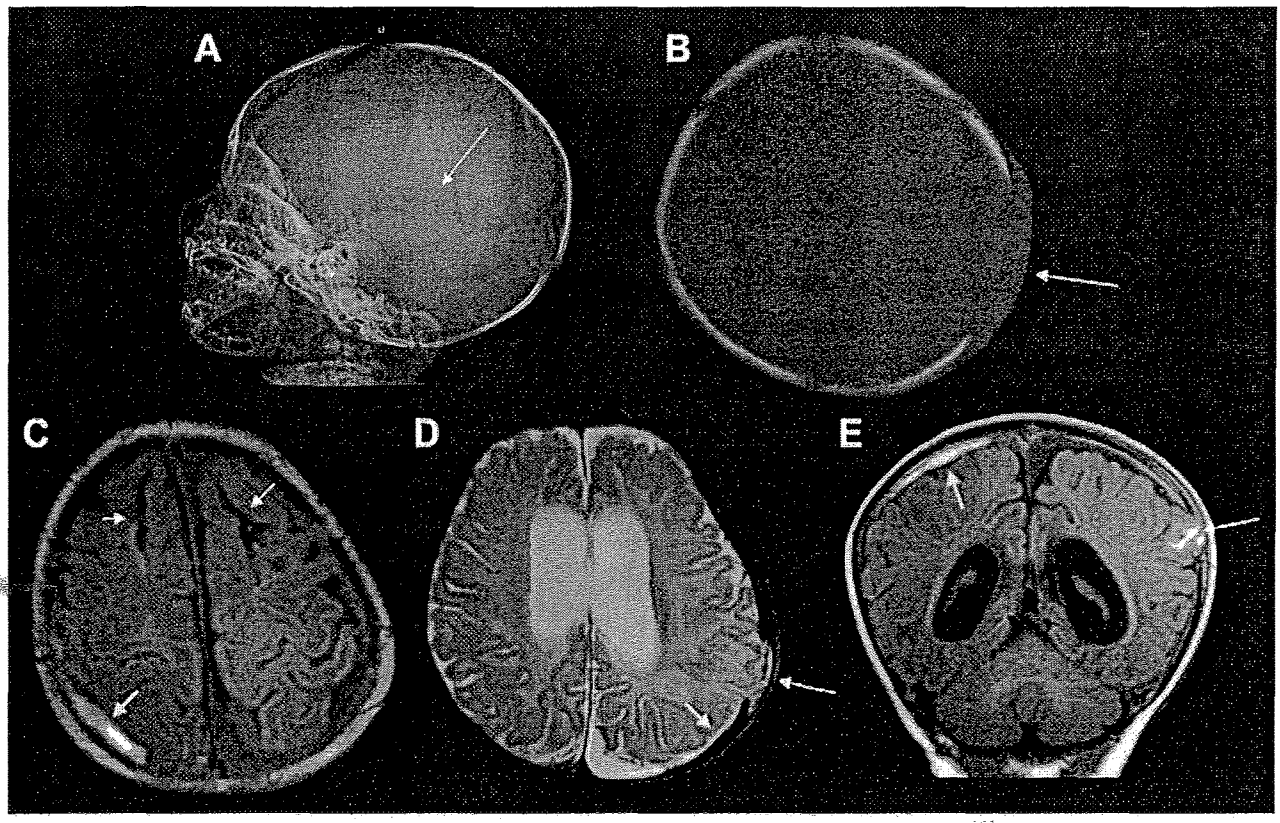

Fig. 1. Nine-week-old infant with triad and alleged NAI; also, history of traumatic labor and delivery. Skull film (*A*), CT (*B*) plus FLAIR (*C*), T2 (*D*), and T1 (*E*) MR imaging shows bilateral skull fractures with left growing fracture (*long white arrows*), chronic bifrontal cerebral white matter clefts (*short white arrows*) (*C*) plus acute, subacute, and chronic SDHs/rehemorrhages (*yellow arrows*).

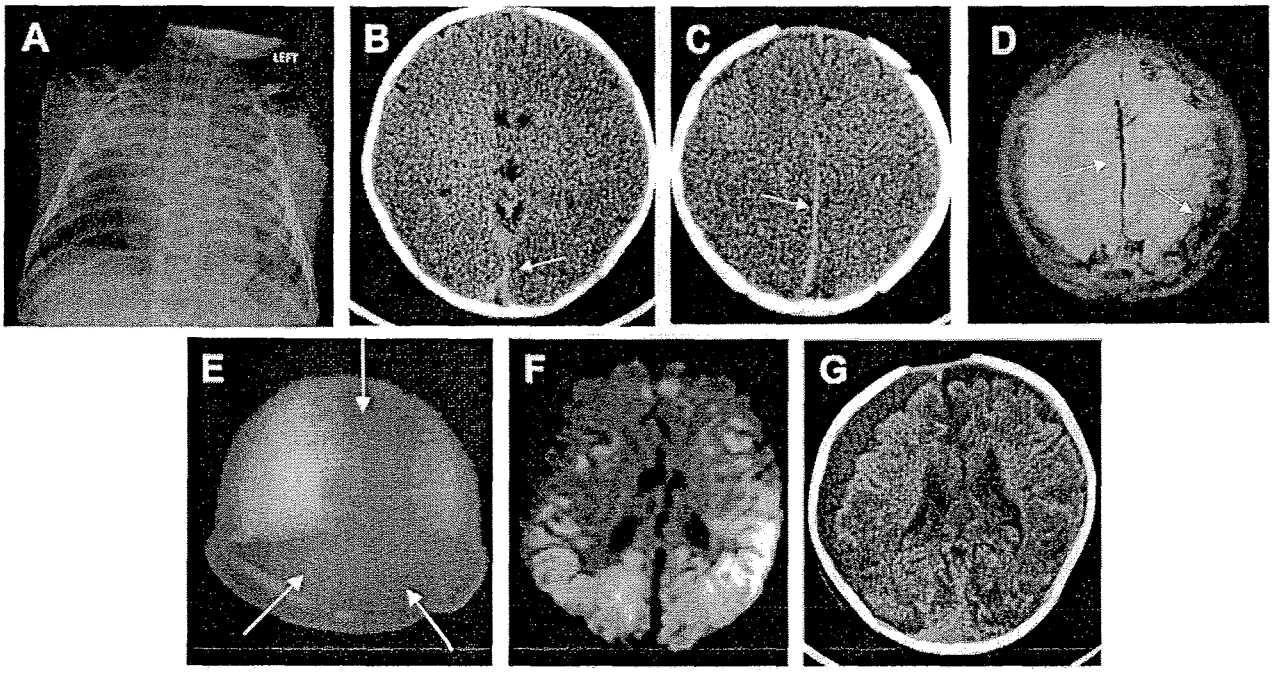

Fig. 2. Five-week-old infant with triad and alleged NAI; also, cold symptoms, vitamin D undersupplemented, acute choking episode during feeding, and status epilepticus. Chest film (*A*) shows bilateral lung opacities. CT (*B, C*) plus T2* MR imaging (*D*) shows bilateral cerebral edema with bilateral thin, acute-subacute hemorrhages (or thromboses) about the falx, tentorium, and convexities (*arrows*). Vertex CT (*E*) shows suture diastasis versus pseudodiastasis (*arrows*) (craniotabes?). DWI (*F*) shows global hypoxic-ischemic injury. Later CT (*G*) shows atrophy and chronic SDH.

undermines the SBS hypothesis by imposing a shaking-impact syndrome. Also, the inclusion criteria provided in many reports are criticized as arbitrary. Examples include suspected abuse, presumed abuse, likely abuse, and indeterminate.[21,22] Furthermore, the diagnostic criteria often seem to follow circular logic, such that the inclusion criteria (eg, the triad equals SBS/NAI) becomes the conclusion (ie, SBS/NAI equals the triad).

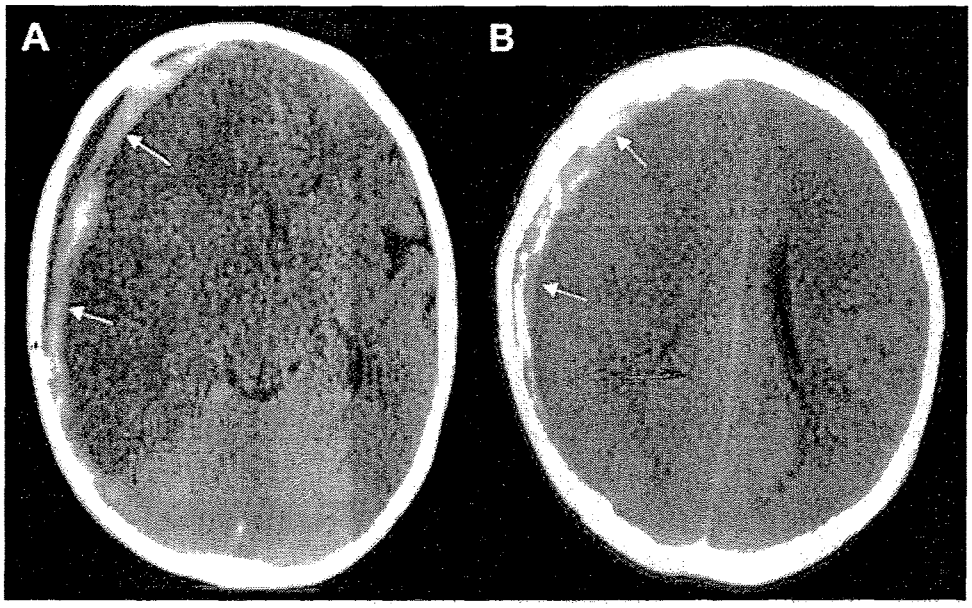

Fig. 3. Eight-month-old infant with triad and alleged NAI; also, right occipital skull fracture (age indeterminate; not shown) and 4- to 6-week-old wrist fracture. Hyperacute right SDH versus chronic SDH with rehemorrhage? CT (*A, B*) shows mixed high- plus low-density right extracerebral collection (*arrows*) with right cerebral edema, mass effect, and left shift. Question of subdural membrane on autopsy.

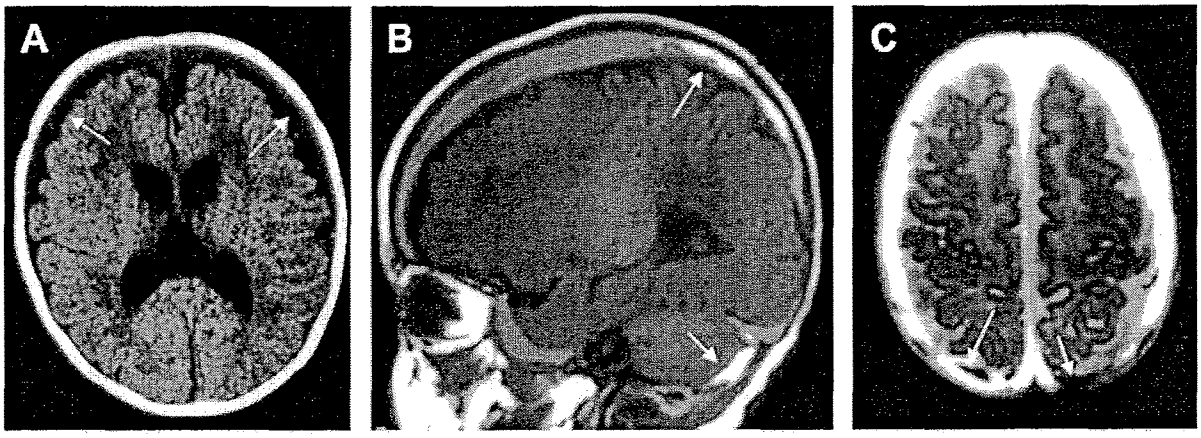

Fig. 4. Six-month-old infant with macrocephaly, the triad, and alleged NAI: BECC versus chronic SDH with rehemorrhage versus acute SDHG plus SDH? CT (*A*) shows bilateral frontal isohypodense extracerebral collections (*arrows*) with minute high densities (not shown). T1 MR imaging (*B*) shows smaller extracerebral high intensities (*arrows*) superimposed on larger isohypointensities. T2 MR imaging (*C*) shows small extracerebral T2 hypointensities (*arrows*) superimposed on large isohyperintensities.

RULES OF EVIDENCE AND EXPERT TESTIMONY

Regarding rules of evidence within the justice system, there are legal standards for the admissibility of expert testimony.[7,8,11,23] The Frye standard requires only that the testimony be generally accepted in the relevant scientific community. The Daubert standard requires assessment of the scientific reliability of the testimony. A criticism of the justice system is that the application of these standards varies with the jurisdiction (eg, according to state versus federal law). Additional legal standards regarding proof are also applied in order for the triar of fact (eg, judge or jury) to make the determination of civil liability or criminal guilt. In a civil action (eg, medical malpractice lawsuit), money is primarily at risk for the defendant health care provider, and proof of liability is based on a preponderance of the evidence (ie, at least 51% scientific or medical probability or certainty). In a criminal action, life or liberty is at stake for the defendant, including the permanent loss of child custody.[7,8,11,23,24] In such cases, the defendant has the constitutional protection of due process that requires a higher level of proof. This includes the principles of innocent until proved guilty beyond a reasonable doubt with the burden of proof on the prosecution and based on clear and convincing evidence. No percentage of level of certainty is provided, however, for these standards of proof in most jurisdictions. Furthermore, only a preponderance of the medical evidence (ie, minimum of 51% certainty) is required to support proof of guilt whether or not the medical expert testimony

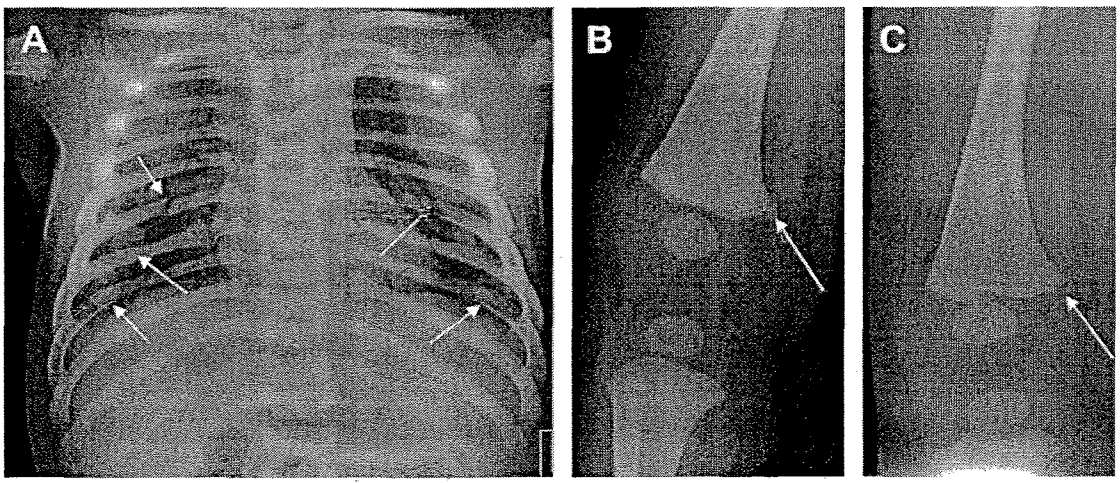

Fig. 5. Three-month-old infant with alleged NAI; also, history consistent with congenital rickets. Chest film (*A*) shows bilateral recent and old, healing rib fractures (pseudofractures? rachitic rosary? [*arrows*]). Knee films before (*B*) and after (*C*) vitamin D supplementation show healing classic metaphyseal lesions (*arrows*)?

complies with the Frye standard (ie, general acceptance requirement) or the Daubert standard (ie, scientific reliability requirement). Further criticism of the criminal justice process is that in NAI cases, medical experts have defined SBS/NAI as "the presence of injury (eg, the triad) without a sufficient historical explanation" and that this definition unduly shifts the burden to the defendant to establish innocence by proving the expert theory wrong.

THE MEDICAL PROSECUTION OF NAI AND ITS EBM CHALLENGES

Traditionally, the prosecution of NAI has been based on the presence of one or more aspects of the triad as supported by the premises that (1) shaking alone in an otherwise healthy child can cause SDH leading to death, (2) such injury can never occur on an accidental basis (eg, short-distance fall) because it requires a massive violent force equivalent to a motor vehicle accident or a fall from a multistory building, (3) such injury is immediately symptomatic and cannot be followed by a lucid interval, and (4) changing symptoms in a child with prior head injury indicates newly inflicted injury and not a spontaneous rebleed.[1–4,7,8,11] Using this reasoning, the last caretaker is automatically guilty of inflicted injury, especially if not witnessed by an independent observer. Also, it has been asserted that RHs of a particular pattern are diagnostic of SBS/NAI.

Reports from clinical, biomechanical, pathology, forensic, and legal disciplines, within and outside of the child maltreatment literature, have challenged the evidence base for NAI/SBS as the only cause for the triad.[5–12] Such reports indicate that the triad may also be seen with AI (including witnessed short-distance falls, lucid intervals, and rehemorrhage) (**Figs. 6** and **7**) as well as in medical conditions. These are the mimics of NAI and often present as acute life-threatening events (ALTEs).[25,26] The medical mimics include hypoxia-ischemia (eg, apnea, choking, or respiratory or cardiac arrest) (see **Figs. 2, 6,** and **7**), ischemic injury (eg, arterial versus venous occlusive disease) (**Fig. 8**), vascular anomalies (eg, arteriovenous malformation [AVM]) (**Fig. 9**), seizures (see **Fig. 2**), infectious or postinfectious conditions (**Fig. 10**), coagulopathies (**Fig. 11**), fluid-electrolyte derangement, and metabolic or connective tissue disorders, including vitamin deficiencies and depletions (eg, C, D, or K) (see **Figs. 1** and **5**; **Fig. 12**).[2,4]

Many ALTEs seem multifactorial and involve a combination, sequence, or cascade of predisposing and complicating events or conditions.[4,25] As an example, an infant may suffer a head impact, or choking spell, followed by seizures or apnea, and then undergo a series of interventions, including prolonged or difficult resuscitation and problematic airway management with subsequent hypoxia-ischemia and coagulopathy (see **Figs. 2, 6, 7,** and **11**). Another example is a young infant with a predisposing condition, such as infectious illness, fluid-electrolyte imbalance, metabolic disorder, or a coagulopathy, who then suffers seizures, respiratory arrest, and resuscitation with hypoxia-ischemia (see **Figs. 10–12; Fig. 13**). In many cases of alleged SBS/NAI, it is often assumed that nonspecific premorbid symptoms (eg, irritability, lethargy, and poor feeding) in an otherwise healthy infant are indicators of ongoing abuse or that such symptoms become the inciting factor for the abuse. A thorough and complete medical investigation in such cases may reveal that the child is not otherwise healthy and is suffering from a medical condition that progresses to an ALTE.[2,4,25]

BIOMECHANICAL CHALLENGES

The mechanical basis for SBS as hypothesized by Guthkelch, Caffey, and other investigators,[27] was originally extrapolated from Ommaya,[28] who used an animal whiplash model to determine the angular acceleration threshold (ie, 40 g) for head injury (ie, concussion, SDH, and shear injury). It was assumed that manual shaking of an infant could generate these same forces and produce the triad. Duhaime and colleagues[29] measured the angular accelerations associated with adult manual shaking (ie, 11 g) and impact (ie, 52 g) in a 1-month-old infant anthropormorphic test device (ATD). Only accelerations associated with impact (4 to 5 times that associated with shakes) on an unpadded or padded surface exceeded the injury thresholds determined by Ommaya. In the same study, the Duhaime and colleagues reported a series of 13 fatal cases of NAI/SBS in which all had evidence of blunt head impact (more than half noted only at autopsy).[29] The investigators concluded that CNS injury in SBS/NAI in its most severe form is usually not caused by shaking alone. Their results contradicted many of the original reports that had relied on the whiplash mechanism as causative of the triad. They suggested the use of the new term, shaken-impact syndrome. More recently, Prange and colleagues,[30] using a 1.5 month-old ATD, showed that inflicted impacts against hard surfaces were more likely associated with brain injury than falls from less than 1.5 m or from vigorous shaking. With further improvements in ATDs, more recent experiments indicate that maximum head

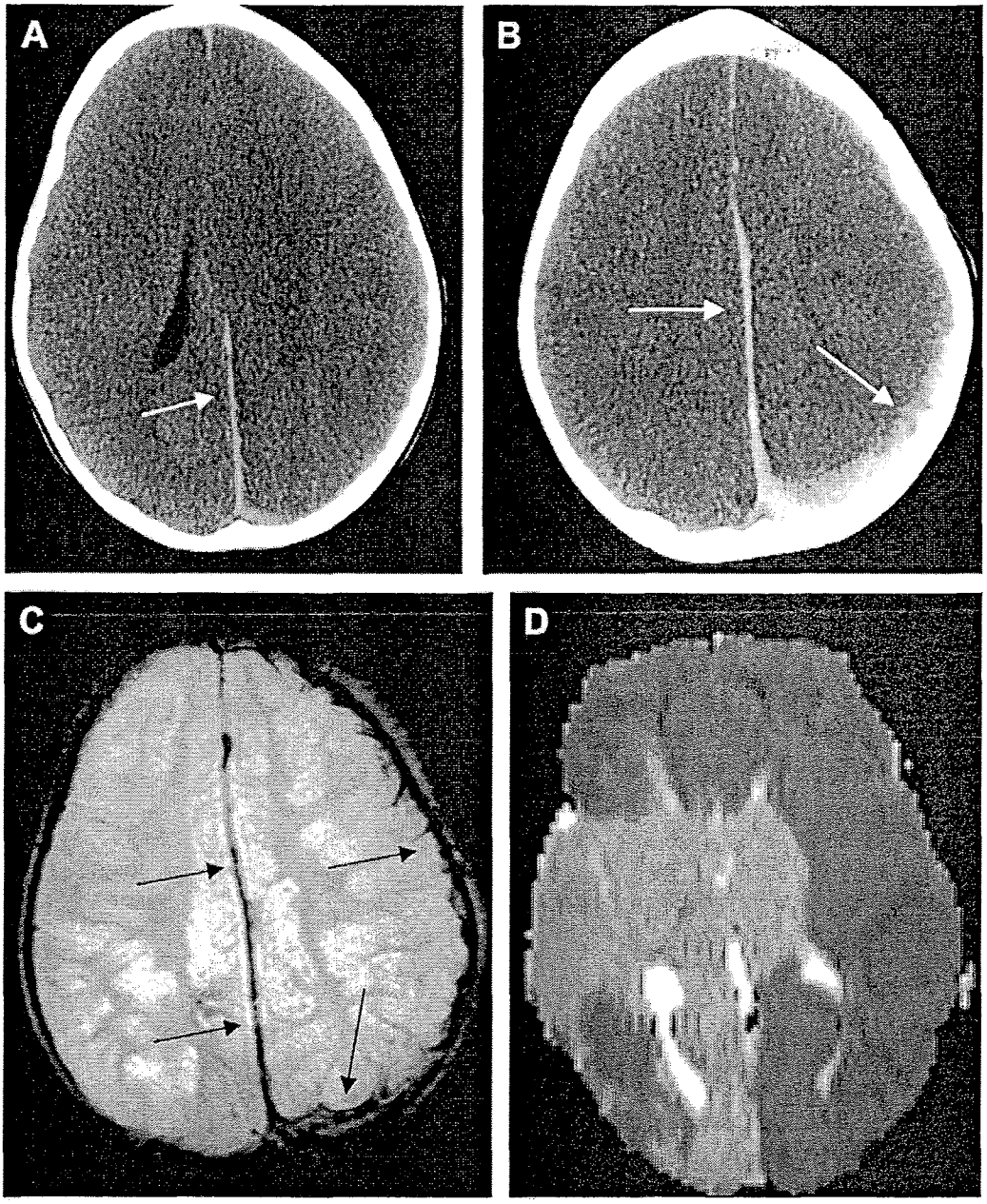

Fig. 6. Twenty-one–month–old toddler with triad and alleged NAI; also, history of prior head impact. Question prior injury with lucid interval versus hyperacute injury. CT (*A, B*) acute left convexity and interhemispheric SDH and SAH (*arrows*) with cerebral swelling, left more than right. T2* MR imaging (*C*) shows low intensity SDH (*arrows*) with T1/T2 isointensity (not shown). ADC map (*D*) shows asymmetric cerebral restricted diffusion (left > right). Autopsy confirms impact with acute SDH, SAH, and hypoxic-ischemic injury.

accelerations may exceed injury reference values at lower fall heights than previously determined (**Fig. 14**).[31] Critics of the Duhaime and Prange studies contend that there is no adequate human infant surrogate yet designed to properly test shaking versus impact.[32] Other reports also show that shaking alone cannot result in brain injury (ie, the triad) unless there is concomitant injury to the neck, cervical spinal column, or cervical spinal cord, because these are the weak links between the head and body of the infant.[33–35] Spinal cord injury without radiographic abnormality (SCIWORA), whether or not AI or NAI, is an important example of primary neck and spinal cord injury with secondary brain injury (see **Fig. 7**).[35] For example, a falling infant experiences a head-first impact with subsequent neck hyperextension (or hyperflexion) from the force of the trailing body mass. There is resultant upper spinal cord injury without detectable spinal column injury on plain films or CT. Compromise of the respiratory center at the cervicomedullary junction results in hypoxic brain injury, including the thin SDH (see **Fig. 7**). CT often shows the brain injury, but only MR imaging may show the additional neck or spinal cord injury.

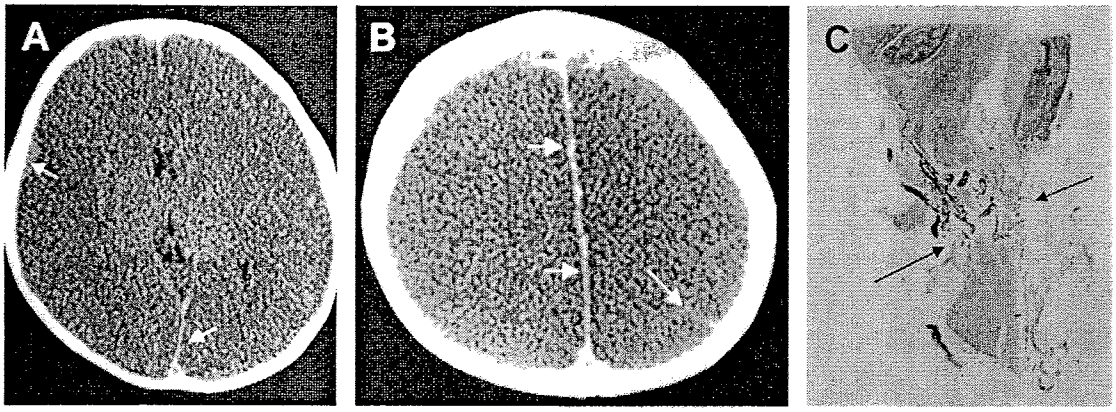

Fig. 7. Twenty-one–month–old with triad and alleged NAI; also, history of 4-ft fall. CT (*A, B*) with high-density SAH and thin SDH (*arrows*) plus cerebral edema. Sagittal plane photomicrograph (*C*) from autopsy shows upper cervical spinal cord disruption (*arrows*) resulting in global hypoxic-ischemic injury.

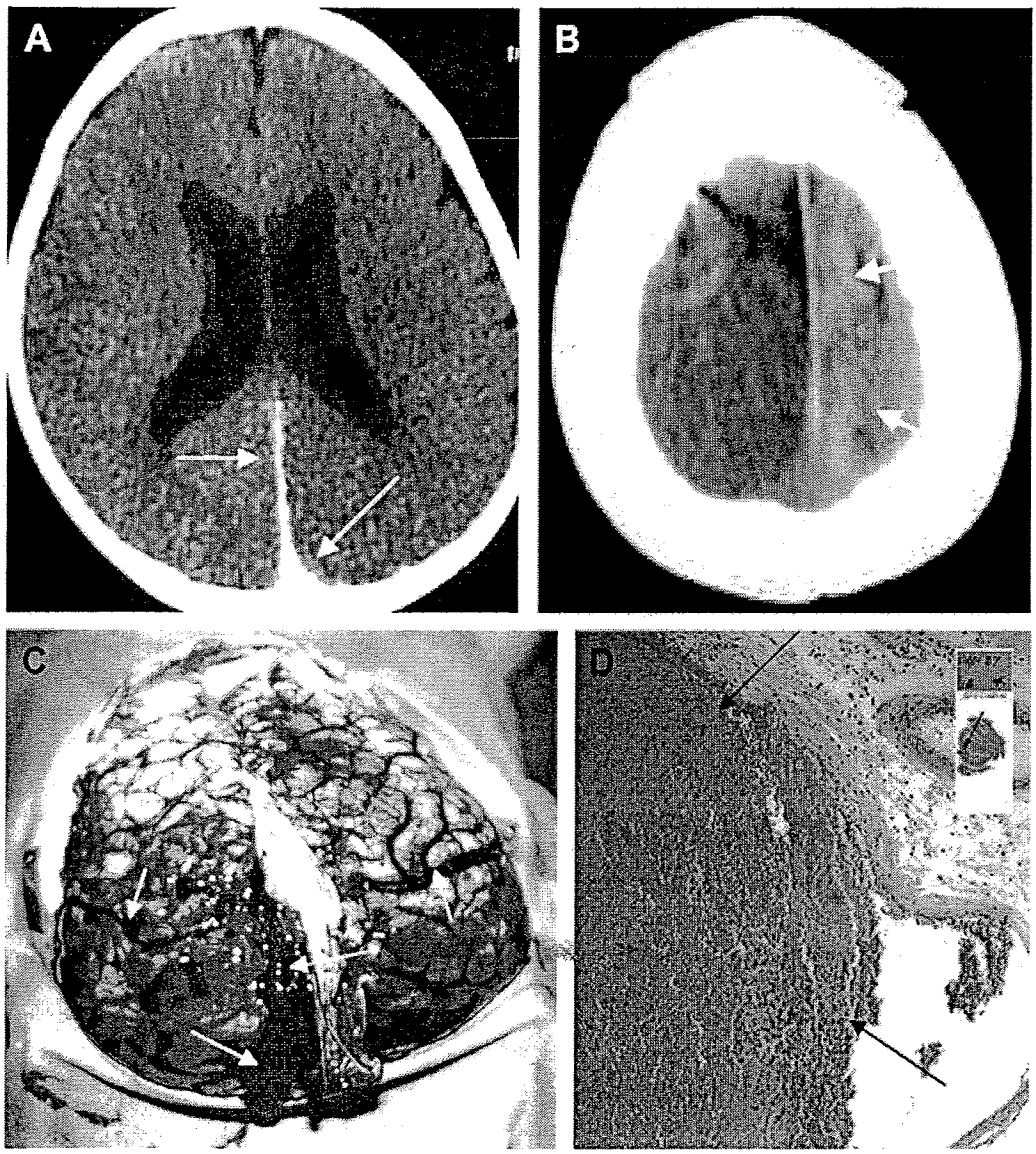

Fig. 8. Fourteen-month-old infant with triad and alleged NAI; also, recent infectious illness: dural and cortical venous sinus thrombosis with dural hemorrhage: CT (*A, B*) shows high densities along the falx and dural venous sinuses (*white arrows*). (*C*) Gross specimen—reflected superior sagittal sinus and cortical venous thromboses with distended veins (*yellow arrows*); (*D*) photomicrograph of cortical venous thrombus with inflammatory reaction (*black arrows*) plus SDH with neomembrane (7–14 days old; not shown). (*Pathology courtesy of* J. Leestma, MD.)

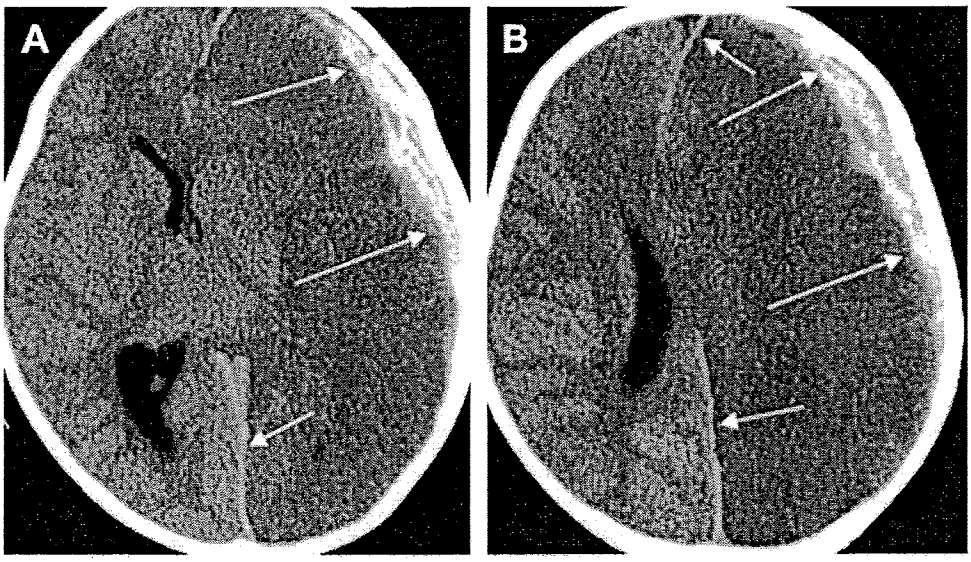

Fig. 9. Twenty-month-old infant with triad and alleged NAI. Left SDH with cerebral cortical and pial AVM at autopsy. CT (*A, B*) shows left mixed-density SDH and SAH (*long arrows*) plus interhemispheric hemorrhage (*short arrows*) with marked left cerebral swelling and shift.

The minimal force required to produce the triad has yet to be established. From the current biomechanical evidence base, however, it can be concluded that (1) shaking may not produce direct brain injury but may cause indirect brain injury if associated with neck and cervical spinal cord injury; (2) angular acceleration/deceleration injury forces clearly occur with impact trauma; (3) such injury on an accidental basis does not require a force that can only be associated with a motor vehicle accident or a multistory fall; (4) household (ie, short-distance) falls may produce direct or indirect brain injury; (5) in addition to fall height, impact surface and type of landing are important factors; and (6) head-first impacts in young infants not having developed a defensive reflex (eg, extension of a limb to break the fall) are the most dangerous and may result in direct or indirect brain injury (eg, SCIWORA).

NEUROPATHOLOGY CHALLENGES

In their landmark neuropathology study of 53 victims of alleged SBS/NAI,[36,37] Geddes and colleagues showed in 37 infants (ages <9 months) that (1) 29 had evidence of impact with only one case of admitted shaking; (2) cerebral swelling was more often due to DAI of hypoxic-ischemic encephalopathy (HIE) rather than shear or traumatic axonal injury (TAI); (2) although fracture, thin SDH (eg, dural vascular plexus origin), and RH are commonly present, the usual cause of

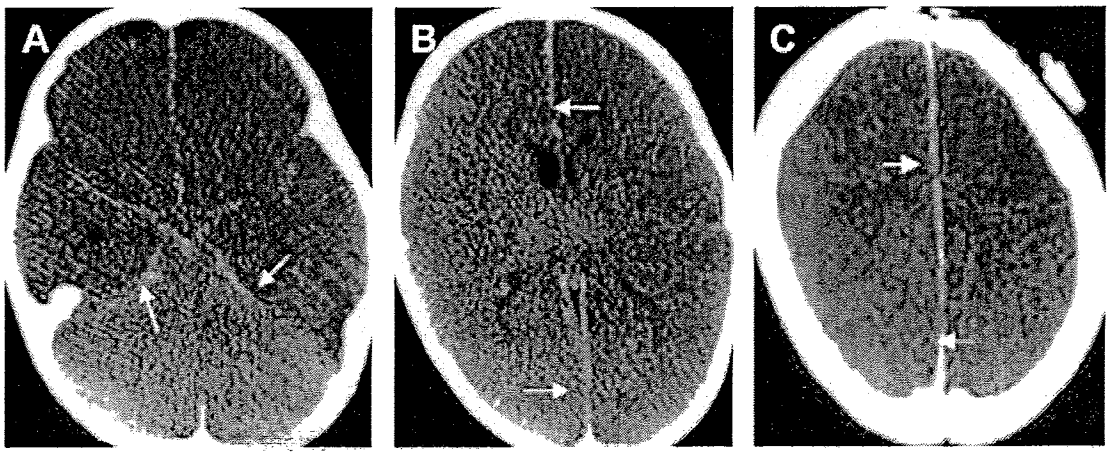

Fig. 10. Twenty-one–month–old infant with triad and alleged NAI. Pneumococcal meningitis, herniation, and hypoxic-ischemic injury confirmed at autopsy. CT (*A–C*) shows high-density thin SDH (*arrows*) plus cerebral edema.

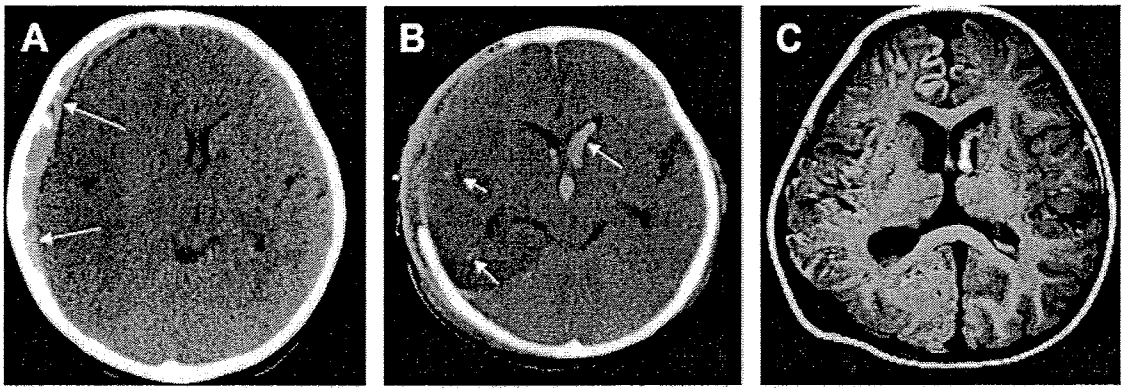

Fig. 11. Nine-month-old girl with triad and alleged NAI; also, recent fall and coagulopathy (later confirmed platelet disorder). Initial CT (*A*) shows mixed-density right SDH (*arrows*) with right cerebral edema. Postoperative CT 5 days later (*B*) shows other cerebral and intraventricular hemorrhages (*arrows*). T1 MR imaging (*C*) 11 days postoperatively shows evolving right cerebral high-intensity cortical injury and hemorrhages.

death was increased intracranial pressure from brain swelling associated with HIE (see **Fig. 2**); and (4) cervical epidural hemorrhage and focal axonal brainstem, cervical cord, and spinal nerve root injuries were characteristically seen in these infants (most with impact). Upper cervical cord/brainstem injury may result in apnea/respiratory arrest and be responsible for the HIE. In the 16 older victims (ages 13 months to 8 years), the pathology findings were primarily those of the battered child or adult trauma syndrome, including extracranial injuries (eg, abdominal), large SDH (ie, bridging vein rupture), and TAI. Additional neuropathology series by Geddes and colleagues[38] have shown that SDHs are also seen in nontraumatic fetal, neonatal, and infant brain injury cases and that such SDHs are actually of intradural vascular plexus origin rather than bridging cortical vein origin.

The common denominator in all these cases is likely a combination of vascular immaturity and fragility further compromised by HIE or infection, cerebral venous hypertension or congestion, arterial hypertension, and brain swelling (see **Fig. 2**). Although the unified hypothesis of Geddes and colleagues[13,14,39] has received criticism, their findings and conclusions have been validated by the research of Cohen and Scheimberg,[40] Croft and Reichard,[41] and others. In their postmortem series, Cohen and colleagues described 25 fetuses (26–41 weeks) and 30 neonates (1 hour–19 days) with HIE who also had macroscopic intradural hemorrhage (IDH), including frank parietal SDH in two-thirds. The IDH was most prominent along the posterior falcine and tentorial vascular plexuses (ie, interhemispheric fissure) (see **Fig. 2**). They concluded from their work, along with the findings of other cited

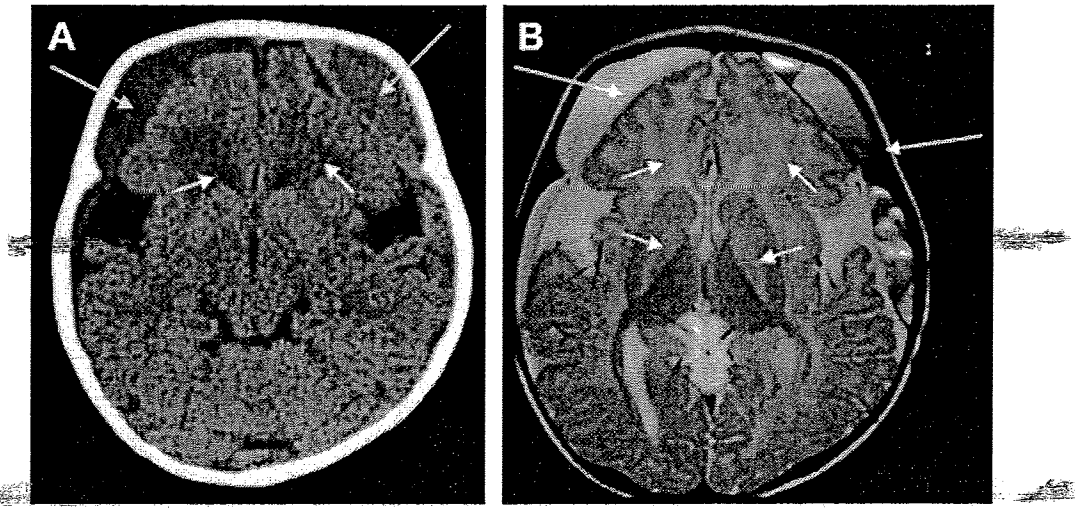

Fig. 12. Twelve-month-old infant with triad and alleged NAI. Glutaric acidopathy type 1. CT (*A*) and T2 MR imaging (*B*) shows bilateral SDH of varying age (*long arrows*), wide sylvian fissures plus basal ganglia, and cerebral white matter abnormalities (*short arrows*).

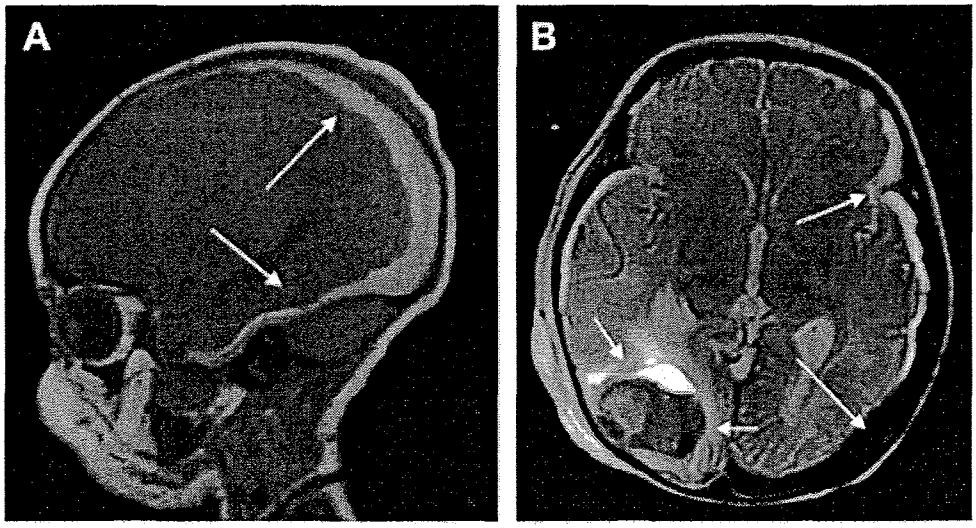

Fig. 13. Home-delivered newborn with seizures at 1 week of age; also, no vitamin K given at birth. T1 (*A*) and T2 (*B*) MR imaging shows acute-subacute left SDH (*long arrows*) plus right cerebral hemorrhage (*short arrows*); vitamin K deficiency confirmed and treated.

researchers, that IDH and SDH are commonly associated with HIE, particularly when associated with increases in central venous pressure. This also explains the frequency of RH associated with perinatal events.[42]

From the current forensic pathology evidence base, it may be concluded that (1) shaking may not cause direct brain injury but may cause indirect brain injury (ie, HIE) if associated with cervical spinal cord injury; (2) impact may produce direct or indirect brain injury (eg, SCIWORA); (3) the pattern of brain edema with thin SDH (dural vascular plexus origin) may reflect HIE whether or not due to AI or NAI; and (4) the same pattern of injury may result from nontraumatic or medical causes (eg, HIE from any cause of ALTE). Furthermore, because the observed edema does not represent TAI (which results in immediate neurologic dysfunction), a lucid interval is possible, particularly in infants whose sutured skull and

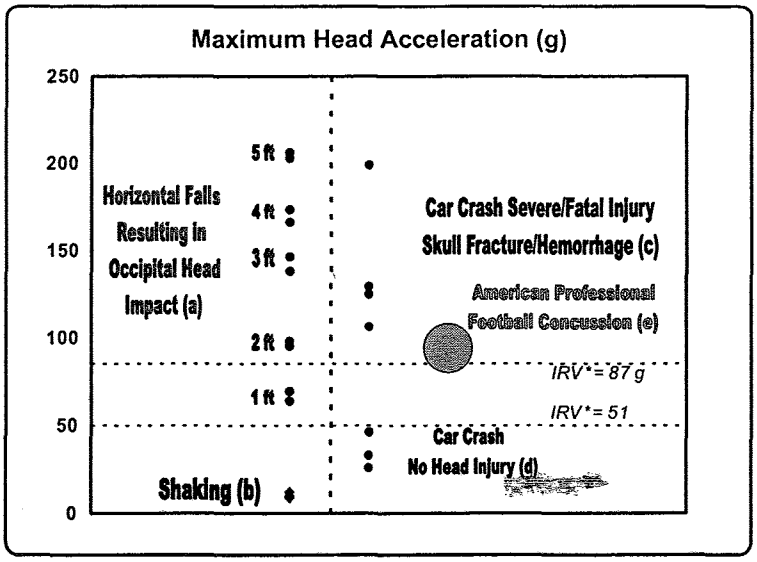

Fig. 14. Maximum head accelerations versus trauma mechanisms as correlated with injury thresholds. CRABI, child restraint air bag interaction; IRV, injury reference values. (*Data from* Van Ee C, PhD. Design research engineering. Available at: www.dreng.com. Accessed September 12, 2010; Leestma J. Forensic neuropathology. 2nd edition. Boca Raton [FL]: CRC Press; 2009; Mertz H. Anthropomorphic test devices. In: Melvin J, Nahum A, editors. Accidental injury: biomechanics and prevention. 2nd edition. New York: Springer; 2002. p. 84; Klinich JD, Hulbert G, Schneider LW. Estimating infant head injury criteria and impact response using crash reconstruction and finite element modeling. Society of Automotive Engineers Paper # 2002–22–0009, 2002; CRABI 12 [a, b]; CRABI 6 [c, d]; and [e] Pellman EJ, Viano DC, Tucker AM, et al. Concussion in professional football: reconstruction of game impacts and injuries. Neurosurgery 2003;53[4]:799–812.)

dural vascular plexus have the distensibility to tolerate early increases in intracranial pressure. Also, the lucid interval invalidates the premise that the last caretaker is always responsible in alleged NAI.

CLINICAL CHALLENGES

In the prosecution of NAI, it is often stipulated that short-distance falls cannot be associated with the triad, serious (eg, fatal) head injury, or a lucid interval. Traditionally, it has also been stipulated that nonintentional new bleeding in an existing SDH is always minor, that SDH does not occur in benign extracerebral collections (BECCs), and that symptomatic or fatal new bleeding in SDH requires newly inflicted trauma.[1–4,7,8,11] Several past and current reports refute the significance of low level falls in children, including in-hospital and outpatient clinic series.[43–51] There are other reports, however, including emergency medicine, trauma center, neurosurgical, and medical examiner series, that indicate a heightened need for concern regarding the potential for serious intracranial injury associated with minor or trivial trauma scenarios, particularly in infants.[52–74] This includes reports of skull fracture or acute SDH from accidental simple falls in infants, SDH in infants with predisposing wide extracerebral spaces (eg, BECCs of infancy, chronic subdural hygromas, arachnoid cyst, and so forth) (see **Fig. 4; Figs. 15** and **16**), and fatal pediatric head injuries due to witnessed, accidental short-distance falls, including those with a lucid interval, SDH, RH, and malignant cerebral edema (see **Fig. 6**). Also included are infants with chronic SDH from prior trauma (eg, at birth) who then develop rehemorrhage (see **Figs. 1, 4,** and **15**).

Short-Distance Falls, Lucid Intervals, and Malignant Edema

Hall and colleagues[44] reported that 41% of childhood deaths (mean age 2.4 years) from head injuries associated with AI were from low level falls (3 feet or less) while running or down stairs. Chadwick and colleagues[45] reported fatal falls of less than 4 feet in seven infants but considered the histories unreliable. Plunkett[56] reported witnessed fatal falls of 2 to 10 feet in 18 infants and children, including those with SDH, RH, and lucid intervals. Greenes and Schutzman[57] reported intracranial injuries, including SDH, in 18 asymptomatic infants with falls of 2 feet to 9 stairs. Christian and colleagues[63] reported three infants with unilateral RH and SDH/SAH due to witnessed accidental household trauma. Denton and Mileusnic[59] reported a witnessed, accidental 30-inch fall in a 9-month-old infant with a 3-day lucid interval before death. Murray and colleagues[60] reported more intracranial injuries in young children (49% <age 4 y; 21% <age 1 y) with reported low level falls (<15 ft), both AI and NAI. Kim and colleagues[61] reported a high incidence of intracranial injury in children (ages 3 mo to 15 y; 52% <age 2 y) accidentally falling from low heights (3 to 15 ft; 80% <6 ft; including 4 deaths). Because of the lucid intervals in some patients, including initially favorable Glascow Coma Scale scores (GCS) with subsequent deterioration, Murray and colleagues[60] and others expressed concern regarding caretaker delays and medical transfer delays contributing to the morbidity and mortality in these patients.[53–56,58–61] Bruce and colleagues[54,55] reported one of the largest pediatric series of head trauma (63 patients, ages 6 months to 18 years), both AI and NAI, associated with malignant brain edema and SAH/SDH (see **Fig. 6**). In the higher GCS (>8) subgroup,

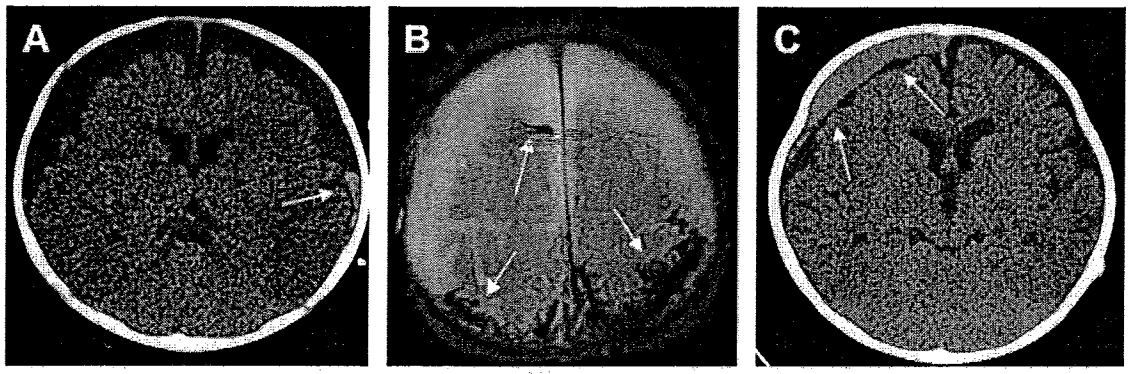

Fig. 15. Five-month-old infant with the triad and alleged NAI; also, macrocephaly from birth, recent seizure but no trauma. CT (*A*) and T2* MR imaging (*B*) shows large extracerebral collections with smaller recent hemorrhages (*arrows*). CT 3 months postdrainage (*C*) shows rehemorrhage (*arrows*). Diagnosis: BECC or chronic SDHG with rehemorrhage?

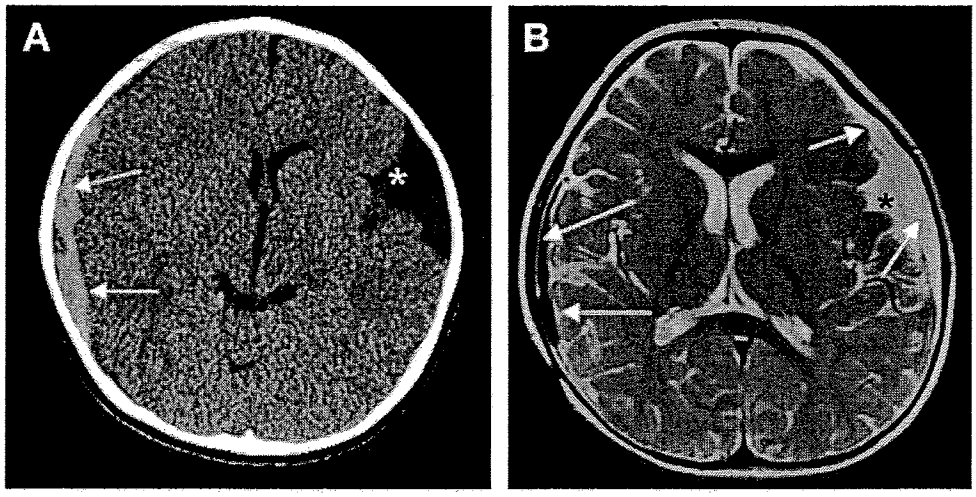

Fig. 16. Sixteen-month-old with triad (right RH) and alleged NAI; also, short-distance fall with right scalp impact. CT (*A*) shows left sylvian arachnoid cyst (*) and right hyperacute SDH (*arrows*). T2 MR imaging (*B*) 2 days later shows acute right SDH (*long arrows*) and smaller left sylvian arachnoid cyst (*) with subdural hygroma (*short arrows*).

there were 8 with a lucid interval and all 14 had complete recovery. In the lower GCS (≤ 8) subgroup, there were 34 with immediate and continuous coma, 15 with a lucid interval, 6 deaths, and 11 with moderate to severe disability. More recently, Steinbok and colleagues[62] reported 5 children (4 <age 2 y; 3 falls) with witnessed AI, including SDH and cerebral edema detected by CT 1 to 5 hours post event. All experienced immediate coma with rapid progression to death (see **Fig. 6**).

Benign Extracerebral Collections

BECCs of infancy (also known as benign external hydrocephalus or benign extracerebral subarachnoid spaces) is a common and well-known condition characterized by diffuse enlargement of the subarachnoid spaces.[65–74] A transient disorder of cerebrospinal fluid (CSF) circulation, probably due to delayed development of the arachnoid granulations, is widely accepted as the cause and develops from birth. BECC is typically associated with macrocephaly but may also occur in infants with normal or small head circumferences, including premature infants. As with any cause of craniocerebral disproportion (eg, BECC, hydrocephalus, chronic SDH or hygroma, arachnoid cyst, or underdevelopment or atrophy), there is a susceptibility to SDH that may be spontaneous or associated with trivial trauma (see **Figs. 4** and **15**). A recent large series report and review by Hellbusch[73] emphasizes the importance of this predisposition and cites other confirmatory series and case reports (30 references). Papasian and Frim[68] designed a theoretic model that predicts the predisposition of benign external hydrocephalus to SDH with minor head trauma. Piatt's[66] case report of BECC with SDH (27 references), including RH, along with McNeely and colleagues'[72] case series are further warnings that this combination is far from specific for SBS/NAI.

Birth Issues

In addition to the examples discussed previously (eg, short-distance falls and BECCs), another important but often overlooked factor is birth-related trauma.[1,4,75–89] This includes normal as well as complicated labor and delivery events (pitocin augmentation, prolonged labor, vaginal delivery, instrumented delivery, cesarean section, and so forth). It is well known that acute SDH often occurs even with the normal birth process and that this predisposes to chronic SDH, including in the presence of BECC (see **Figs. 1, 4,** and **15**). Intracranial hemorrhages, including SDH and RH, have been reported in several CT and MR imaging series of normal neonates including a frequency of 50% by Holden and colleagues,[81] 8% by Whitby and colleagues,[76] 26% by Looney and colleagues,[82] and 46% by Rooks and colleagues.[78] Chamnanvanakij and colleagues[75] reported 26 symptomatic term neonates with SDH over a 3-year period after uncomplicated deliveries. Long-term follow-up imaging has not been provided in many of these series, although Rooks and colleagues[78] reported one child in their series who developed SDH with rehemorrhage superimposed on BECC (**Fig. 17**).

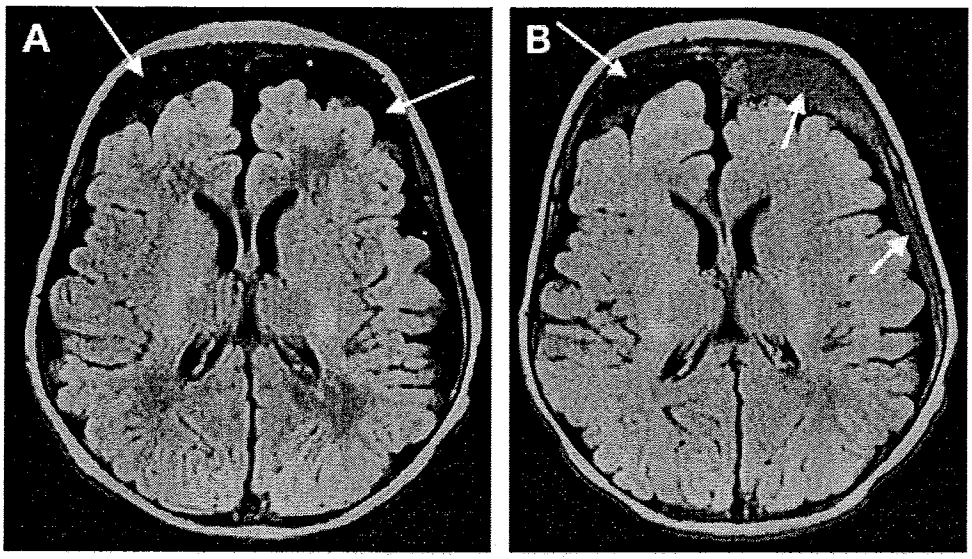

Fig. 17. BECC versus SDHG at birth (*A*) (*long arrows*) with SDH versus rehemorrhage 1 month later (*B*) (*yellow arrows*) on axial FLAIR MR images. (*Courtesy of* Veronica J. Rooks, MD, Tripler Army Medical Center, Honolulu, HI.)

Chronic SDH and Rehemorrhage

Chronic SDH is one of the most controversial topics in the NAI versus AI debate.[1–4,12,21,22,36–41] Unexplained SDH is often ascribed to NAI. By definition, a newly discovered chronic SDH started as an acute SDH that, for whatever reason, may have been subclinical. There is likely more than one mechanism for SDH that has prompted a revisiting of the concept of the subdural compartment.[12,40,41,90,91] Mack and colleagues[90] have provided an updated review on this important topic. In some cases of infant trauma, dissection at the relatively weak dura-arachnoid border zone (ie, dural border cell layer) may allow CSF to collect and enlarge over time as a dural interstitial (ie, intradural) hygroma. In other cases, there is bridging vein rupture within the dural interstitium that results in an acute subdural or intradural hematoma that extends along the dural border cell layer. Furthermore, traumatic disruption of the dural vascular plexus (ie, venous, capillary, or lymphatic), which is particularly prominent in young infants, may also produce an acute intradural hematoma. Some of these collections undergo resorption whereas others progress to become chronic SDH. Some progressive collections may represent mixed CSF-blood collections (see **Figs 1, 4,** and **15**).

The pathology and pathophysiology of neomembrane formation in chronic SDH, including rebleeding, is well established in adults and seems similar, if not identical, to that in infants.[83,92–112] Although acute SDH is most often due to impact or deformational trauma, whether or not AI or NAI, it must be differentiated from chronic SDH with rehemorrhage. Progression of chronic SDH and rehemorrhage is likely related to capillary leakage and intrinsic thrombolysis.[92,93] Other factors include dural vascular plexus hemorrhage associated with increases in intracranial or central venous pressures (eg, birth trauma, congenital heart disease, venous thrombosis, or dysphagic choking) or with increased meningeal arterial pressure (eg, reperfusion after hypoxia-ischemia) with resultant acute hemorrhage (or rehemorrhage) in normal infants or superimposed on predisposing chronic BECC, hygromas, hematomas, or arachnoid cysts (see **Figs. 1, 2, 4,** and **15–17**).[12,38,40,65–74,90,91] The phenomenon of acute infantile SDH, whether or not AI or NAI, evolving to chronic SDH and rehemorrhage, including RH, is well documented in several neurosurgical series reports, including those by Aoki and colleagues,[97,98] Ikeda and colleagues,[99] Parent,[94] Howard and colleagues,[102] Hwang and Kim,[95] Vinchon,[103,104] and others.

Conclusions

From the clinical evidence base, in addition to the biomechanical and neuropathology evidence bases, it may be concluded that (1) significant head injury, including SDH and RH, may result from low fall levels; (2) such injury may be associated with a lucid interval; (3) in some, the injury may result in immediate deterioration with progression to death; (4) BECC predisposes to SDH; (5) SDH may date back to birth; and (6) rehemorrhage into an existing SDH occurs in childhood and may be serious.

RH CHALLENGES

Many guidelines for diagnosing NAI depend on the presence of RH, including those of a particular pattern (eg, retinal schisis, and perimacular folds) and based on the theory of vitreous traction due to inflicted acceleration/deceleration forces (eg, SBS).[1–4,113–132] The specificity of RH for NAI has been repeatedly challenged, however. Plunkett[56] reported RH in two-thirds of eye examinations in children with fatal AI. Goldsmith and Plunkett[132] reported a child with extensive bilateral RH in a videotaped fatal accidental short-distance fall. Lantz and colleagues[122] reported RH with perimacular folds in an infant crush injury. Gilles and colleagues[120] reported the appearance and progression of RH with increasing intracranial pressure after head injury in children. Obi and Watts[125] reported RH with schisis and folds in two children, one with AI and the other with NAI. Forbes and colleagues[126] reported RH with epidural hematoma in five infant AI cases. From a research perspective, Brown and colleagues[128] found no eye pathology in their fatal shaken animal observations. Binenbaum and colleagues[127] observed no eye abnormalities in piglets subjected to acceleration/deceleration levels greater than 20 times what Prange and colleagues[30] predicted possible in inflicted injury. Emerson and colleagues[129] found no support for the vitreous traction hypothesis as unique to NAI. The eye and optic nerve are an extension of, and therefore a window to, the CNS, including their shared vascularization, meningeal coverings, innervation, and CSF spaces. RH has been reported with a variety of conditions, including AI, resuscitation, increased intracranial pressure, increased venous pressure, subarachnoid hemorrhage, sepsis, coagulopathy, certain metabolic dis,orders, systemic hypertension, and other conditions.[121,123,131] The common pathophysiology seems to be increased intracranial pressure or increased intravascular pressure. Furthermore, many cases of RH (and SDH) are confounded by the sequence or cascade of multiple conditions (eg, the unified hypothesis of Geddes) that often has a synergistic influence on the type and extent of RH. For example, consider the common situation of a child who has had trauma (factual or assumed) followed by seizures, apnea, or respiratory arrest and resuscitation with resultant HIE or coagulopathy. In much of the traditional NAI/SBS literature, little if any consideration has been given to any predisposing or complicating factors, and often there is no indication of the timing of the eye examinations relative to the clinical course or the brain imaging.[113,114,119,130]

From the research and clinical evidence base, it may be concluded that (1) RH is not specific for NAI, (2) RH may occur in AI and medical conditions, and (3) predisposing factors and complicating cascade effects must be considered in the pathophysiology of RH.

MEDICAL CONDITIONS MIMICKING NAI

A significant part of the controversy is the medical conditions that may mimic the clinical presentations (ie, the triad) and imaging findings of NAI.[1,2,4,25,26,89,101] Furthermore, such conditions may predispose to or complicate AI or NAI, as part of a cascade that results in or exaggerates the triad. In some situations, it may be difficult or impossible to tell which of these elements are causative and which are the effects. These include HIE, seizures, dysphagic choking ALTE, cardiopulmonary resuscitation, infectious or post-infectious conditions (eg, sepsis, meningoencephalitis, or postvaccinial), vascular diseases, coagulopathies, venous thrombosis, metabolic disorders, neoplastic processes, certain therapies, extracorporeal membrane oxygenation, and other conditions.[4,25,89,101] Regarding pathogenesis of the triad (with or without other organ system involement [eg, skeletal]) and whether or not due to NAI, AI, or medical etiologies, the pathophysiology seems to be a combination or sequence of factors, including increased intracranial pressure, increased venous pressure, systemic hypotension or hypertension, vascular fragility, hematologic derangement, and/or a collagenopathy imposed on the immature CNS, including the vulnerable dural vascular plexus as well as other organ systems.[4,12,25,38,90] Although the initial medical evaluation, including history, laboratory tests, and imaging studies, may suggest an alternative condition, the diagnosis may not be made because of a rush to judgment regarding NAI.[4–11] Such bias may have devastating effects on an injured child and family. It is important to be aware of these mimics, because a more extensive work-up may be needed beyond routine screening tests. Also, lack of confirmation of a specific condition does not automatically indicate the default diagnosis of NAI. In all cases, it is critical to review all past records dating back to the pregnancy and birth as well as the postnatal pediatric records, family history, more recent history preceding the acute presentation, details of the acute event itself, resuscitation, and the subsequent management, all of which may contribute to the clinical and imaging findings. An incomplete medical evaluation may result in unnecessary cost shifting to

child protection and criminal justice systems and have further adverse effects regarding transplantation organ donation in brain death cases and custody/adoptive dispositions for the surviving child and siblings.

Sirotnak's[89] recent review, along with others', extensively catalogs the many conditions that may mimic NAI[4,25,101]:

Birth Trauma and Neonatal Conditions

Manifestations of birth trauma, including fracture, SDH, and RH, may persist beyond the neonatal period. Other examples are the sequelae of extracorporeal membrane oxygenation therapy, at-risk prematurity, and congenital heart disease. When evaluating a young infant with apparent NAI, it is important to consider that the clinical and imaging findings may actually stem from parturitional and neonatal issues.[75–112] These include hemorrhage or rehemorrhage into extracerebral collections existing from birth (see **Figs. 1, 4, 13**, and **15**). There may be associated skeletal findings of birth trauma (eg, new or healing clavicle, rib, or long bone fractures), particularly in the presence of a bone fragility disorder (see **Figs. 1, 2** and **5**).[133–137]

Developmental Anomalies and Congenital Conditions

Vascular malformations are rarely reported causes for the triad but may be underdiagnosed (see **Fig. 9**). BECCs and arachnoid cysts are also known to be associated with SDH and RH, spontaneously and with trauma (see **Figs. 4, 15–17**).[65–74]

Genetic and Metabolic Disorders

Several conditions in the genetic and metabolic disorders category may present with intracranial hemorrhage (eg, SDH) or RH. These include osteogenesis imperfecta, glutaric aciduria type I (see **Fig. 12**), Menkes' kinky hair disease, Ehlers-Danlos and Marfan syndromes, homocystinuria, and others.[4,89,101,138–142]

Hematologic Disease and Coagulopathy

Conditions in the hematologic disease and coagulopathy category predispose to intracranial hemorrhage and RH (see **Figs. 11** and **13**). The bleeding or clotting disorder may be primary or secondary. A more extensive work-up beyond the usual screening tests is needed, including a hematology consultation. Conditions in the category include the anemias, hemorrhagic disease of the newborn (vitamin K deficiency), the hemophilias, thrombophilias, disseminated intravascular coagulation and consumption coagulopathy, liver or kidney disease, hemophagocytic lymphohistiocytosis, and anticoagulant therapy.[4,89,101,143–145] Venous thrombosis includes dural venous sinus thrombosis (DVST) and cerebral venous thrombosis (CVT). DVST or CVT may be associated with primary or secondary hematologic or coagulopathic states.[4,89,101,146–152] Risk factors include acute systemic illness, dehydration, fluid-electrolyte imbalance, sepsis, perinatal complications, chronic systemic disease, cardiac disease, connective tissue disorder, hematologic disorder, oncologic disease and therapy, head and neck infection, hypercoagulable, and trauma states. Infarction, SAH, SDH, or RH may be seen, especially in infants. High densities on CT may be present along the dural venous sinuses, tentorium, falx, or the cortical, subependymal, or medullary veins and be associated with SAH, SDH, or intracerebral hemorrhage (see **Fig. 8**). There may be focal infarctions, hemorrhagic or nonhemorrhagic, intraventricular hemorrhage, and massive, focal, or diffuse edema. Orbit, paranasal sinus, or otomastoid disease may be present. The thromboses and associated hemorrhages have variable MR imaging appearances depending on their age. CT venography (CTV) or magnetic resonance venography (MRV) may readily detect DVST but not CVT. The latter may be better detected as abnormal hypointensities on susceptibility-weighted T2* sequences but difficult to distinguish from hemorrhage (SDH or SAH), hemorrhagic infarction, contusion, or hemorrhagic shear injury.

Infectious and Postinfectious Conditions

Meningitis, encephalitis, or sepsis may involve the vasculature resulting in vasculitis, arterial or venous thrombosis, mycotic aneurysm, infarction, and hemorrhage.[4,89,101] SDH and RH may also be seen (see **Fig. 10**). Postinfectious illnesses may also be associated with these findings. Included in this category are the encephalopathies of infancy and childhood, hemorrhagic shock and encephalopathy syndrome, and postvaccinial encephalopathy.[4,89,101,153–158]

Toxins, Poisons, and Nutritional Deficiencies

The category of toxins poisons, and nutritional deficiencies includes lead poisoning, cocaine, anticoagulants, over-the-counter cold medications, prescription drugs, and vitamin deficiencies or depletions (eg, K, C, or D).[4,89,101,136,143,155–159] Preterm neonates, and other chronically ill infants, are particularly vulnerable to nutritional deficiencies and complications of prolonged immobilization that often primarily effect bone development. Furthermore, the national and

international epidemic of vitamin D deficiency and insufficiency in pregnant mothers, their term fetuses, and their undersupplemented breastfed term neonates predisposes them to rickets (ie, congenital). Such infants, who have also been subjected to the trauma of birth, may have skeletal imaging findings (eg, multiple healing fractures or pseudofractures) that are misinterpreted as NAI, especially in the presence of the triad (see **Figs. 2** and **5**).[136,137]

Dysphagic Choking ALTE as a Mimic of NAI

Apnea is an important and common form of ALTE in infancy whose origin may be central, obstructive, or combined.[25] The obstructive and mixed forms may present with choking, gasping, coughing, or gagging due to mechanical obstruction. When paroxysmal or sustained, the result may be severe brain injury or death due to a combination of central venous hypertension and hypoxia-ischemia. It is this synergism that produces cerebral edema and dural vascular plexus hemorrhage with SDH, SAH, and RH (see **Fig. 2**; **Fig. 18**). Examples include dysphagic choking (eg, aspiration of a feed or gastroesophageal reflux), viral airway infection (eg, RSV), and pertussis, particularly when occurring in a predisposed child (eg, prematurity, Pierre Robin syndrome, or sudden infant death syndrome).[25,160–167]

IMAGING CHALLENGES AND THE IMPORTANCE OF A DIFFERENTIAL DIAGNOSIS

CT

Because of the evidence-based challenges to NAI, imaging protocols should be designed to evaluate not only NAI versus AI but also the medical mimics. Noncontrast CT has been the primary modality for brain imaging because of its access, speed, and ability to show lesions (eg, hemorrhage and edema) requiring immediate neurosurgical or medical intervention.[4,77,83–99,102–112,168–181] Cervical spinal CT may also be needed. CT angiography (CTA) or CTV may be helpful to evaluate the cause of hemorrhage (eg, vascular malformation or aneurysm) or infarction (eg, dissection or venous thrombosis). A radiographic or scintigraphic skeletal survery should also be obtained according to established guidelines.[179,180]

MR Imaging

Brain and cervical spinal MR imaging should be done as soon as possible because of its sensitivity and specificity regarding pattern of injury and timing parameters.[4,104,181–190] Brain MR imaging should include T1, T2, T2*, fluid-attenuated inversion recovery (FLAIR), and diffusion-weighted imaging/apparent diffusion coefficient (DWI/ADC). Gadolinium-enhanced T1 images should probably be used along with MRA and MRV. T1 and T2 are necessary for estimating the timing of hemorrhage, thrombosis, and other collections using published criteria.[4,104,181] T2* techniques are most sensitive for detecting hemorrhage or thromboses but may not distinguish new (eg, deoxyhemoglobin) from old (eg, hemosiderin). DWI plus ADC can be quickly obtained to show hypoxia-ischemia or vascular occlusive ischemia.[4,154,189,190] Restricted or reduced diffusion, however, may be seen with other processes, including encephalitis, seizures, or metabolic disorders, and with suppurative collections and some tumors.[4,154,189,190] Gadolinium-enhanced sequences and MRS can be used to evaluate for these other processes. Additionally, MRA and

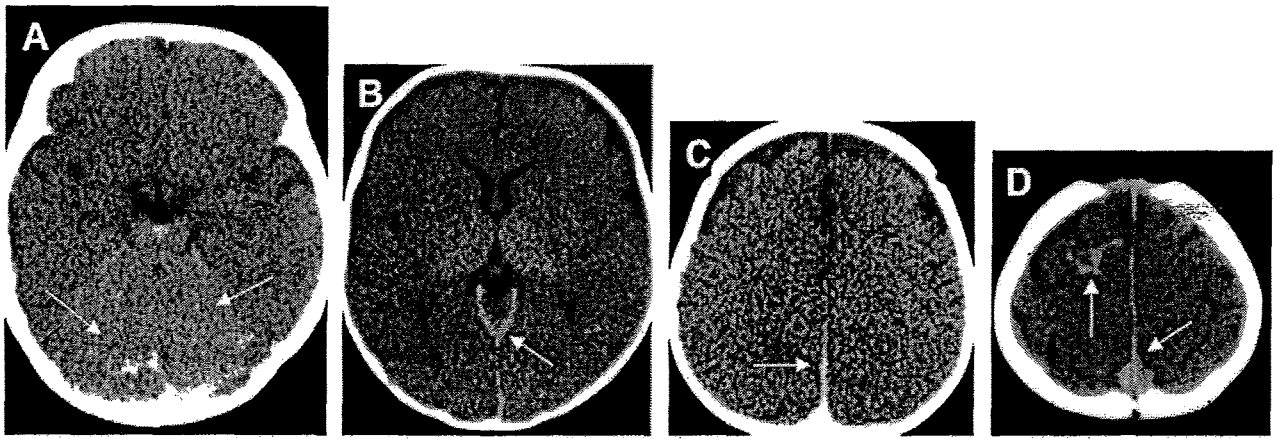

Fig. 18. Six-month-old infant with triad and alleged NAI; acute choking event while feeding. CT (*A–D*) shows bilateral cerebral edema with acute SAH and SDH (*arrows*), including along the falx, and tentorium. Autopsy confirmed the hemorrhages, a subdural membrane, and hypoxic-ischemic brain injury. (*Courtesy of* The Wisconsin Innocence Project.)

MRV are important to evaluate for arterial occlusive disease (eg, dissection) or venous thrombosis, although they cannot rule out small vessel disease. The STIR technique is particularly important for cervical spine imaging.

Scalp and Skull Abnormalities

Scalp injuries (eg, edema, hemorrhage, and laceration) are difficult to precisely time on imaging studies and depend on the nature and number of traumatic events or other factors (circulatory compromise, coagulopathy, medical interventions, and so forth).[1,4] Skull abnormalities may include fracture and suture splitting. Fracture may not be readily distinguished from sutures, synchondroses, their normal variants, or from wormian bones (eg, osteogenesis imperfecta) on CT or skull films. 3-D–CT surface reconstructions may be needed. In general, the morphology of a fracture cannot differentiate NAI from AI and must be correlated with the trauma scenario (eg, biomechanically) (see **Fig. 1**). Skull fractures are also difficult to time because of the lack of periosteal reaction.[1,4] Suture diastasis may be traumatic or a reflection of increased intracranial pressure but must be distinguished from pseudodiastasis due to a metabolic or dysplastic bone disorder (eg, congenital rickets) (see **Fig. 2**).[1,4,136,137] The growing fracture (eg, leptomeningeal cyst) is not specific for NAI and may follow any diastatic fracture in a young infant, including birth related (see **Fig. 1**).[1,2,4] Nondetection of scalp or skull abnormalities on imaging should not be interpreted as the absence of impact injury.

Intracranial Collections

It should not be assumed that such collections are always traumatic in origin. A differential diagnosis is always necessary and includes NAI, AI, coagulopathy (hemophilic and thrombophilic conditions), infectious and postinfectious conditions, metabolic disorders, and so forth.[2,4,22,89,90,101,106–110] It may not be possible to specify with any precision the components or age of an extracerebral collection because of meningeal disruptions (eg, acute or subacute subdural hygroma [SDHG] versus chronic SDH, or subarachnoid versus thin SDH).[1,4,103,104,173–176,181] Vezina[181] has recently summarized the literature regarding the complexity of timing of intracranial collections. Subarachnoid and subdural collections, hemorrhagic or nonhemorrhagic, may be localized or extensive and may occur about the convexities, interhemispheric (along the falx), and along the tentorium. With time and gravity, these collections may redistribute to other areas, including into or out of the spinal canal, and cause confusion.[4,177,181,191] For example, a convexity SDH may migrate to the peritentorial and posterior interhemispheric regions or into the intraspinal spaces. SDH migration may lead to a misinterpretation that there are hemorrhages of different timing. The distribution or migration of the sediment portion of a hemorrhage with blood levels (ie, hematocrit effect) may cause further confusion because density/intensity differences between the sediment and supernatant may be misinterpreted as hemorrhages (and trauma) of differing age and location.[4,104,178,181] Prominent subarachnoid CSF spaces are commonly present in infants (ie, BECCs). This entity predisposes infants to SDH, which may be spontaneous or associated with trauma of any type (eg, dysphagic choking ALTE) (see **Figs. 4, 15,** and **17**).[4,65–73] A hemorrhagic collection may continually change or evolve with regard to size, extent, location, and density/intensity characteristics. Rapid spontaneous resolution and redistribution of acute SDH over a few hours to 1 to 2 days has been reported.[4,177,191] A tear in the arachnoid may allow SDH washout into the subarachnoid space or CSF dilution of the subdural space.

For apparent CT high densities, it may be difficult to differentiate cerebral hemorrhage from subarachnoid hemorrhage or from venous thrombosis (see **Figs. 2, 3, 6–11, 15, 16,** and **18**).[4] According to the literature, hemorrhage or thromboses that are high density (ie, clotted) on CT (ie, acute to subacute) have a wide timing range of 0 to 3 hours up to 7 to 10 days.[4,104,178,181] Hemorrhage that is isohypodense on CT (ie, nonclotted) may be hyperacute (<3 h) or chronic (>10 d) (see **Figs. 3** and **11**). The low density may also represent pre-existing, wide, CSF-containing subarachnoid spaces (eg, BECC) or SDHG (ie, CSF-containing) that may be acute or chronic (see **Figs. 3, 12** and **15**).[4,103,104,175,181] Blood levels are unusual in the acute stage unless there is coagulopathy.[4,104,181,188] CT cannot distinguish acute hemorrhage from rehemorrhage on existing chronic collections (BECC or chronic SDHG) (see **Figs. 3** and **15**).[4,66,72,92,164,173,178,181] Traditionally, the interhemispheric SDH as well as mixed-density SDH were considered characteristic, if not pathognomonic, of SBS/NAI.[1,2,4,168,171–173] This has been proved unreliable. Interhemispheric SDH may be seen with AI or with nontraumatic conditions (eg, HIE, venous thrombosis, venous hypertension, or dysphagic choking ALTE) (see **Figs. 2, 6–10**).[178] Mixed-density SDH also occurs in AI as well as in other conditions (see **Figs. 3, 9,** and **11**).[178] Furthermore, SDH may occur in BECC

spontaneously or result from minor trauma (ie, AI), and rehemorrhage within SDH may occur spontaneously or with minor AI (see **Figs. 1, 4, 15,** and **17**).[4,12,38,40,72,90,104,178,181]

Only MR imaging may provide more precise information than CT regarding pattern of injury and timing, particularly with regard to (1) hemorrhage versus thromboses (**Table 1**) and (2) brain injury.[104,181–190] As a result, MR imaging has become the standard and should be done as soon as possible. Mixed-intensity collections, however, are problematic regarding timing.[181] Matching the MR imaging findings with the CT findings may help along with follow-up MR imaging. Blood levels may indicate subacute hemorrhage versus coagulopathy. The timing guidelines are better applied to the sediment than to the supernatant. With mixed-intensity collections, MR imaging cannot reliably differentiate BECC with acute SDH from acute SDHG/SDH, from hyperacute SDH, or from chronic SDH or chronic SDHG with rehemorrhage (see **Figs. 1, 4,** and **13–17**).[4,104,181] T2* hypointensities are iron sensitive but may not differentiate hemorrhages from venous thromboses that are not detected by MRV (eg, cortical, medullary, or subependymal).

BRAIN INJURY

Edema or swelling in pediatric head trauma may represent primary injury or secondary injury and be acute-hyperacute (eg, minutes to a few hours) or delayed (eg, several hours to a few days), including association with short-distance falls and lucid intervals.[4,53–62] The edema or swelling may be further subtyped as traumatic, malignant, hypoxic-ischemic, or related to (or combined with) other factors. Traumatic edema is related to areas of primary brain trauma (ie, contusion or shear) or to traumatic vascular injury with infarction (eg, dissection, herniation, or spasm) (see **Figs. 3, 6, 9,** and **11**). Traumatic edema is usually focal or multifocal, whether or not hemorrhagic. CT, however, may not distinguish focal or multifocal cerebral high densities as hemorrhagic contusion, hemorrhagic shear, or hemorrhagic infarction.[4] Focal or multifocal low density edema may also be seen with infarction (eg, arterial or venous occlusive), encephalitis, demyelination (eg, ADEM), or seizure edema.[4,89,146–154] Also, MR imaging often shows shear and contusional injury as focal/multifocal restricted diffusion, GRE hypointensities, and/or T2/FLAIR high intensities.[4] Focal/multifocal ischemic findings may also be due to traumatic arterial injury (eg, dissection) or venous injury (eg, tear or thrombosis), arterial spasm (as with any cause of hemorrhage), herniation, or edema with secondary perfusion deficit or seizures (eg, status epilepticus) (see **Figs. 2, 6,** and **11**).[4,64,154,189,192] These may not be reliably differentiated, however, from focal/multifocal ischemic or hemorrhagic infarction from nontraumatic causation (eg, dissection, vasculitis, venous, or embolic) even without supportive MRA, CTA, MRV, or angiography. Also, similar cortical or subcortical intensity abnormalities (including restricted diffusion) may also be observed with

Table 1
MR imaging of intracranial hemorrhage and thrombosis[a]

Stage	Biochemical Form	Site	T1–MR Imaging	T2–MR Imaging
Hyperacute (+ edema) (<12 hours)	Fe II oxyHb	Intact RBCs	Iso-low I	High I
Acute (+ edema) (1–3 days)	Fe II deoxy Hb	Intact RBCs	Iso-low I	Low I
Early subacute (+ edema) (3–7 days)	Fe III metHb	Intact RBCs	High I	Low I
Late subacute (–edema) (1–2 weeks)	Fe III metHb	Lysed RBCs (extracellular)	High I	High I
Early chronic (–edema) (>2 weeks)	Fe III transferrin	Extracellular	High I	High I
Chronic (cavity)	Fe III ferritin and hemosiderin	Phagocytosis	Iso-low I	Low I

[a] Fe II, ferrous; Fe III, ferric; Hb, hemoglobin; I, signal intensity; Iso, isointense; RBCs, red blood cells; +, present; –, absent. *Data from* Refs. [4,188,189]

encephalitis, seizures, and metabolic disorders. Therefore, a differential diagnosis is always required.[4,154,189,192]

Malignant brain edema, a term used for severe cerebral swelling after head trauma, may lead to rapid deterioration.[1,4,54,55,62] The edema is usually bilateral and may be related to cerebrovascular congestion (ie, hyperemia) as a vasoreactive rather than an autoregulatory phenomenon and associated with global ischemia. A unilateral form may also occur in association with an ipsilateral SDH that progresses to bilateral edema (see **Figs. 3** and **6**).[64] There may be rapid or delayed onset (ie, lucid interval). Predisposing factors are not well established but likely include a genetic basis. Hyperemic edema may appear early as accentuated gray-white matter differentiation on CT, then progresses to loss of differentiation.

Global hypoxia (eg, apnea or respiratory failure) or ischemia (eg, cardiovascular failure or hypoperfusion) is likely a major cause of or contributor to brain edema in a child with head trauma (eg, malignant edema).[4,38,40,54,55,62] HIE, depending on its severity and duration, may have a diffuse appearance acutely (ie, diffuse or vascular axonal injury) with decreased gray-white differentiation throughout the cerebrum on CT (eg, white cerebellum sign) and then evolve to a more specific pattern on CT or MR imaging (eg, border zone or watershed, basal ganglia/thalamic, cerebral white matter necrosis, reversal sign) (see **Figs. 2, 6, 7, 10,** and **18**).[4,189] It is typically bilateral but may not be symmetric. This more diffuse pattern may distinguish HIE from the multifocal pattern of primary traumatic injury, although they may coexist. Hypoxia-ischemic brain injury due to apnea/respiratory arrest may occur with head trauma or with neck/cervical spine/cord injuries (eg, SCIWORA) whether or not AI or NAI (see **Fig. 7**).[4,35,54,55,62] It may also occur with any nontraumatic cause (choking, paroxysmal coughing, aspiration, and so forth) (see **Figs. 2** and **18**).[4,25,160–166] In addition to the diffuse brain injury, there may be associated subarachnoid and SDH without mass effect (see **Figs. 2, 7, 10,** and **18**).[4,38,40,54,55,62] MR imaging shows hypoxic-ischemic injury, depending on timing, as diffuse-restricted diffusion on DWI/ADC plus matching T1/T2 abnormalities as the injury evolves (see **Figs. 2, 6** and **11**).[4,189] Other important contributors to edema or swelling include such complicating factors as seizures (eg, status epilepticus [see **Fig. 2**], fluid-electrolyte imbalance, other systemic or metabolic derangements (eg, hypoglycemia, hyperglycemia, hyperthermia), or hydrocephalus.[4] It is well known that many of these may also be associated with restricted diffusion along with other nontraumatic processes (encephalitis, seizures, and metabolic disorders).[4,154,186,187,189] Again, a differential diagnosis is required.

SUMMARY

An extensive review of the literature to date fails to establish an evidence base for reliably distinguishing NAI from AI or from the medical mimics. The medical and imaging findings alone cannot diagnose intentional injury. Only a child protection investigation may provide the basis for inflicted injury in the context of supportive medical, imaging, or pathologic data. The duty of a radiologist is to give a detailed description of the imaging findings, provide a differential diagnosis, and communicate the concern for NAI, directly to the primary care team in a timely manner. Radiologists should be prepared to consult with child protection services; other medical and surgical consultants, including a pathologist or biomechanical specialist; law enforcement investigators; and attorneys for all parties as appropriate. Radiologists must also be aware of certain conditions that are known to have clinical and imaging features that may mimic abuse. These should be properly evaluated, and the possibility of combined or multifactorial mechanisms with synergistic effects should also be considered. Furthermore, a negative medical evaluation does not make NAI the default diagnosis. A timely and thorough multidisciplinary evaluation may be the difference between appropriate child protection versus an improper breakup of a family or a wrongful indictment and conviction.

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STATE OF ILLINOIS
IN THE CIRCUIT COURT OF THE EIGHTEENTH JUDICIAL CIRCUIT
DUPAGE COUNTY

Randy Liebich, Petitioner	)	Cir. Ct. No. 02-CF-654
v.	)	Post Conviction No.
People of the State of Illinois	)	

AFFIDAVIT OF MICHAEL LAPOSATA, M.D., Ph.D.

1. My name is Michael Laposata. I am a Professor of Pathology and Medicine at Vanderbilt University School of Medicine. I am also Pathologist-in-Chief, Vanderbilt University Hospital, and Director, Division of Laboratory Medicine and Clinical Laboratories, Vanderbilt University Medical Center. Before assuming these responsibilities, I was a Professor of Pathology at Harvard Medical School with clinical responsibilities at various institutions, including Massachusetts General Hospital (Mass General). I have an M.D. and Ph.D. in Biochemistry, Cellular and Molecular Biology from the Johns Hopkins University School of Medicine. My curriculum vita is attached as Exhibit 1.

2. I have particular expertise in coagulation, and I teach and publish regularly in this area. When I was at Mass General (which is affiliated with Harvard), we found that some children with coagulopathies (bleeding or clotting disorders) were being misdiagnosed as victims of abuse. This is an easy mistake since it is frequently impossible to distinguish visually between bruises and bleeding caused by abuse, and bruises and bleeding caused by a coagulopathy. See Exhibits 2 and 3.

3. I have been asked to comment on the laboratory reports for Steven Quinn. In addition to the laboratory reports, I have been provided with the autopsy findings, a brief clinical history, and hospital and autopsy photographs of various marks on the child's body.

Lab reports

4. Mt. Sinai hospital (2/8, 6:22 pm). The most striking abnormalities are the highly elevated amylase and lipase. The amylase was 3025 U/L (ref. range 20-120) and the lipase was 2368 U/L (ref. range 22-51). These lab results confirm that the child had pancreatic damage but do not indicate what caused it or when it began.

5. The glucose was high at 517 mg/dL (ref. range 60-100), and the urine showed high glucose (250 mg/dL), high ketones (15 mg/dL), high protein (over 300 mg/dL), moderate blood, and high red blood cells (16/hpf). These results are found in patients with pancreatic injury.

6. The elevated white blood cell count (19.1 th wbc/microL), high neutrophils (81%) and low lymphocytes (12.8%) are common indicators of infection and/or inflammation.

7. The prothrombin time (PT) was mildly elevated at 15.3 seconds, the PTT was normal and the INR was mildly elevated at 1.9.

8. The lab tests showed low amounts of acetaminophen and salicylate, suggesting that he had been given pain medications (e.g., Tylenol, aspirin).

9. It is my understanding that an abdominal CT scan was scheduled but cancelled.

10. Rush Hospital (2/8, 9 p.m.). The lab tests taken at Rush shortly after transfer show pancreatic damage and liver involvement with an evolving coagulopathy (bleeding/clotting disorder).

11. The lipase increased from 2368 to 9598 U/L while the amylase decreased from 3025 to 1131 U/L. The glucose decreased but remained elevated at 207 mg/dL. Liver function tests were very abnormally high (SGOT 5429 U/L; SGPT 2964/3130 U/L). The SGOT may reflect heart or liver dysfunction, or damage to other organs; the SGPT is more specific for liver involvement.

12. The platelets decreased from 512 to 399 th/microL, with the likely explanation that they were being rapidly consumed and that the child had disseminated intravascular coagulation (DIC).

13. A neurosurgical operation beginning at approximately 10 p.m. did not find the expected subdural hemorrhage but found severe brain swelling to an extent nearly inconsistent with life. The abdominal CT was again cancelled.

14. Subsequent labs. Approximately one hour after surgery, the child's platelets had dropped to 118 th/microL, confirming persistent thrombocytopenia as found in DIC. Despite repeated transfusions, the low platelet count continued throughout the hospital stay, decreasing at one point to 71 th/microL.

15. Coagulation tests taken at 5 a.m. on 2/9 showed normal results for PT, PTT and INR following two transfusions. The presence of schistocytes at 6:50 a.m. was consistent with a diagnosis of DIC. At 11:25 a.m., all three coagulation tests were elevated (PT 18.4 sec, PTT 41 sec, INR 1.67).

16. A blood culture taken at 1:45 p.m. on 2/9 did not show growth of any organisms, suggesting that the child was not yet septic and that the infection was likely limited to the abdomen. Antibiotics had been ordered approximately half an hour earlier but probably had not yet been given.

17. A penrose drain was inserted on the morning of 2/9 and 500 mL of serosanguinous pink fluid was drained. No cultures were taken.

18. There was no abdominal CT, no abdominal surgery and no culture of the peritoneal fluid, limiting the full assessment of the cause and/or timing of the abdominal findings .

Clinical history

19. According to the information provided, the child had cold-like symptoms and possible lethargy for some days prior to collapse. He may have had a stomach ache two or three days before collapse. The night before collapse, he cried and refused to eat, and this provoked his mother to spank him. There are conflicting reports on whether he ate later that evening.

20. The following morning, the child ate some cereal but left the milk. At about 3 p.m., he drank orange juice and water, and ate some portion of a hot dog, on which he choked. This was followed by vomiting. Choking on a hot dog is consistent with a child who has abdominal inflammation/infection and whose stomach is unable to accept food.

21. The mother returned home around 4:30 or 5 p.m. and noticed that the child was breathing oddly and appeared to be trying to vomit. The child reportedly vomited a small amount of greenish-black fluid.

22. The child was taken to the hospital, with a stop en route at the boyfriend's work. The boyfriend's manager described the child as blinking and looking ill, with no evident signs of trauma. The child may have vomited on the way to the hospital.

23. At the hospital, the child was initially viewed as ill rather than traumatized. However, the diagnosis changed to trauma when a subdural hemorrhage was identified on a CT scan and various marks and lines began to appear on the child's body.

Autopsy

24. The autopsy identified a necrotic bowel (7 inches) with a small bowel perforation (0.1 inch); peritonitis (inflammation in the peritoneal cavity surrounding the organs); pancreatitis; and a healing subcapsular hematoma on the liver. It is my understanding that only a portion of the pancreas was involved, suggesting that pancreatitis was a late development, possibly occurring at about the time of hospitalization.

25. The autopsy also identified a residual subdural hematoma, a severely swollen hypoxic ischemic brain, and many bruises/discolorations, some of which were identified as artifacts.

Comments

26. Given the history and autopsy findings, the child's illness likely began with an ischemic bowel. As the walls of the ischemic bowel deteriorated, the contents leaked into the peritoneal cavity, affecting the surrounding organs, including the pancreas.

27. As the inflammation spread, the body would have produced additional platelets to help stop the process of bleeding. The platelets were consumed in the process known as DIC, which produces bleeding, bruising and thrombosis.

28. A child in DIC may bruise spontaneously or from minor trauma, including medical intervention or handling. It is not possible to determine visually whether bruises or contusions are caused by trauma or a coagulopathy. Since the marks on the child were not apparent on admission but appeared during hospitalization, some or all were likely due to DIC and handling of the child as care was being provided, rather than pre-existing trauma.

29. DIC also produces thrombosis, which can cut off the blood supply or return from arteries or veins, producing ischemia. If the thrombosis occurs in the arteries or veins that supply or drain the brain, this can cause a hypoxic ischemic brain.

30. The most likely progression in this case is an ischemic bowel progressing to peritonitis and pancreatitis. The final stage (severe pancreatitis and a hypoxic ischemic brain) is likely what brought him to the hospital. Before that, he may have been only mildly symptomatic (lethargy, cold symptoms, refusal of food, etc.).

31. Ischemic bowel, *i.e.,* decreased flow of blood to the bowel, can result from many different causes. In my experience, ischemic bowels are most often natural in origin. In children, one relatively common cause is intussusception, a process in which one portion of the bowel twists into another portion, cutting off circulation. The most common symptoms are abdominal pain and vomiting. Since intussusceptions can resolve spontaneously, they may not be present at autopsy. *See, e.g.,* LE Swischuk et al, *Spontaneous reduction of intussusception: verification with US,* Radiology 1994; 192(1):269-271 (spontaneous reduction of intussusception probably more common than generally thought); Mehran Peyvasteh et al, *Intussuception at Atypical Ages in Children and Adults – 11 Years Experience,* Przeglad Chirurgiczny 2011; 83(6):304-309 (predominance of males in 2-10 yr age groups; 36% of males had history of common cold or gastroenteritis, 84% had abdominal pain, 64% had vomiting; less frequent complaints included restlessness, diarrhea and anorexia, with clinical manifestations of 3 days or more in 52% of cases; the etiology was unknown in 38% of all cases in 2-10 year age group).

32. Other causes of ischemic bowel include impacted food or thrombosis, *i.e.,* formation of a blood clot in the blood vessels supplying blood to the bowel. Pneumonia has also been implicated as a possible cause, suggesting that an ischemic bowel may result from a reduced oxygen supply from any source. *See, e.g.,* Rashmi Kabre, *Bowel Ischemia Following Pneumonia in Compromised Children,* Clin Pediatr 2008; 47(6):598-601. If a child has been kept on life support for some days before autopsy, it may be difficult or impossible to determine the source of the ischemic bowel since intussusceptions, impacted food and/or thrombosed veins or arteries may have been present and then resolved.

33. An ischemic bowel may also be caused by trauma (accidental or abusive). This is more likely when the child presents with a history of trauma, abrasions, patterned injuries or the like. In this case, most if not all of the marks and lines on the child's body appeared after hospital

admission and likely reflect DIC with standard handling of the patient rather than trauma. The small bowel perforation may have occurred during hospitalization as the walls of the bowel continued to break down. If the process began with trauma, I would look for a traumatic event occurring at least a day before hospital admission and possibly longer.

34. The best way to determine when the bowel became ischemic and the order in which these events occurred is to examine the pathology (microscopic slides). Since the process continues after hospitalization until the child is taken off life support, it is important to look for the earliest findings on pathology as these will provide the best indications of when the process began. The slides may also give some idea of relative timing.

35. Irrespective of the pathology, I would not expect this entire process (ischemic bowel, peritonitis, pancreatitis, liver inflammation and DIC) to occur within approximately eight hours of hospitalization. Instead, I would expect the process to evolve over a period of days.

Conclusion

36. The laboratory reports confirm pancreatitis and DIC. When viewed in the context of the clinical history and autopsy findings, it is likely that the process began with a mildly symptomatic ischemic bowel, leading to peritonitis, pancreatitis and a coagulopathy. The small perforation was likely a side effect of the ischemic bowel and may have occurred during the hospitalization.

37. It is unlikely that this process began within 8 hours or so of hospital admission. To more precisely determine when it began, it is important to look at the earliest findings on pathology. From a clinical perspective, since pancreatic damage was already present on hospital arrival, the process likely began at least a day before hospital admission and possibly earlier.

I swear under penalty of perjury that the foregoing is true and correct.

Affiant by Atty Ercole
Michael Laposata, M.D., Ph.D.

Date: 4/4/12

(Approved by email)
(attached)

Escuder, Jaime

From: hkirkwood2@comcast.net
Sent: Wednesday, April 04, 2012 11:54 PM
To: Escuder, Jaime
Subject: Fwd: Affidavit
Attachments: Laposata declaration kirkwood march 30 20012.docx; Laposata CV.doc; Children With Signs of Abuse Article.pdf

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From: "Ondrea Simmons" <ondrea.simmons@Vanderbilt.Edu>
To: "Heather Kirkwood" <hkirkwood2@comcast.net>
Sent: Wednesday, April 4, 2012 2:01:25 PM
Subject: FW: Affidavit

Hi Heather,
Attached are the documents you requested. I am not sure the attached article is the one you were speaking of but take a look at it.
Have a great afternoon,
Ondrea

From: hkirkwood2@comcast.net [mailto:hkirkwood2@comcast.net]
Sent: Monday, April 02, 2012 6:29 PM
To: Simmons, Ondrea
Subject: Re: Affidavit

Here it is. Many thanks. H.

From: "Ondrea Simmons" <ondrea.simmons@Vanderbilt.Edu>
To: "hkirkwood2@comcast.net" <hkirkwood2@comcast.net>
Sent: Monday, April 2, 2012 4:23:32 PM
Subject: RE: Affidavit

After speaking with Dr. Laposata today, I don't think he made any changes on the affidavit so could you email me a copy of what you sent him and I will try to get it notarized, then scan it and send it back to you.
Thanks,
Ondrea

From: hkirkwood2@comcast.net [mailto:hkirkwood2@comcast.net]
Sent: Monday, April 02, 2012 11:11 AM
To: Simmons, Ondrea
Subject: Re: Affidavit

Could you arrange for him to get it notarized and scanned Thurs AM?? Thanks, H.

From: "Ondrea Simmons" <ondrea.simmons@Vanderbilt.Edu>
To: "hkirkwood2@comcast.net" <hkirkwood2@comcast.net>
Sent: Monday, April 2, 2012 8:59:07 AM
Subject: RE: Affidavit

I am not sure what I am expected to do. I do not have a copy of the affidavit - Dr. Laposata did not forward it to me in the email and he is will not be back in the office until Thursday morning.

Ondrea

From: hkirkwood2@comcast.net [mailto:hkirkwood2@comcast.net]
Sent: Monday, April 02, 2012 6:37 AM
To: Laposata, Michael
Cc: Simmons, Ondrea
Subject: Re: Affidavit

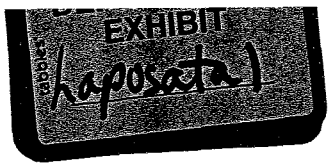
We need a notarized copy to file Thursday . . . Ondrea, ideas? Can scan and send. Many thanks. H.

From: "Michael Laposata" <michael.laposata@Vanderbilt.Edu>
To: hkirkwood2@comcast.net
Cc: "Ondrea Simmons" <ondrea.simmons@Vanderbilt.Edu>
Sent: Sunday, April 1, 2012 10:48:36 PM
Subject: RE: Affidavit

This looks fine to me Heather - I am out of town till Wed late

How do we do it from here? Ondrea is copied if we need her help

Mike



UPDATED: 3/12/12

CURRICULUM VITAE

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4605-A The Vanderbilt Clinic
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Vanderbilt University Medical Center
Nashville, TN 37232

Telephone: (615) 322-7862
Fax: (615) 343-8976
E-Mail: Michael.laposata@vanderbilt.edu

PERSONAL HISTORY

Date of Birth:	April 22, 1952
Place of Birth:	Johnstown, Pennsylvania
Citizenship:	United States

EDUCATIONAL BACKGROUND

Undergraduate Education:	Bachelor of Science in Biology, 1974 Bucknell University, Lewisburg, PA
Medical & Graduate Education:	M.D. - Ph.D. Program in Biochemistry, Cellular, and Molecular Biology, Johns Hopkins University School of Medicine 1981 M.D. 1982 Ph.D.

PROFESSIONAL TRAINING

July, 1981- June, 1983	Postdoctoral Research Fellow Division of Hematology-Oncology Department of Medicine Washington University School of Medicine, St. Louis, MO

July, 1983- June, 1985	Resident Division of Laboratory Medicine Departments of Pathology and Medicine Washington University School of Medicine, St. Louis, MO
July, 1984- June, 1985	Chief Resident Division of Laboratory Medicine Departments of Pathology and Medicine Washington University School of Medicine, St. Louis, MO

LICENSURE — Tennessee License, number MD0000043161 (active)

BOARD STATUS — Diplomate, American Board of Pathology,
Clinical Pathology - 1989

HOSPITAL APPOINTMENTS

July, 1985- June, 1986	Assistant Director of the Hemostasis Laboratory, Hospital of the University of Pennsylvania, Philadelphia, PA
July, 1986- July, 1989	Co-Director of the Hemostasis Laboratory, Hospital of the University of Pennsylvania, Philadelphia, PA
August, 1989- October, 2006	Director of Clinical Laboratories and Chief of the Division of Laboratory Medicine, Massachusetts General Hospital, Boston, MA (For the full period of its existence)
October, 2006- January, 2008	Staff pathologist and physician, Massachusetts General Hospital
August, 1989- June, 1998	Joint Appointment at the Massachusetts General Hospital, Boston, MA: Associate Pathologist, Department of Pathology & Associate Physician (Laboratory Medicine), Department of Medicine
June, 1998- January, 2008	Pathologist, Department of Pathology & Physician (Laboratory Medicine), Department of Medicine
1992-2008	Director of the Coolidge Clinical Laboratory of Massachusetts Eye and Ear Infirmary, Boston, MA

2000-2008	Consulting Staff Physician, Spaulding Rehabilitation Hospital, Boston, MA
2006-2007	Associate Director of Residency Training for Laboratory Medicine
2006-2007	Director of the Clinical Laboratory, Shriners Burn Hospital, Boston, MA
2006-2008	Director of the Clinical Laboratory, Spaulding Rehabilitation Hospital, Boston, MA
2008 – Present	Pathologist-in-Chief, Chief of Clinical Services and Division of Laboratory Medicine, and Director of Clinical Laboratories, Vanderbilt University Hospital

ACADEMIC APPOINTMENTS

July 1985 - July 1989	Assistant Professor of Pathology and Laboratory Medicine, University of Pennsylvania School of Medicine
August 1989 - June 1999	Associate Professor of Pathology, Harvard Medical School
June 1999 - January 2008	Professor of Pathology, Harvard Medical School
2002- 2008	Scholar, Harvard Academy of Scholars Harvard Medical School Faculty Selected on the Basis of Their Dedication to Medical Student Education
2008 – Present	Professor of Pathology and Professor of Medicine, Executive Vice Chair, Department of Pathology, Microbiology and Immunology, Vanderbilt University School of Medicine
2009 - Present	Vanderbilt University School of Medicine Academy of Educators

GRANT SUPPORT

Principal Research Interests:

1999 - Current	- Fatty acid alterations in cystic fibrosis
1994 - 2005	- Fatty acid ethyl esters, nonoxidative ethanol metabolites
1989 - 1993	- Fatty acidacylation of platelet proteins
1985 - 1994	- Fatty acids and fatty acid metabolites in cell activation

1986 - 1995	R01 DK37454 National Institutes of Health Control of Icosanoid Precursor Fatty Acid Metabolism
1987-1988	Sheryl N. Hirsch Award of the Lupus Foundation of Philadelphia Direct Costs: $10,000
1990 - 1993	R01 DK43159 National Institutes of Health Extracellular Fatty Acid Supply and Icosanoid Production
1994 - 2008	Funding from clinical revenue at Massachusetts General Hospital of basic research program Annual Direct Costs: $70,000 – 111,000
2006	Cystic Fibrosis Foundation Determining the Role of Fatty Acids and Diet in Cystic Fibrosis Co-Principal Investigator Co-PI Annual Direct Costs: $250,000
2008	Center for Minimally Invasive Therapy "Lab-In-A-Box" Project Development Principal Investigator $28,000 (Declined because of move to Vanderbilt)
2008 - 2011	Funding from Vanderbilt University School of Medicine of basic research program Annual Direct Costs: $120,000
2010 -	Funding from Endowed Chair directed to research program Annual Direct Costs: $80,000
2011-2013	Project Title: Pharmacogenomic Resource for Enhanced Decisions in Clopidogrel Treatment (PREDICT) Principal Investigator: Michael Laposata Grant Number: 1U47CI000824-01 Project Period: 09/30/2011 – 09/29/2013 Total Direct Costs: $499,013 annually for 2 years

GRANT REVIEW APPOINTMENTS

1. Special Reviewer. Biochemistry 2 Study Section.
   National Institutes of Health. July, 1987

2. Ad Hoc Reviewer. Molecular Biosciences Section.
   National Science Foundation. 1988-1989

3. Special Reviewer. Biochemistry 2 Study Section.
   National Institutes of Health. October, 1990

4. Special Review Committee Member for the National Institutes of Health
   to Evaluate Program Project on Alcohol Metabolism. July, 1996

5. Special Reviewer. Alcohol & Toxicology I Study Section.
   National Institutes of Health. October, 1996

6. Alcohol & Toxicology Clinical Sciences Special Emphasis Panel.
   National Institutes of Health. November, 1996

7. Special Reviewer. Alcohol & Toxicology 1 Study Section.
   National Institutes of Health. October, 1997

8. Special Reviewer. Alcohol & Toxicology 4 Study Section.
   National Institutes of Health. October, 1997

9. Special Reviewer. Alcohol & Toxicology 1 Study Section.
   National Institutes of Health. February, 1999

10. Ad hoc Reviewer. Special Emphasis Panel. Small Business Innovative
    Research Grant Study Section. National Institutes of Health. June, 2003.

CLINICAL RESPONSIBILITIES

Interpretation for the patient's medical record
of >500 Coagulation Test Batteries with Complete Medical
Consultation on 10-15 Coagulation Cases Monthly

HOSPITAL AND MEDICAL SCHOOL COMMITTEES

Chairman, Interdepartmental Clinical Laboratory Advisory Committee
Massachusetts General Hospital
2002 - 2007

Innovative Diagnostics and Therapeutics Committee
Massachusetts General Hospital
2002 – 2004

Committee for the selection of fellows for the Harvard Academy of Scholars
2003 – 2007

Steering Committee for the Clinical Skills Assessment Examination (OSCE) Harvard Medical School
1998 - 2002

Pathology Executive Committee
Massachusetts General Hospital (for the full period of its existence)
2000 – 2006

Anticoagulation Advisory Committee
Massachusetts General Hospital
2000 - 2002

Pathology Resident Selection Committee
Massachusetts General Hospital
2004 – 2007

Pathology Department Professors Committee to evaluate faculty for promotion
Massachusetts General Hospital
1999 – 2007

Housestaff Committee
Department of Pathology, Vanderbilt University School of Medicine
2008 – Present

Executive Committee
Department of Pathology, Vanderbilt School of Medicine
2008 – Present

Chair, Laboratory Services Committee, Vanderbilt University Hospital
2008 – Present

Appointments & Promotions Committee, Department of Pathology, Vanderbilt University School of Medicine
2008 – Present

Emphasis Program, Vanderbilt University School of Medicine, Co-Leader of Section on Laboratory Based Research
2009 – Present

Chair, Pharmacy and Therapeutics Subcommittee on Pharmacogenomics
2010 - Present

EDITORIAL BOARD APPOINTMENTS

American Journal of Clinical Pathology
Associate Editor

Il Pathology Clinica: Journal of Molecular and Clinical Pathology
Scientific Committee

PROFESSIONAL ORGANIZATIONS

College of American Pathologists, 1992-
Coagulation Resource Committee, 1995 –2001
Vice-Chair, 1999-2001
Massachusetts Society of Pathologists, 1993-
President 2001-2003
Academy of Clinical Laboratory
Physicians and Scientists, 1986-
Executive Council, 1993-1999
President, 1997-1998
Organizer and Host of the 1998
National Meeting in Boston, MA

American Society of Clinical Pathologists, 1990-
Chairman, Council on Resident Education 1995-1999

Mid-Atlantic Lipid Research Annual Symposium,
Program Committee, 1990-2000

International Society for the Study of
Fatty Acids and Lipids, 1993-
Organizer and Host of the Fourth
International Roundtable Conference
on Fatty Acids in Cell Signaling
in Chatham, MA in 1998

Research Society on Alcoholism, 1996-2009

American Board of Pathology
Clinical Pathology Test Committee, 1996-2002

American Society of Hematology, 2002-
Laboratory Hematology Committee, 2002-2004

International Society on Thrombosis & Hemostasis, 2002-
Co-Chairman, Subcommittee on Control of Anticoagulation Therapy,
2003-2008

American Association for Clinical Chemistry, 2002-
Evidence-Based Medicine Planning Group, 2003
Task Force on Laboratory Consulting, 2004

ADVISORY BOARDS

ChilDx – Children's Health Improvement through Laboratory Diagnostics, 2002 –

Board of Scientific Counselors for review of the Department of Laboratory Medicine
National Institutes of Health, Bethesda, MD
2005

Clinical Laboratory Improvement Advisory Committee (CLIAC) of the Centers for Disease Control and Prevention (CDC)
(Appointment is made by the U.S. President's Secretary of Health and Human Services), 2002 – 2006

Instrumentation Laboratory, International Scientific Advisory Board for Coagulation, 2004 -

National Advisory Board, BD Preanalytical Systems, 2006-

Co-Chair, Clinical Laboratory Integration into Healthcare Collaborative (CLIHC) sponsored by Centers for Disease Control and Prevention (CDC) on Integration of Laboratory Medicine into Clinical Practice, 2008-

INVITED LECTURES

Lipid Biochemistry

The American Red Cross, St. Louis, MO.
December, 1983. "A Coenzyme A Synthetase Enzyme Specific for Icosanoid Precursor Fatty Acids."

The Merck Institute for Therapeutic Research, Rahway, NJ. August, 1985. "Mechanisms for Icosanoid Precursor Uptake and Release by a Tissue Culture Cell Line."

The Lipid Club of Philadelphia, Philadelphia, PA. May, 1986. "Control of Icosanoid Production at the Level of Arachidonoyl-CoA Synthesis."

The Tissue Culture Association, Philadelphia, PA. May, 1986. "The Reproducible Delipidation of Serum for Use in Tissue Culture."

Academy of Clinical Laboratory Physicians and Scientists National Meeting, Philadelphia, PA. June, 1987. "Control of Prostaglandin Production by Fatty Acid Supply."

Washington University School of Medicine, St. Louis, MO. July, 1987. "Control of Prostaglandin Production by Restriction of Exogenous Arachidonate."

Case Western Reserve University School of Medicine, Cleveland, OH. January, 1989. "The Availability of Cellular Arachidonate for Prostaglandin Production."

Washington University School of Medicine, St. Louis, MO. February, 1989. "Fatty Acid Acylation of Glycoprotein Ib and Glycoprotein IX in Human Platelets."

Massachusetts General Hospital, Boston, MA. March, 1989. "The Availability of Cellular Arachidonate for Prostaglandin Production."

Thomas Jefferson University School of Medicine, Philadelphia, PA. April, 1989. "Covalent Modification of Platelet Proteins by Palmitate."

Thrombosis Institute, Temple University School of Medicine, Philadelphia, PA. May, 1989. "Covalent Modification of Platelet Proteins by Palmitate."

University of Alabama, Birmingham, AL, August, 1990. "The Availability of Cellular Arachidonate for Prostaglandin Production."

Philadelphia College of Pharmacy and Science, Philadelphia, PA. November, 1990. "Icosanoid Precursor Fatty Acid Metabolism."

University Hospital of Debrecen, Hungary. September, 1991. "Mechanisms of Fatty Acid Transport to Cells."

Northeastern University, Boston, MA. March 1992. "Fatty Acids: Basic Biochemistry to Clinical Significance."

T Cell Sciences, Cambridge, MA. July, 1992. "Fatty Acid Ethyl Esters: A New Biochemical Marker for Alcoholism."

New England Lipid Group, Boston, MA. September, 1992. "Fatty Acid Ethyl Ester Metabolism."

ASCP/CAP National Meeting, Multiple Sites. 1992-1995 (annually). "Lipids: Biochemistry to Clinical Significance." Half-Day Workshop.

Basic Science Seminar Series, Tufts University School of Medicine, Boston, MA. January, 1993. "Fatty Acid Modification of Proteins."

Johns Hopkins University School of Medicine, Baltimore, MD. July, 1993. "Fatty Acid Ethyl Esters: A Marker for Ethanol Ingestion."

Jefferson Medical College, Philadelphia, PA. May, 1994. "Fatty Acid Ethyl Esters: Biochemistry to Clinical Significance."

Mid-Atlantic Lipid Research Symposium, Atlantic City, NJ. May, 1994. "The Covalent Binding of Fatty Acids to Proteins: A Metabolic Pathway of Increasing Complexity."

Second International Round Table Conference on Fatty Acids in Cell Signaling (35 scientists by invitation only), Madison, WI. June, 1994. "Fatty Acid Ethyl Esters: Toxic Ethanol Metabolites."

New York Lipid Research Club, New York City, NY. September, 1994. "The Covalent Modification of Platelet Proteins by Fatty Acids."

Institute for Scientific Applications, Lyon, France. November, 1994. "Fatty Acid Acylation of Platelet Proteins" and "Fatty Acid Ethyl Esters."

Yale University School of Medicine, New Haven, CT. December, 1994. "Fatty Acid Ethyl Esters: Toxic Metabolites of Ethanol."

Department of Pathology, Visiting Professor, Albany Medical College, Albany, NY. January 1995. "Fatty Acid Ethyl Esters: New Markers for Ethanol Ingestion."

International Society for the Study of Fatty Acids & Lipids (ISSFAL), Bethesda, MD. June, 1995. "Thioesterification of Platelet Proteins with Saturated and Polyunsaturated Fatty Acids."

Baystate Medical Center (Affiliate of Tufts University School of Medicine), Springfield, MA. September, 1995. "Fatty Acids: Biochemistry to Clinical Significance."

Division of Laboratory Medicine, Visiting Professor, Washington University School of Medicine, St. Louis, MO. April, 1996. "Fatty Acid Ethyl Ester: A Toxic Nonoxidative Metabolic of Ethanol."

Third International Round Table Conference on Fatty Acids in Cell Signaling (35 scientists by invitation only), Maastricht, The Netherlands. July, 1996. "Fatty Acids Covalently Bound to Platelet Proteins in the Native State."

Department of Gastroenterology, Warsaw Medical Academy, Warsaw, Poland. June, 1996. "Fatty Acid Ethyl Esters: Toxic Nonoxidative Metabolites of Ethanol."

Medical University of Debrecen, Hungary. July, 1996. "Fatty Acid Ethyl Esters: Toxic Nonoxidative Ethanol Metabolites."

20th Annual Arnold O. Beckman Conference. New Orleans, LA. February, 1997. "Alcoholic Liver Disease: Short Term and Long Term Markers of Ethanol Intake."

Biochemistry Colloquium Series. University of Massachusetts, Lowdl, MA. March, 1997. "Fatty Acid Ethyl Esters: New Markers for Ethanol Intake."

Biochemistry Seminar Series, Chiron Diagnostics, Walpole, MA. March, 1997.
"Serum Fatty Acid Ethyl Esters: A New Indicator of Ethanol Intake."

Neurology Grand Rounds, Massachusetts General Hospital, Boston, MA. May, 1997. "Fatty Acid Ethyl Ester: A New Ethanol Metabolite Associated with Toxicity and Intoxication."

Anesthesia Grand Rounds, Massachusetts General Hospital, Boston, MA. July, 1997. "Alcohol Abuse: Mechanisms of Toxicity and Monitoring Intake."

Breakthroughs in Pathology, National ASCP/CAP meeting, Philadelphia, PA. September, 1997. "New Markers for Monitoring Ethanol Intake."

Northeastern Chapter, American Association of Clinical Chemistry meeting, Waltham, MA. November, 1997. "Chemical Diagnosis of Alcoholism."

Department of Biophysics Seminar Series, Boston University School of Medicine, Boston, MA. January, 1998. "Fatty Acid Ethyl Esters: A Missing Link in Ethanol Induced Organ Damage."

University of Pennsylvania School of Medicine, Philadelphia, PA. February, 1998. "Ethanol Metabolism & Innovations in Laboratory Medicine."

University of Florida School of Medicine, Gainesville, FL. February, 1998. "Ethanol Metabolism/Innovative Approaches to Diagnostic Testing."

Ball Memorial Hospital, Visiting Professor, Muncie, IN. March, 1998. "Fatty Acid Ethyl Esters: A New Marker for Ethanol Intake."

Scientific Group on Obesity, Boston University School of Medicine, Boston, MA. March 1998 "Fatty Acid Ethyl Esters: Nonoxidative Ethanol Metabolites."

University of North Carolina School of Medicine, Chapel Hill, NC. April, 1998. "Ethanol Metabolism/Innovative Approaches to Diagnostic Testing."

Department of Pathology and Laboratory Medicine, University of Arizona School of the Health Sciences, Visiting Professor, Tucson, AZ. April, 1998. "Fatty Acid Ethyl Ester: A Toxic Ethanol Metabolite."

International Society for the Study of Fatty Acids and Lipids (ISSFAL). Lyon, France. June,1998. "Covalent Modification of Proteins by Fatty Acids."

International Society for the Study of Fatty Acids and Lipids (ISSFAL). Lyon, France. June, 1998. "Fatty Acid Ethyl Esters: Nonoxidative Metabolites of Ethanol."

Fourth International Roundtable on Fatty Acids in Cell Signaling, (30 scientists by invitation only) Chatham, MA. June 1998. "Fatty Acid Ethyl Esters: Ethanol Metabolites with a Role in Organ Damage and Ethanol Monitoring."

The Cleveland Clinic, Visiting Professor, Cleveland, OH. January, 1999. "Fatty Acid Ethyl Esters in the Blood: A New Test for Ethanol Intake."

The Food & Drug Administration, Tissue Engineering Course, Rockville, MD. February, 2000. "Inflammation and Implantable Devices."

Vascular Biology Research Group, Tufts University School of Medicine, Boston, MA. May, 2000. "Fatty Acid Ethyl Esters: Nonoxidative Ethanol Metabolites."

Grand Rounds, Pathology Department, Massachusetts General Hospital, Boston, MA. January, 2001. "Fatty Acid Ethyl Esters: Nonoxidative Ethanol Metabolites for Monitoring Ethanol Intake."

24th Annual Meeting of the Research Society on Alcoholism, Montreal, Canada. June, 2001. "Role of Fatty Acid Ethyl Esters in Alcohol Induced Organ Damage."

Cellular & Molecular Aspects of Omega-3 Fatty Acids and Cancer Symposium, Breckenridge, CO. June, 2001. "Fatty Acid Metabolism in Chronic Alcoholism, an Induction State for Hepatocellular Carcinoma."

The Fifth International Conference on Fatty Acids and Cell Signaling, Gargano, Italy. September, 2001. "Fatty Acid Ethyl Esters: Their Role in Ethanol– Induced Cell Injury and Monitoring Ethanol Intake."

The MGH Child Psychiatry Section, Boston, MA. March, 2002. "Omega-3 fatty acids and attention deficit disorders."

The 4th Annual Frank M. Townsend Lecture, University of Texas at San Antonio, San Antonio, TX. November, 2002. "Fatty acid ethyl esters: toxic nonoxidative ethanol metabolites."

Gastroenterology Research Seminar, University of Pennsylvania School of Medicine, Philadelphia, PA. January, 2003. "Fatty acid ethyl esters."

Pathology Grand Rounds, University of Pennsylvania School of Medicine, Philadelphia, PA. January, 2003. "Fatty acid ethyl ester: A toxic nonoxidative ethanol metabolite."

Department of Pathology, Louisiana State University School of Medicine, New Orleans, LA. February, 2003. "Fatty acid ethyl esters: How much ethanol is good or bad?"

Dana Farber Cancer Institute, Boston, MA. April, 2003. "An overview of fatty acid metabolism and its implications for anti-cancer treatment."

Conemaugh Valley Memorial Hospital, Johnstown, PA. May, 2003. "Ethanol: The good and the bad."

Current Concepts in Clinical Pathology, Boston, MA. June, 2003. "Lipid Testing and the Prediction of Cardiovascular Risk."

Memorial Sloane Kettering Cancer Center, New York City, NY. July, 2003. "Fatty Acid Ethyl Esters: Toxic Nonoxidative Ethanol Metabolites."

The 6th International Conference on Fatty Acids and Cell Signaling. Bethesda, MD. July, 2003. "Fatty Acid Ethyl Esters in Red Blood Cells and Platelets."

Alcohol Birth through Death Conference sponsored by the California Association of Toxicologists. Santa Rosa, Ca. August, 2003. "Fatty Acid Ethyl Esters as Postmortem Markers of Premortem Ethanol Intake."

Ospedale Pediatrico Bambina Gesu, Rome, Italy. December 2003. "Fatty Acid Alterations in Cystic Fibrosis."

Massachusetts General Hospital, Pathology Grand Rounds, Boston, MA. February, 2003. "Fatty Acid Alterations in Cystic Fibrosis."

Basic Science Lecture Series, University of Texas Medical Branch, Galveston, TX. March, 2004. "Fatty Acid Ethyl Esters: Nonoxidative Metabolites of Ethanol."

Department of Pathology Visiting Professor Lecture Series, University of Alabama at Birmingham, Birmingham, AL. March, 2004. "Fatty Acid Ethyl Esters: Toxic Nonoxidative Ethanol Metabolites."

17th Annual Research Conference of the Cystic Fibrosis Foundation, Williamsburg, VA. June, 2004. "Fatty acid metabolism in cystic fibrosis: What have we learned in 20 years and where do we go from here?"

University of Rome, Study Group on Liver Disease and Alcoholism, Rome, Italy. June, 2004. "Fatty Acid Ethyl Esters: Toxic Nonoxidative Metabolites of Ethanol."

Tufts University School of Medicine, MD-PhD Research Seminar Series, Boston, MA. January, 2005. "Fatty Acid Ethyl Esters."

Scientific Symposium, Hilton Head, SC. June, 2005. "Alcohol: The Good and The Bad."

Scientific Symposium, Hilton Head, SC. June, 2005. "Evaluation of Cardiovascular Risk."

12th Annual Massachusetts Prosecutors Conference, Boston, MA. March, 2006. "Operating Under the Influence"

Research Conference on Autism, Boston, MA. October, 2006. "Fatty Acid Metabolism– Is It Relevant To Autism?"

Case Western Reserve University Medical Center, Department of Pathology, Visiting Professor, Cleveland, OH. January, 2007. "Fatty Acid Alterations in Cystic Fibrosis– Moving beyond the Original Biochemical Observations and Understanding the Basis of the DHA Therapeutic Effect."

Temple University School of Medicine, Department of Pathology. Philadelphia, PA. April, 2007. "Cystic Fibrosis: Fatty Acid Alterations and Fatty Acid Supplementation."

Vanderbilt University School of Medicine, Department of Pathology. Nashville, TN. April, 2007. "Fatty Acid Alterations in Cystic Fibrosis: A Target for Therapy."

Fatty Acids in Cell Signaling– 8th Symposium, Quebec City, Canada. June, 2007. "Fatty Acid Alterations in Cystic Fibrosis."

21st Annual North American Cystic Fibrosis Conference, Anaheim, CA. October, 2007. "A Unifying Theory to Explain How CFTR Leads to the Lipid Abnormalities in CF and Altered Inflammatory Signaling" and "Introduction to Fatty Acid Metabolism"

Gastroenterology Research Conference, Beth Israel Deaconess Medical Center, Boston, MA. November, 2007. "Cystic Fibrosis: Fatty acid alterations and fatty acid supplementation."

Loyola University School of Medicine, Pathology Grand Rounds, Chicago, IL. January, 2008. "Cystic Fibrosis".

22nd Annual North American Cystic Fibrosis Conference, Orlando, FL. October, 2008. In the session on Fatty Acids: Crossroads of Nutrition and Science, "Monitoring Adequacy of Omega-3 Fatty Acid Intake."

Clinical Pharmacology Grand Rounds, Vanderbilt University Medical Center, Nashville, TN. February, 2010. "Fatty Acid Abnormalities in Cystic Fibrosis."

Visiting Professor, University of Illinois at Champaign-Urbana, Champaign, IL. November, 2010. "Fatty Acids: Metabolism and Clinical Significance."

Visiting Professor, McGill University, Montreal, Canada. November, 2011. "Fatty Acid Abnormalities in Cystic Fibrosis."

Clinical Coagulation

The American Red Cross, Philadelphia, PA. April, 1986. "The Diagnosis and Treatment of Coagulation Factor Deficiencies."

Internal Medicine Board Review Course, Hospital of the University of Pennsylvania, Philadelphia, PA. April, 1987-1989 (annually). "Diagnosis and Treatment of Coagulation Disorders."

Academy of Clinical Laboratory Physicians and Scientists National Meeting, Philadelphia, PA. June, 1987. "Monitoring Anticoagulant Therapy."

Hospital of the University of Pennsylvania, Philadelphia, PA. October, 1987. Critical Care Medicine Symposium "Coagulation in the ICU."

Philadelphia College of Osteopathic Medicine, Philadelphia, PA. March, 1988. "Aspirin Ingestion and Myocardial Infarction."

Blood Bank Association of New York State Annual Meeting, Syracuse, NY. June, 1988. "Secondary Hemostasis."

General Surgery Continuing Education Course, Harvard Medical School, Boston, MA. September/October, 1990-1995 (annually). "Preoperative and Postoperative Evaluation of Hemostasis."

American Society of Hematology/International Society of Hematology, Boston, MA. December, 1990. "Clinical Utility of Platelet Aggregometry"

Massachusetts Association of Blood Banks, Waltham, MA. May, 1991. "Acquired Hemostatic Defects".

Ortho Pharmaceutical Corporation, Raritan, NJ. August, 1991. "Clinical Coagulation Test Batteries".

Instrumentation Laboratories, Lexington, MA. December, 1991. "Clinical Coagulation Test Batteries."

ASCP/CAP National Meeting, Las Vegas, NV. October 1992. "Platelet Aggregation Studies."

Coagulation Resource Committee of the College of American Pathologists, Lake Buena Vista, FL. January, 1993. "Bedside Testing in Coagulation."

Anesthesia Review and Update Continuing Education Course, Harvard Medical School, Boston, MA. 1994-Present (annually). "Why Doesn't the Blood Clot?"

Intensive Care Medicine Continuing Education Course, Harvard Medical School, Boston, MA. 1994-Present (annually). "Diagnosis and Management of Coagulation Disorders."

Annual Review for Dental Practitioners - Continuing Education Course, Harvard Medical School, April, 1994-1996 (annually). "Coagulopathies."

Grand Rounds, MGH Department of Medicine, Boston, MA. July, 1994. "von Willebrand's Disease."

Cardiovascular Anesthesia Continuing Education Course, Harvard Medical School, Boston, MA. September, 1994. "Diagnosis and Treatment of Common Coagulopathies."

Department of Pathology, Visiting Professor, Emory University School of Medicine, Atlanta, GA. October, 1994. "Hypercoagulable States."

Management Decisions for the General Surgeon Continuing Education Course, Harvard Medical School, Boston, MA. October, 1994. "Necessity for Blood Transfusion Therapy" and September, 1995 "The Diagnosis of Hypercoagulable States."

Department of Gastroenterology, Warsaw Medical Academy, Warsaw, Poland. June, 1996. "Hypercoagulable States."

Scientific Symposium on Anticoagulation Monitoring, Dedham, MA. June, 1996. "The Therapeutic Use and Clinical Laboratory Monitoring of Low Molecular Weight Heparin."

Grand Rounds, MGH Department of Medicine, Boston, MA. December, 1996. "Hypercoagulable States."

Department of Anesthesia, Beth Israel Hospital, Boston, MA. January, 1997. "The Diagnosis of Commonly Encountered Coagulopathies."

Youville Rehabilitation Hospital, Cambridge, MA. February, 1997. "Clinical Use and Monitoring of Low Molecular Weight Heparin."

American Association for Clinical Chemistry/Clinical Ligand Society, Boston, MA. May, 1997. "Cardiology & the Clinical Laboratory: Thrombosis and Hemostasis in Myocardial Infarction."

Penobscot Bay Medical Center, Rockland, ME. October, 1997. "Coagulation Problems in the Community Hospital."

College of American Pathologists Concensus Conference on Anticoagulant Monitoring. Atlanta, GA. October, 1997. "Leader of Session to Formulate Recommendations for Monitoring Low MolecularWeight Heparin, Danaparoid, Hirudin, and Argatroban."

Braintree Rehabilitation Hospital, Braintree, MA. March, 1998. "The Clinical Indications for Use and Monitoring of Low Molecular Weight Heparin."

Massachusetts Society of Pathologists, Worcester, MA. March, 1998. "What the Pathologist Needs to Know about Coagulation Testing."

Department of Surgery Grand Rounds, St. Elizabeth's Medical Center, Boston, MA. September, 1998. "The Pharmacology of Low Molecular Weight Heparin."

Deaconess-Glover Hospital Grand Rounds, Needham, MA. December, 1998. "Commonly Encountered Problems in Coagulation."

Department of Pathology, The Cleveland Clinic, Visiting Professor, Cleveland, OH. January, 1999. "Interesting Coagulopathy Cases from the Massachusetts General Hospital."

Current Issues in Vascular and Endovasclar Surgery, Continuing Education Course, Harvard Medical School, Boston, MA. May, 1999 and May, 2000. "What the Vascular Surgeon Needs to Know about Coagulation Disorders and Anticoagulation Problems."

Medical Grand Rounds, Faulkner Hospital, Boston, MA. August, 1999. "The Clinical Indications and Laboratory Monitoring of Low Molecular Weight Heparin."

Rheumatology Grand Rounds, Massachusetts General Hospital, September, 1999. "Antiphospholipid Antibodies."

Grand Rounds, Harvard University Health Services, Cambridge, MA. September 1999. "The Diagnosis and Treatment of Hypercoagulable States."

Medical Grand Rounds, Healthsouth New England Rehabilitation Hospital, Woburn, MA. September, 1999. "The Clinical Indications and Laboratory Monitoring of Low Molecular Weight Heparin."

Vascular Surgery Grand Rounds, Massachusetts General Hospital, Boston, MA. September, 1999. "Thrombosis in the Patient with a Hypercoagulable State."

Department of Pathology, UCLA School of Medicine, Los Angeles, CA. October, 1999. "Hypercoagulable States: Diagnosis and Treatment."

Advances in Rheumatology, Continuing Education Course, Harvard Medical School, Boston, MA. October, 1999 and September, 2000. "Hypercoagulability Testing: Current Practice to Assess Thrombotic Risk."

Conference of Maine Physicians, Scarborough, ME. October, 1999. "Low Molecular Weight Heparin: When and How to Use it and Monitor its Effect."

Orthopedics Grand Rounds, Carney Hospital, Boston, MA. November, 1999. "Low Molecular Weight Heparin: an Alternative to Unfractionated Heparin."

The Kessler Rehabilitation Institute, W. Orange, N.J. November, 1999. "The Use and Monitoring of Low Molecular Weight Heparin."

Grand Rounds, Holy Family Hospital, Methuen, MA. November, 1999. "The Diagnosis and Treatment of Hypercoagulable States."

Department of Pathology, St. Louis University School of Medicine, St. Louis, MO. January, 2000. "The Hypercoagulable States."

Rheumatology Grand Rounds, Brigham and Women's Hospital, Boston, MA. February, 2000. "Diagnosis and Treatment of Hypercoagulable States."

7th Annual Progress in Clinical Pathology, Dallas, TX. March, 2000. "Diagnosis and Treatment of Hypercoagulability."

American Association for Clinical Chemistry, Connecticut Valley Section, Hartford, CT. March, 2000. "One of Five is Predisposed to Thrombosis - Are You One and Do You Want to Find Out?"

Twelfth Annual Lectures in Contemporary Hemostasis and Thrombosis, Miami, FL. March, 2000. "Diagnostic Mistakes in Coagulation with Catastrophic Outcomes."

Dade Behring Hemostasis Seminar Series, Boston, MA. April, 2000. "Hypercoagulable States: Diagnosis and Treatment."

Department of Anesthesia Grand Rounds, Tufts/New England Medical Center, Boston, MA. April, 2000. "Hypercoagulable States."

Symposium on Anticoagulation Management in the 21st Century, Brooklyn, NY. June, 2000. "Outpatient Management of Deep Vein Thrombosis Using Low Molecular Weight Heparin."

Role of the Laboratory in the Treatment of Patients, Mayo Symposium, Prouts Neck, ME. June, 2000. "The Diagnosis and Treatment of Hypercoagulable States."

The Wagih Bari Society of St. Louis Pathologists, St. Louis, MO. September, 2000. "Diagnostic Errors in Coagulation with Catastrophic Outcomes."

The North Shore Medical Center/Salem Hospital, Salem, MA. October, 2000. "Hypercoagulable States."

Brown University School of Medicine, Department of Surgery Grand Rounds, Providence, RI. November, 2000. "Everything You Wanted to Know about Low Molecular Weight Heparin."

Newport Hospital, Newport, RI. November, 2000. "New Markers for Hypercoagulable States."

Anna Jacques Medical Staff Grand Rounds, Newburyport, MA. January, 2001. "Hypercoagulable States."

Department of Anesthesia Grand Rounds, Massachusetts General Hospital, Boston, MA. January, 2001. "The Diagnosis and Treatment of Hypercoagulable States."

Distinguished Lecturer Series, Spaulding Rehabilitation Hospital, Boston, MA. January, 2001. "Identification and Treatment of Hypercoagulable States."

Mt. Sinai Medical Center, Miami, FL. February, 2001. "Genetic markers of hypercoagulability."

Rheumatology Grand Rounds, Massachusetts General Hospital, Boston, MA. March, 2001. "The Appropriate Use of Low Molecular Weight Heparin."

Pharmacia Symposium for Healthcare Practitioners, Pittsburgh, PA. March, 2001. "The Diagnosis and Treatment of Hypercoagulable States."

Mercy Hospital of Pittsburgh Medical Grand Rounds, Pittsburgh, PA. March, 2001. "Low Molecular Weight Heparin."

Rheumatology Grand Rounds, Roger Williams Hospital of Brown University School of Medicine, Providence, RI. March, 2001. "Hypercoagulability and the Antiphospholipid Syndrome."

The North Shore Medical Center/Salem Hospital, Salem, MA. April, 2001. "von Willebrand's Disease."

American Association of Clinical Chemistry, Review Course, Alexandria, VA. April, 2001. "An Overview of Coagulation."

Vascular Surgery Grand Rounds, Massachusetts General Hospital, April, 2001. "The New Anticoagulants."

Symposium on Expanding the Horizons of Antithrombotic Therapy, Staten Island, NY. May, 2001. "Diagnosis and Treatment of Hypercoagulable States."

Pulmonary Medicine Grand Rounds, SUNY Downstate Medical Center, Brooklyn,NY. May, 2001. "Heritable Causes of Venous Thrombosis."

General Medicine Grand Rounds, Massachusetts General Hospital, September, 2001. "Hypercoagulable States: Diagnosis, Treatment, and Controversies in Long Term Patient Management."

Transplantation Surgery Grand Rounds, Massachusetts General Hospital, September, 2001. "Antiphospholipid Antibody Syndrome."

Obstetrics & Gynecology Grand Rounds, Massachusetts General Hospital, September, 2001. "Thrombophilia: Obstetrical and Gynecologic Issues."

Grand Rounds, Memorial Sloane-Kettering Cancer Center, New York, NY. October, 2001. "The Diagnosis and Treatment of Hypercoagulable States."

Surgery Grands Rounds, Massachusetts General Hospital, October, 2001. "New Anticoagulants and Indications for Their Use."

Medical Grand Rounds, University of Massachusetts Memorial Medical Center, Worcester, MA. October, 2001. "Sepsis, Anticoagulation, and a Drug That Connects Them."

Lab Med 2001 Symposium, New York City, NY. October, 2001. "Genetic Markers of Hemostasis."

Massachusetts General Hospital Special Seminar, October, 2001. "Sepsis and Recombinant Activated Protein C."

Medical Grand Rounds, Allegheny General Hospital. December, 2001. "Indications & Monitoring of Low Molecular Weight Heparin."

New England Medical Center, December, 2001. "Inflammation, Coagulation, and a Drug that Connects the Two."

Merrimack Valley Hospital, Haverhill, MA. December, 2001. "Anticoagulation with Warfarin and Heparin – Basic Principles."

Surgery Grand Rounds, Newton Wellesley Hospital, Newton, MA. January, 2002. "DVT Prophylaxis and Treatment."

Audioconference for American Association for Clinical Chemistry. February, 2002. "Point of Care Testing for Coagulation."

Emma Sadler Moss Lectureship, Louisiana State University Medical Center, February, 2002. "Bleeding and Thrombotic Disorders."

Cardiac Surgery Grand Rounds, Massachusetts General Hospital, February, 2002. "A Review of Hypercoagulable States."

Roger Williams Hospital, Providence, RI. March, 2002. "Low Molecular Weight Heparin."

Palisades Medical Center, North Bergen, NJ. March, 2002. "A New Drug for Patients with Sepsis."

The Massachusetts Society of Pathologists Spring CME Program. Newton, MA. April, 2002. "The Diagnosis and Treatment of Hypercoagulable States."

19th Annual Hemostasis & Thrombosis Update, Philadelphia, PA. April, 2002. "The Diagnosis & Treatment of Hypercoagulable States."

The 16th Annual Northeast Region Meeting of the Clinical Laboratory Management Association, Clinical Ligand Society and American Association of Clinical Chemistry. Boxborough, MA. April, 2002. "The Diagnosis & Treatment of Hypercoagulable States."

Department of Pediatrics Cambridge Health Alliance and Mount Auburn Hospital, Cambridge, MA. May, 2002. "Hypercoagulable States."

Anesthesia Review & Update. Boston, MA. May, 2002. "Why Doesn't the Blood Clot?"

Lawrence Memorial Hospital, Visiting Clinician Program, Medford, MA, June, 2002. "The Hypercoagulable State."

Massachusetts General Hospital, Department of Neurology, June, 2002. "Hypercoagulable States."

Cambridge Hospital, Department of Surgery Grand Rounds, June, 2002. "Low Molecular Weight Heparin."

Exeter Hospital, Grand Rounds, Exeter, NH. June, 2002. "The Diagnosis and Treatment of Hypercoagulable States."

Pharmacia Sponsored Symposium, Portland, ME. June, 2002. "Low Molecular Weight Heparin: Mechanism of Action, Indications and Monitoring."

"Issues in the Treatment of Women" symposium of the American College of Obstetriciansand Gynecologists, Burlington, MA, July, 2002. The Johns Figgs Jewet Memorial Lectureship "Thrombophilia in Pregnancy."

Neurosurgery Grand Round, Massachusetts General Hospital, July, 2002. "Hypercoagulable States."

Greater New York Clinical Laboratory Management Association Program on the Early Diagnosis of Life Threatening Sepsis and DIC, New York City, NY, July, 2002. "Sepsis, Coagulation, and a Drug that Connects Them."

54th National Meeting of the American Association of Clinical Chemistry, Orlando, FL, July 2002. "Point of Care Coagulation Testing," "The Diagnosis of Bleeding Disorders," and "The Diagnosis of Thrombotic Disorders" (3 invited talks).

Dade Fall Conference 2002, Waltham, MA. September, 2002. "The Diagnosis & Treatment of Hypercoagulable States."

Hematology-Oncology Grand Rounds, New York Hospital/Cornell Medical College, New York, NY. September, 2002. "The Use of Low Molecular Weight Heparin."

Rhode Island Blood Bankers Society, Providence, RI, September, 2002. "Factor VIII Inhibitors: Diagnosis and Treatment."

Neuro-Oncology Grand Rounds, Massachusetts General Hospital, September, 2002. "The Use of Low Molecular Weight Heparin and Fondaparinux as Prophylaxis for Venous Thrombosis."

Podiatry Grand Rounds, Massachusetts General Hospital, September, 2002. "Deep Vein Thrombosis: Prophylaxis and Treatment."

Cancer Medicine and Hematology, Harvard Medical School Continuing Education Course, September, 2002. "Case Management Problems in Coagulation."

Surgery Grand Rounds, Mt. Auburn Hospital, Cambridge, MA. October, 2002. "Low Molecular Weight Heparin: Indications and Monitoring."

Birmingham Fall Hemostasis Symposium, Birmingham, AL. October, 2002. "Hypercoagulable States."

American Thrombosis Institute, Public Educational Symposium on Thrombosis, Mountain Brook, AL. October, 2002. "Blood Clots – Am I at Risk?"

University of Hawaii Pathology Residency Program, Honolulu, HI. October, 2002. "Hypercoagulable States: Diagnosis and Treatment."

North Shore Medical Center, Grand Rounds, Salem, MA. November, 2002. "The Diagnosis and Treatment of Hypercoagulable States."

Spaulding Rehabilitation Hospital, Boston, MA. November, 2002. "New Anticoagulants."
Medical Grand Rounds, Massachusetts General Hospital, December, 2002. "DVT Prophylaxis and Treatment in 1978: When I Was a Caveman Clotter."

St. Francis Hospital and Medical Center/University of Connecticut. Medical Grand Rounds, Hartford, CT. January, 2003. "Clinical and Laboratory Approach to Thrombophilia."

Connecticut Society of Pathologists, New Haven, CT. January, 2003. "Clinical and Laboratory Approach to Thrombotic Disorders."

Pathology Continuing Education Program, St. Francis Hospital, Hartford, CT. January, 2003. "Laboratory Testing for Thrombophilia."

Rheumatology Grand Rounds, Beth Israel Deaconess Hospital, Boston, MA. January, 2003. "Hypercoagulable States."

Neurology Grand Rounds, Lahey Clinic, Burlington, MA. January, 2003. "Diagnosis and Treatment of Hypercoagulable States."

2nd Year Louisiana State University Medical Student Lecture, New Orleans, LA. February, 2003. "A Review of Hemostasis."

Safe Transitions in Anticoagulation Therapy Symposium, Boston, MA. February, 2003. "Overview of Normal Hemostasis and Thrombosis.

Department of Anesthesia Didactic Course, Massachusetts General Hospital, Boston, MA. February, 2003. "Why Doesn't the Blood Clot?"

Cedars-Sinai Hospital Grand Rounds, Miami, FL. February, 2003. "Inflammation, Coagulation, and a Drug that Connects the Two."

Sylvester Cancer Center, University of Miami, Miami, FL. February 2003. "The Mechanism of Action of Recombinant Activated Protein C."

Lowell General Hospital Grand Rounds, Lowell, MA. February, 2003. "The Diagnosis and Treatment of Hypercoagulable States."

Texas Children's Hospital Grand Rounds Hematology – Oncology. Houston, TX. March, 2003. "Hypercoagulable States and Their Association with Malignancy."

Houston City Wide Hematology Conference, Houston, TX. March, 2003. "Hypercoagulable States andthe 2001 Consensus Conference Recommendations for Thrombophilia Testing."

ASCP Resident Review Course, Hoffman Estates, IL. April, 2003. "Coagulation Overview."

Professional Practice in Clinical Chemistry: A Review and Update, Arlington, VA. April, 2003. "An Overview of Bleeding and Thrombotic Disorders."

Updates in Anesthesia, Boston, MA. May, 2003. "Why Doesn't the Blood Clot?"

New York State Society of Pathologists, 34th Annual Continuing Education Meeting, Binghamton, NY. May, 2003. "Hypercoagulability."

New Frontiers in Vascular and Endovascular Surgery, Boston, MA. May, 2003. "What Surgeons Should know about New Heparins and Hypercoagulability."

Conemaugh Valley Memorial Hospital, Johnstown, PA. May, 2003. "Hypercoagulable States."

North Shore University Hospital, Forest Hills, NY. May, 2003. "Low Molecular Weight Heparin: Prophylaxis and Treatment of DVT and PE."

Windham Hospital Grand Rounds, Willamantic, CT. June, 2003. "Diagnosis and Treatment of Hypercoagulable States."

Current Concepts in Clinical Pathology, Boston, MA. June, 2003. "Whom to Monitor for Hypercoagulability – Report of the CAP Consensus Conference on Thrombophilia Testing" and "Common Errors in Coagulation."

Reproductive Endocrinology & Gynecology Seminar Series, Brigham and Women's Hospital, Boston, MA. September, 2003. "Hypercoagulable States."

Advances in Rheumatology, Continuing Education Course, Harvard Medical School, Boston, MA. September, 2003. "Hypercoagulability Testing: Current Practice to Assess Thrombotic Risk."

Cancer Medicine and Hematology, Harvard Medical School Continuing Education Course, September, 2003. "Case Management Problems in Coagulation."

30th Meeting of the New England Society for Vascular Surgery, Newport, RI. September 2003. "Diagnosis of Hypercoagulable States."

A Comprehensive Board Review in Hematology and Medical Oncology, Houston, TX. October, 2003. "Hypercoagulable States."

Montefiore Medical Center/Albert Einstein College of Medicine, Bronx, NY. Visiting Professor. January, 2004. "Diagnosis and Treatment of Hypercoagulable States."

Cardiology and Cardiac Surgery Practice Management Rounds, Massachusetts General Hospital, Boston, MA. February, 2004. "The Pathogenesis, Diagnosis, and Treatment of Heparin-Inducted Thrombocytopenia."

Anesthesia Grand Rounds, Newton-Wellesley Hospital, Newton, MA. March, 2004. "Anticoagulants Old and New."

5th Annual Louisiana Coagulation Conference, Metairie, LA. March, 2004. "Diagnosis and Treatment of Thrombophilic States."

Newport Hospital Grand Rounds, Newport, RI. March, 2004. "What is New & Old in Anticoagulation."

Cedars-Sinai Hospital, Visiting Professor Los Angeles, CA. April, 2004. "The Diagnosis & Treatment of Hypercoaguable States."

ASCP Resident Review Course, Hoffman Estates, IL. April, 2004. "Coagulation."

Harvard Medical School Anesthesia Review and Update 2004, Boston, MA. May, 2004. "Why Doesn't the Blood Clot and Why Does the Blood Keep Clotting?"

University of Rome, Department of Experimental Medicine and Pathology, Rome, Italy. June, 2004. "The Diagnosis and Treatment of Hypercoagulable States."

Society of Hospital Medicine, 2004 Northeast Regional Meeting, Boston, MA. June, 2004. "Anticoagulants for the Practicing Hospitalist: Monitoring of New Agents, Pitfalls in the Use of Older Agents."

Current Concepts in Hemostasis Symposium, John F. Kennedy Library and Museum, Boston, MA. June, 2004. "Hypercoagulable States: Their Role in Thrombosis with Air Travel, Pregnancy, and Estrogen Supplementation."

Westerly Hospital Grand Rounds, Westerly, RI. September, 2004. "Anticoagulants and Thrombosis Prevention."

South County Hospital Grand Rounds, Wakefield, RI. September, 2004. "Anticoagulants: An Overview" and "Heparin-Induced Thrombocytopenia."

Cancer Medicine and Hematology, Harvard Medical School Continuing Education Course, September, 2004. "Case Management Problems in Coagulation."

Advances in Anticoagulation Symposium, Massachusetts General Hospital, Boston, MA. October, 2004. "The Coagulation Cascade and Patients at Risk" and "Heparin-Inducted Thrombocytopenia."

Update in Hemostasis and Thrombosis, Lehigh Valley Hospital, Allentown, PA. October, 2004. "The Diagnosis and Treatment of Hypercoagulable States."

Massachusetts General Hospital Department of Anesthesia Grand Rounds, Boston, MA. January, 2005. "Hypercoagulable States."

Children's Memorial Hospital (The pediatrics hospital for Northwestern University Medical Center), Grand Rounds, Chicago, IL. March, 2005. "Coagulation Disorders in Children."

Emerson Hospital Grand Rounds, Concord, MA. April, 2005. "Anticoagulants: Indications and Monitoring."

Spaulding Rehabilitation Hospital, Grand Rounds. "Heparin-Induced Thrombocytopenia: Diagnosis and Treatment with Argatroban." Boston, MA. April, 2005

Coagulation Symposium sponsored by Diagnostica Stago, New York City, NY. May, 2005. "Thrombophilia."

ASCP Resident Review Course, Hoffman Estates, IL. May, 2005. "Coagulation."

Current Concepts and Controversies in Vascular and Endovascular Surgery Symposium, Boston, MA. May, 2005. "What Surgeons need to know about Hypercoagulable States" and "HIT Syndrome: A Major Concern."

Professional Practice in Clinical Chemistry: A Review and Update, Arlington, VA. May, 2005. "An Overview of Bleeding and Thrombotic Disorders."

Anesthesia Review and Update 2005, Boston, MA. May, 2005. "Why doesn't the blood clot and why does it clot too much?"

Harvard Medical School Continuing Education Course, Updates in Laboratory Medicine,Boston, MA. June, 2005. "Anticoagulants."

Scientific Symposiums, Hilton Head, SC. June, 2005. "Bleeding Disorders."

Scientific Symposiums, Hilton Head, SC. June, 2005. "Hypercoagulable States."

J. Heinrich Joist First Memorial Lecture, St. Louis, MO. September, 2005. "Hypercoagulable States: Diagnosis and Treatment."

Harvard Medical School Continuing Education Course, Advances in Rheumatology, Boston, MA. September, 2005. "Coagulopathies."

Harvard Medical School Continuing Education Course, Cancer Medicine and Hematology, Boston, MA. September, 2005. "Errors in Diagnosis: Thrombosis and Hemostasis Core Studies."

Current Concepts in Hemostasis, Industry Sponsored Symposium, National Constitution Center Museum, Philadelphia, PA. October, 2005. "The Diagnosis and Treatment of Hypercoagulable States."

Comprehensive Update on Vascular Disease for the Primary Care and Specialty Provider, Waltham, MA. November, 2005. "Hypercoagulable States: Update on How, Who, and When to Screen."

Lahey Clinic Surgery Grand Rounds. Burlington, MA. January, 2006. "An Overview of Coagulation for the Surgeon."

Brigham and Women's Hospital, Division of Rheumatology. Boston, MA. January, 2006. "Antiphospholipid Antibodies: What they are and What they do."

Beth Israel Deaconess Medical Center, Division of Rheumatology, Boston, MA. March, 2006. "Antiphospholipid Antibodies and Antiphospholipid Syndrome: Description, Diagnosis, and Treatment."

The Hemostasis Scientific Advisory Committee 2006 Meeting, InstrumentationLaboratory, Lexington, MA. April, 2006. "Point of Care Coagulation Testing: What Assays Could Move Out of the Main Laboratory and Institutional Factors which Influence that Decision."

The Knight Nursing Center for Clinical and Professional Development, Boston, MA. May 2005. "The causes and common bleeding and clotting disorders and what to do about them."

Harvard Medical School Anesthesia Review and Update 2006, Boston, MA. May, 2006. "Why Doesn't the Blood Clot and Why Does the Blood Keep Clotting?"

Current Concepts and Controversies in Vascular and Endovascular Surgery Symposium, Boston, MA. May, 2006. "New Age Anticoagulants" and "HIT: A New Major Problem."

In Vein: Treatment and Techniques Symposium, Uncasville, CT, June, 2006. "Hypercoagulable States: A primer for the office based physician."

American Association of Clinical Chemistry Critical & Point-of-Care Testing Division, 4th Annual POC Coordinators Forum, Chicago, IL. July, 2006. "Anticoagulation with Warfarin and Heparin: How the Drugs Work, How They are Monitored, and What the Results Mean" and "Point of Care Testing in Coagulation: Barriers to Effective Implementation."

American Association of Clinical Chemistry Annual Meeting, Chicago, IL. July, 2006. "Coagulation Testing: The Role of the Laboratory Director in Optimizing Patient Outcome."

Cancer Medicine and Hematology, Harvard Medical School Continuing Education Course. September, 2006. "Case Studies in Hemostasis".

In Vein Treatment and Techniques, Las Vegas, NV. October, 2006. "Hypercoagulable States: A Primer for Office Management."

Life After a Heart Attack: A Lecture for Community Health Awareness (for the general public). Malden, MA. November, 2006.

Critical Care and Trauma Symposium, Boston, MA. November, 2006. "Diagnosing a Coagulopathy in the ICU."

Sixth Annual Florida East Coast Point of Care Conference, Cocoa Beach, FL. November, 2006. "POCT in Coagulation: The INR and Much More".

Visiting Professor, Ball Memorial Hospital, Department of Pathology, Muncie, IN. December, 2006. "An Overview of Thrombotic and Bleeding Disorders."

Youville Rehabilitation Hospital, Grand Rounds, Cambridge, MA. February, 2007. "The Management of Venous Thromboembolic Disease."

Melrose-Wakefield Hospital Grand Rounds, Malden, MA. January, 2007. "Hypercoagulable states, Anticoagulants, and Heparin-Induced Thrombocytopenia".

Vascular and Abdominal Interventional Radiology Conference, Massachusetts General Hospital, Boston, MA. February, 2007. "Old Age and New Age Anticoagulants: How They Are Used, How They Are Monitored, and How They are Reversed."

Cedar-Sinai Medical Center Grand Rounds, Los Angeles, CA. February, 2007. "The Diagnosis and Treatment of Hypercoagulable States."

Evergreen Hospital Grand Rounds, Seattle, WA. February, 2007. "The Diagnosis and Treatment of Hypercoagulable States."

University of Rome (La Sapienza), School of Medicine. Lecture to Medical Students. Rome, Italy. March, 2007. "An Overview of Hemostasis."

Massachusetts General Hospital Vascular Summit: A Comprehensive Update for the Primary Care Provider, Boston, MA. March, 2007. "Special Course Lecture: Hypercoagulable States: Update on How, Who, and When to Screen."

New England Regional Chemistry Exposition (NERCE), Boxborough, MA. April, 2007. "The Diagnosis and Treatment of Hypercoagulable States."

Obstetrics and Gynecology Grand Rounds, Massachusetts General Hospital, Boston, MA. April, 2007. "Hypercoagulable States and Their Many Connections to Obstetrics and Gynecology."

Professional Practice in Clinical Chemistry: A Review and Update. Washington, DC. April, 2007. "Coagulation."

In Vein: Treatment and Techniques. Uncasville, CT. April, 2007. "What Do I Really Need to Know About Hypercoagulable States." and "How to Evaluate The Patient with Idiopathic DVT."

ASCP Resident Review Course, Director of Laboratory Medicine Section and Lecturer. Chicago, IL. April, 2007. "An Overview of Coagulation."

New England Pathology Resident Forum. Boston, MA. April, 2007. "Coagulation Disorders."

Anesthesia Review and Update, Harvard Continuing Medical Education Symposium, Boston, MA. May, 2007. "Why Doesn't The Blood Clot?" and "Why Does the Blood Keep Clotting?"

Pulmonary and Critical Care, Harvard Continuing Medical Education Symposium, Boston, MA. May, 2007. "Coagulation."

Contemporary Practice of Vascular and Endovascular Surgery: What the Vascular Surgeon Needs to Know, Harvard Continuing Medical Education Symposium, Boston, MA. June, 2007. "Hypercoagulable States, Heparin-Induced Thrombocytopenia, and New Age Anticoagulants."

Cancer Related Emergencies Symposium, Massachusetts General Hospital, Boston, MA. June, 2007. "Coagulopathies and DIC."

Rheumatology Grand Rounds, University of Massachusetts Medical School, Worcester, MA. September, 2007. "Antiphospholipid Antibodies: What They Are and What To Do About Them."

Advances in Rheumatology, Harvard Continuing Medical Education Course, Boston, MA. September, 2007. "Coagulopathies".

Vascular Medicine Continuing Education Symposium Massachusetts General Hospital, Boston, MA. October, 2007. "Anticoagulation Pathways"

Applying Anatomic and Clinical Pathology to Reach a Diagnosis. Harvard Continuing Medical Education Course. "Lymphaigitic Spread of Tumor and DIC" and "Patent Foramen Ovale and Hypercoagulable States"

Hackensack University Medical Center, Surgery Grand Rounds, Hackensack, NJ. October, 2007. "Coagulation for the Surgeon."

ASCP Resident Review Course, Director of Laboratory Medicine Section and Lecturer. Chicago, IL. April, 2008. "An Overview of Coagulation."

Vascular Medicine Annual Meeting, San Diego, CA. June, 2008. "Hypercoagulability in Vascular Disease: When Should You Consider This, and What Tests Should You Order?".

American Association of Clinical Chemistry (AACC) National Meeting, Washington, D.C. July, 2008 "Overdiagnosis of Child Abuse Due to Undiagnosed Underlying Disease".

Department of Medicine Grand Rounds, University of Alabama at Birmingham, Birmingham, AL. September, 2008. "The Use and Monitoring of Anticoagulants".

College of American Pathologists Annual Meeting, San Diego, CA. September, 2006. "Pitfalls in Diagnosis".

American Academy of Pediatrics Premeeting Satellite Session on Diagnosis and Management of Coagulation Disorders, Boston, MA. October, 2008. "Diagnosis and Treatment of Common Bleeding Disorders."

XXXVIII Mexican Congress on Clinical Pathology, Acapulco, Mexico. October, 2008. Half-day symposium on "The Diagnosis and Treatment of Coagulation Disorders."

Department of Pathology, University of Texas- San Antonio. Visiting Professor, San Antonio, TX. February, 2009. "The Appropriate Use and Laboratory Monitoring of Anticoagulants."

ASCP Resident Review Course, Director of Laboratory Medicine Section and Lecturer. Chicago, IL. April, 2009. "An Overview of Coagulation."

American Association of Clinical Chemistry, Professional Practice Course, Alexandria, VA. April, 2009. "Diagnosis of Coagulation Disorders."

Anesthesia Review and Update, Harvard Continuing Medical Education Symposium, Boston, MA. May, 2009. "Why Doesn't The Blood Clot?" and "Why Does the Blood Keep Clotting?"

The Clinical Laboratory Management Association of Central New York and the American Association of Clinical Chemistry Upstate New York sections, Verona, NY. October, 2009. "Laboratory Tests in the Diagnosis of Bleeding and Thrombotic Disorders."

Harold Bernard Stroke and Neurosciences Symposium, Nashville, TN. November, 2009. "Clotting Factors as a risk for Stroke."

Visiting Professor, Ball Memorial Hospital, Department of Pathology, Muncie, IN. January, 2010. "Hypercoagulable states: Diagnosis and Treatment"

Visiting Professor, Ball Memorial Hospital, Department of Pathology, Muncie, IN. January, 2010. "Anticoagulants: Clinical Indications and Monitoring"

ASCP Resident Review Course, Director of Laboratory Medicine Section and Lecturer. Chicago, IL. April, 2010. "An Overview of Coagulation."

Harvard Anesthesia Update 2010, Boston, MA. May, 2010. "Why Doesn't the Blood Clot?" and "Why Does the Blood Keep Clotting?"

Tennessee Donor Services Medical Advisory Committee, Nashville, TN. April, 2010. "Disseminated Intravascular Coagulation in the Transplant Patient."

University of South Florida, Pathology Grand Rounds, Tampa, FL. January, 2011. "Case Studies in Laboratory Management: Coagulation Disorders."

ASCP Resident Review Course, Director of Laboratory Medicine Section and Lecturer. Chicago, IL. April, 2011. "An Overview of Coagulation."

Harvard Anesthesia Update 2011, Boston, MA. May, 2011. "Why Doesn't the Blood Clot?" and "Why Does the Blood Keep Clotting?"

AACC New York Metro Section, New York, NY. May, 2011. "Mistakes in Diagnostic Coagulation Made by Others – So You Will Not Make Them Yourself."

ASCP Resident Review Course, Tampa, FL. February, 2012. "Coagulation"

7th New Orleans Coagulation Conference, Keynote Address. New Orleans, LA. February, 2012. "The Diagnostic Management Team in Coagulation at Vanderbilt: How It Works and Its Clinical and Financial Impact.":

Laboratory Medicine/Clinical Pathology

Association of Pathology Chairperson's Annual Meeting, Aspen, CO. July, 1990. "Why Medical Students Choose Careers in Pathology."

American Society of Clinical Pathologists National Meeting, Nashville, TN. March, 1991. "Development of a Residency Training Program in Laboratory Medicine."

Hungarian National Academy of Sciences, Budapest, Hungary. September, 1991. "Laboratory Medicine Training in the United States."

The Ontario Medical Association Annual Meeting, Toronto, Canada: October, 1991. "The Rapidly Evolving Specialty of Laboratory Medicine."

University of Massachusetts School of Medicine, Worcester, MA. October, 1991. "Laboratory Medicine Training at the Massachusetts General Hospital."

Academy of Clinical Laboratory Physicians & Scientists National Meeting, San Francisco, CA. June, 1992. "Laboratory Medicine Teaching Programs for Medical Students in the Basic Science Curriculum."

Canadian Association of Pathologists National Meeting, Toronto, ONT. June, 1992. "The Role of the Clinical Laboratory in Patient Care".

42nd Congress of the Hungarian Society of Clinical Pathology, Veszprem, Hungary. September 1992. "The Practice of Clinical Pathology in the U.S."

ASCP/CAP National Meeting, Las Vegas, NV. October 1992. "Graduated Responsibility for Residents in Clinical Pathology."

Department of Laboratory Medicine, University of Washington, Seattle, WA. December, 1993. Visiting Professor.

Department of Pathology, University of Utah School of Medicine, Salt Lake City, UT. December, 1993. "Training Residents in Clinical Pathology to be Effective Clinical Consultants."

Department of Pathology, University of Vermont School of Medicine, Burlington, VT. January, 1994. "Point of Care Testing."

Department of Pathology, Cornell University School of Medicine, New York, NY. February, 1994. "Establishing a Residency Program and Consult Service in Clinical Pathology."

Department of Pathology, Duke University School of Medicine, Durham, NC. February, 1994. "Alternate Site Laboratory Testing."

Massachusetts Society of Pathologists, Framingham, MA. May, 1994. "Bedside Testing."

Department of Pathology, Visiting Professor, Emory University School of Medicine, Atlanta, GA. October, 1994. "Bedside Testing" and "Initiating a Consult Service in Clinical Pathology."

Northeast Chapter of the American Association for Clinical Chemistry, October, 1994. "Alternate Site Laboratory Testing."\

ASCP/CAP National Meeting, Washington, D.C. October, 1994. "The Introductory Rotation in Clinical Pathology Residency Training."

ASCP/CAP National Meeting, Washington, D.C. October, 1994. "Stump the Stars: Review of Clinical Cases by Expert Panelists."

ASCP/CAP National Meeting, Washington, D.C. October, 1994. "Should This Test Be Done: Lipid Screening Tests."

Department of Pathology, Visiting Professor, Albany Medical College, Albany, NY. January, 1995. "Development of a Clinical Pathology Consultation Service."

College of American Pathologists Symposium on Alternate Site Testing, Washington, DC. January, 1995. "A Successful Model of Bedside Glucose Testing."

Fifth Annual Symposium on Coagulation Testing Sponsored by the BioData Corporation, Philadelphia, PA. March, 1995. "Bedside Coagulation Testing."

Department of Pathology & Laboratory Medicine, Hahnemann University School of Medicine, Philadelphia, PA. March, 1995. "Point of Care Testing."

Ninth Annual Meeting of Northeast Section of American Association of Clinical Chemistry, Clinical Laboratory Management Association, and Clinical Ligand Society, Danvers, MA. May, 1995. "Cost & Quality in Point-of-Care Testing."

Baystate Medical Center (Affiliate of Tufts University School of Medicine, Springfield, MA. September, 1995. "Development of a Residency Training Program in Clinical Pathology."

ASCP/CAP National Meeting, New Orleans, LA. September, 1995. "Bedside Glucose Testing: The Facts and Nothing but the Facts."

Division of Laboratory Medicine, Visiting Professor, Washington University School of Medicine, St. Louis, MO. April, 1996. "The Development of the Clinical Consultation Service in Laboratory Medicine at the Massachusetts General Hospital"

Tenth Annual Meeting of the Northeast Section of the Clinical Laboratory Management Association and the Clinical Ligand Assay Society, Boxborough, MA. May, 1996. "Point of Care Testing at the Massachusetts General Hospital."

Department of Welfare, Hungarian National Government, Budapest, Hungary. July, 1996. "The Clinical Laboratory Inspection and Accreditation Process in the United States."

Department of Pathology, New York University, New York, NY. January, 1997. "The Development of a Residency Training Program and Clinical Consultation Service in Clinical Pathology at Massachusetts General Hospital."

Biomedical Marketing Association, Boston, MA. March, 1997. "Maximizing the Output and Diagnostic Testing in the Clinical Laboratory."

Laboratory Parameter Analysis Panel, Chicago, IL. March, 1997. "Clinical Protocols and Laboratory Medicine."

ASCP Advisory Council Meeting. Chicago, IL. April, 1997. "Clinical Pathology: Its Evolution into a Laboratory Test Interpretive Service Indispensable to Clinicians."

Massachusetts Society of Pathologists, Framingham, MA. April, 1997. "Clinical Pathology- How to Do It and How to Get Paid for It."

Northeastern University, Boston, MA. May, 1997. "Trends in Laboratory Medicine."

American Association of Clinical Chemistry National Meeting, Atlanta, GA. July, 1997. "The Clinical Impact of Point of Care Testing."

Controversies in Critical Care, Society of Critical Care Medicine Meeting, Boston, MA. September, 1997. "Does Point of Care Testing Improve Patient Care and Is It Cost Effective?"

Integrating Point-of-Care Testing with Continuity of Care: Effects on Outcome, Meeting of the National Academy of Biochemistry. Philadelphia, PA. September, 1997. "Case Studies on Point of Care Testing."

ASCP/CAP National Meeting, Philadelphia, PA. September, 1997. "The New Role of the Pathologist as a Direct Medical Consultant."

Northeastern University Centennial Celebration, Boston, MA. November, 1997. "The Future of Laboratory Medicine in an Era of Managed Care."

Ball Memorial Hospital, Muncie, IN. March, 1998. "Value Added Services in Clinical Laboratory Testing."

Department of Pathology and Laboratory Medicine, Dartmouth Medical School, Hanover, NH. April, 1998. "Value Added Clinical Laboratory Services: What the Clinicians Have Been Waiting For."

Department of Medical Laboratory Sciences, University of New Hampshire, Durham, NH. April, 1998. "Providing Clinicians with More Than Just a Laboratory Test Result."

Department of Pathology and Laboratory Medicine, Visiting Professor, University of Arizona School of the Health Sciences, Tucson, AZ. April, 1998. "Clinical Laboratory Medicine: New Roles in Health Care."

College of American Pathologists Conference on Automated Information Management in the Clinical Laboratory, Ann Arbor, MI. May, 1998. "The Pathologist as a Direct Medical Consultant."

Academy of Clinical Laboratory Physicians and Scientists National Meeting, Boston, MA. June, 1998. "Narrative Interpretations in the Clinical Laboratory: The Basic Concepts."

Biomedical Marketing Association, Philadelphia, PA. August, 1998. "Current Trends in Clinical Laboratories."

Brazilian Congress of Clinical Pathology, Rio de Janeiro, Brazil. September, 1998. "Redefining Expectations of Clinical Laboratory Services."

Department of Pathology, Ohio State University School of Medicine, Columbus, Ohio. September, 1998. "Providing More than a Laboratory Test Result: A New Package of Value Added Services from the Clinical Laboratory."

Association of Pathology Chairpersons - Northeast Section, Bermuda. October, 1998. "The Added Value of the Clinical Pathologist in the Practice of Laboratory Medicine."

Department of Pathology, The Cleveland Clinic, Visiting Professor, Cleveland, OH. January, 1999. "The Value Added Clinical Laboratory Services at the Massachusetts General Hospital."

Department of Pathology, The Cleveland Clinic, Visiting Professor, Cleveland, OH. January, 1999. "The Development of the Laboratory Medicine Residency Training Program at the Massachusetts General Hospital."

Department of Pathology, Thomas Jefferson University School of Medicine, Visiting Professor, Philadelphia, PA. January, 1999. "Providing More than a Test Result: Redefining Expectations from the Clinical Laboratory."

Department of Pathology, University of Texas Houston, Visiting Professor, Houston, TX. March, 1999. "Narrative Interpretations in Laboratory Medicine."

Department of Pathology, University of Texas Houston, Visiting Professor, Houston, TX. March, 1999. "Finding Your Place in the World of Pathology."

Department of Pathology, University of Texas Houston, Visiting Professor, Houston, TX. March, 1999. "Laboratory Medicine Training at the Massachusetts General Hospital."

Department of Pathology, Penn State-Hershey Medical Center, Hershey, PA. April, 1999. "The Value Added Clinical Laboratory Services at the Massachusetts General Hospital."

Department of Pathology, Berkshire Medical Center, Pittsfield, MA April, 1999. "Redefining Expectations from the Clinical Laboratory."

College of American Pathologists Conference on Automated Information in the Clinical Laboratory, Executive Briefing, Ann Arbor, MI. May, 1999. "Enhanced Clinical Consulting: Getting Closer to the Sweet Spot."

Fourth International Bayer Diagnostics Laboratory Testing Symposium, Tuczon, AZ. June, 1999. "A Clinician's View of the Future."

Department of Pathology, University of Virginia School of Medicine, Charlottesville, VA. July, 1999. "Redefining Expectations from the Clinical Laboratory."

Associations of Pathology Chairs/Pathology Residency Program Directors Meeting, Boulder, CO. July, 1999. Workshop entitled, "Training Pathologists to be Clinical Consultants: Providing Added Value-Let's Get Real!"

Department of Pathology, University of Pittsburgh Medical Center, Pittsburgh, PA. September, 1999. "The Value Added Clinical Laboratory Services at the Massachusetts General Hospital."

ASCP/CAP National Meeting, New Orleans, LA. September, 1999. "Clinical Pathology Narrative Interpretations - How to do Them and How to get Paid for Them."

Department of Pathology, UCLA School of Medicine, Los Angeles, CA. October, 1999. "Redefining Expectations from the Clinical Laboratory."

Kaiser Permanente Medical Group, Los Angeles, CA. October, 1999. "The New Array of Services Provided by the Massachusetts General Hospital Clinical Laboratories."

Advancing Pathology Informatics, Imaging and the Internet, Pittsburgh, PA. October, 1999. "The Pathologist as Digital Consultant: The Enhanced Clinical Consultation."

American Society for Clinical Laboratory Scientists 1999 National Meeting for Advanced Hematology and Hemostasis, Providence, RI. October, 1999. "Providing More than Just a Coagulation Test Result: The Value-Added Clinical Laboratory Services at the Massachusetts General Hospital."

Department of Pathology, St. Louis University School of Medicine, St. Louis, MO. January, 2000. "Value Added Clinical Laboratory Services - A New Approach to Laboratory Medicine."

Pathology Education Institute, Snowmass, CO. February, 2000. "Redefining the Role of the Pathologist in the Clinical Laboratory."

Department of Pathology, University of South Florida School of Medicine, Tampa, FL. February, 2000. "The Value Added Clinical Laboratory Services at the Massachusetts General Hospital."

7th Annual Progress in Clinical Pathology, Dallas, TX. March, 2000. "Redefining the Role of the Clinical Laboratory: Clinical Pathology for the New Millenium."

Department of Pathology and Laboratory Medicine, Hartford Hospital, Hartford, CT. March, 2000. "Establishing a Clinical Pathology Consultation Service."

Department of Pathology, Northwestern University School of Medicine, Chicago, IL. April, 2000. "Redefining Laboratory Medicine."

Resident Physician Forum, American Society of Clinical Pathologists National Meeting, Boston, MA. April, 2000. "Emerging Changes in the Field of Laboratory Medicine."

New England Sunquest Users Group, Sturbridge, MA. April, 2000. "The Value-Added Clinical Laboratory Services at the Massachusetts General Hospital."

2000 Executive War College on Lab and Pathology Management, New Orleans, LA. May, 2000. "Clinical Pathology Professional Services for which Managed Care Companies will Reimburse."

Evidence-Based Medicine: Optimizing Decision-Making to Improve Patient Care, Arlington, VA. May, 2000. "The Role of Evidence-Based Medicine in Medical Decision Making-Case Studies in Hemostasis" and "Techniques to Improve Physicians' Use of Diagnostic Tests."

The 5th International Bayer Diagnostics Laboratory Symposium, Seville, Spain. June, 2000. "The Future and Ethics of Screening and Preventive Medicine."

The Wagih Bari Society of St. Louis Pathologists, St. Louis, MO. September, 2000. "The Nuts and Bolts of Operating an Interpretive Service in Clinical Pathology. The Regulations on Reflex Testing, Performing Interpretations, and Billing."

The Wagih Bari Society of St. Louis Pathologists, St. Louis, MO. September, 2000. "The Value Added Clinical Pathology Services Provided at the Massachusetts General Hospital and a Glance into the Future."

Sharp Health Care, Educational Symposium for the Physicians in the Sharp Health Care System, San Diego, CA. October, 2000. "Reducing Error Through Appropriate Test Ordering."

ASCP/CAP National Meeting, San Diego, CA. October, 2000. "Clinical Pathology Narrative Interpretations: How to Do Them and How to Get Paid."

Harvard Medical School at the Millennium: What's New and What's Happening In and Around the Quadrangle, Boston, MA. October, 2000. "Demonstration of a Web-Based System to Assist Physicians in the Selection and Interpretation of Laboratory Tests."

Illinois Society of Pathologists 2000 Fall Meeting, Rosemont, IL. November, 2000. "A Complete Re-Definition of the Services Provided by the Clinical Laboratory."

Baystate Medical Center, Update in Diagnostic Pathology and Medicine Series, Springfield, MA. November, 2000. "The Value Added Clinical Laboratory Services at the Massachusetts General Hospital."

Johns Hopkins University School of Medicine, Distinguished Visiting Professorship of Pathology, Baltimore, MD. February, 2001. "A Complete Redefinition of the Clinical Laboratory Service that Reduces Medical Error at the Massachusetts General Hospital."

Mt. Sinai Medical Center, Miami, FL. February, 2001. "New Expectations from the Clinical Laboratory."

University of Miami School of Medicine, Miami, FL. February, 2001. "Redefining the Services of the Clinical Laboratory."

American Association of Clinical Chemistry and the College of American Pathologists Symposium on the Clinical Laboratory Meets the Internet, Miami, FL. February, 2001. "Enhanced Clinical Consulting Meets the Web."

2001 Executive War College on Lab and Pathology Management, Cincinnati, OH. May, 2001. "Converting Lab Test Results into the Value Added Services Wanted by Physicians and HMOs" and "Doing It Right: How Clinical Pathologists Can Build Value-Added Professional Opinions into Lab Test Reports."

Automated Information Management in the Clinical Laboratory Symposium, Ann Arbor, Michigan. May, 2001. "Enhanced Clinical Consulting: Moving Toward the Core Competencies of Lab Professionals."

ASCP/CAP National Meetings, Philadelphia, PA. October, 2001. "The Clinical Laboratory Meets the Internet: Strategies and Solutions."

International Conference on Laboratory Medicine– Continuous Education, Duties, and Responsibilities of Professionals in Medical Laboratories, Padua, Italy. October, 2001. "Enhanced Clinical Consulting: Moving Toward the Core Competencies of Laboratory Professionals."

University of Massachusetts Medical School, Worcester, Massachusetts. November, 2001. "A Web-Based System for Patient-Specific Narrative Interpretations in Clinical Pathology."

Louisiana State University Medical Center, Department of Pathology, New Orleans, LA. February, 2002. "A Complete Re-Definition of Services Provided by the Clinical Laboratory."

18th Annual Regional Meeting of the Clinical Laboratory Managers Association, Pittsburgh, PA. March, 2002. "Redefining Expectations from the Clinical Laboratory."

9th Annual Progress in Clinical Pathology Symposium, Dallas, TX. April, 2002. "Medical Errors in Reality" & "Evidenced-Based Medicine-Identifying Appropriate Tests for the Evaluation of Thrombophilia."

Hawaii Society of Pathologists, Honolulu, HI. October, 2002. "Redefining Expectation from the Clinical Laboratory."

University of Texas at San Antonio, San Antonio, TX. November, 2002. "Redefining expectationsfrom the clinical laboratory."

XVI Congress Latinoamericano de Patologia Clinica VI and Congreso Iberoamericano de Medicina Tansfusion, Acapulco, MX. November, 2002. "Redefinition of Clinical Laboratory Services."

Department of Pathology, University of Pennsylvania School of Medicine, Philadelphia, PA. January, 2003. "Redefining expectations from the Clinical Laboratory."

Connecticut Society of Pathologists, New Haven, CT. January, 2003. "Establishing a fee for service clinical pathology consultation service."

Department of Pathology, Louisiana State University School of Medicine, New Orleans, LA. February, 2003. "Value Added Clinical Pathology."

Department of Pathology, Baylor University School of Medicine, Houston, TX. March, 2003. "Redefining Expectations from the Clinical Laboratory."

Centers for Disease Control and Prevention, Quality Institute Conference on "Making the Laboratory a Key Partner in Patient Safety," Atlanta, GA. April, 2004. "Reducing Medical Errors by Providing Expert Advice in the Selection and Interpretation of Laboratory Tests."

Dade Behring Hemostasis Symposium, Raleigh-Durham, NC. May, 2003. "A Complete Redefinition of the Services Provided by the Clinical Laboratory."

Automated Information Management in the Clinical Laboratory, 21st annual symposium, Ann Arbor, MI. May, 2003. "How to Turn Laboratory Consulting into a Revenue-Generating Operation."

Conemaugh Valley Memorial Hospital, Johnstown, PA. May, 2003. "Redefining Clinical Laboratory Services."

Federal Government Laboratorians Seminar (sponsored by Roche). July, 2003. National Liberty Museum, Philadelphia, PA. "Reducing Medical Errors by Providing Expert Advice in the Selection and Interpretation of Laboratory Tests."

Washington G-2 reports, 21st Annual Lab Institute. Arlington, VA. October, 2003. "Lab & Pathology Trailblazers: Early Adapter Solutions and Strategies."

Centers for Disease Control (CDC) Executive Session on the formation of the Quality Institute for Laboratory Medicine, Atlanta, GA. December, 2003. "Quality Enhancement of Clinical Laboratory Services – An External Perspective on the Issues."

Montefiore Medical Center/Albert Einstein College of Medicine, Bronx, NY. January, 2004. Visiting Professor. "Finding your Place in the World of Pathology."

Washington G-2 reports, Achieving Outreach Leadership for Lab & Pathology Services: Positioning your Program for Success, Atlanta, GA. February, 2004. "Succeeding in the Outreach Market: Applying Critical IT Solutions."

Frontiers in Laboratory Medicine 2004: Changing Process, Improving Outcomes, Manchester, England. February, 2004. "Making Laboratory Medicine More Valuable to Physicians and Patients."

Lab Infotech Summit, Las Vegas, NV. March, 2004. "Clinical Laboratory Consulting: Quality and Financial Implications."

Department of Pathology, University of Alabama at Birmingham, Birmingham, AL. March, 2004. "Finding your Place in the World of Pathology."

Instrumentation Laboratories International Advisory Board, Cambridge,MA. April, 2004. "The Added Value of Patient-Specific Narrative Interpretations."

Cedars-Sinai Hospital, Visiting Professor, Los Angeles, CA. April, 2004. "Finding your Place in the World of Pathology."

Cedars-Sinai Hospital, Visiting Professor, Los Angeles, CA. April, 2004. "Redefining clinical laboratory services for improved patient safety."

Association of Clinical Scientists, Abraham J. Gitlitz Memorial Lecture as Keynote address at 124th Annual Meeting, Houston, TX. May, 2004. "The Future of Physicians in Laboratory Medicine."

Department of Pathology, University of Texas, Houston, TX. May, 2004. "Finding Your Place in the World of Pathology."

Department of Pathology, University of Texas, Houston, TX. May, 2004. "Value Added Services in Clinical Pathology."

Department of Laboratory Medicine and Pathology, University of Alberta, Edmonton, Canada. June, 2004. "Patient Safety: Have We Overlooked Errors in Test Selection and Interpretation of Laboratory Tests?"

Clinical Laboratory Management Association (CLMA), Bay State Chapter, Boston, MA. September, 2004. "Laboratory Practices to Increase Patient Safety and Decrease Errors."

Michigan Society of Pathologists Winter Seminar, Ann Arbor, MI. December 2004. "Coagulation in the Clinical Laboratory."

Top Challenges in Laboratory Medicine Management, American Association of Clinical Chemistry, Dallas, TX. December, 2004. "Clinical Utility: Making the Most of your Results."

Diagostica Stago National Sales Meeting, Palm Springs, CA. February, 2005. "Characteristics of the Ideal Coagulation Laboratory Service Representative.

College of American Pathologists, National House of Delegates Meeting, San Antonio, TX. February, 2005. "The Role of the Laboratory Medical Director."

2005 Institute for Quality in Laboratory Medicine Conference. Atlanta, GA. April, 2005. "Ask the Experts Session."

2005 Annual Meeting of the American Association for Clinical Chemistry, Orlando, FL. July, 2005. "Value Added Test Ordering and Interpreting Services Improve Safety and Effectiveness in Healthcare."

2005 Annual Meeting of the College of American Pathologists, Chicago, IL. September, 2005. "Pathologist Physician Communication," a presentation as part of the Practice Management Institute: Service and Quality.

Clinical Diagnostics: Creating Greater Value, Industry Sponsored Symposium, Boston, MA. October, 2005. "The Health Economics of Biomarkers in Cardiopulmonary Disease" (one of 7 plenary session talks following keynote address by nobelist, Dr. James Watson).

Florida Society of Pathologists National Meeting. Orlando, FL. January, 2006. "What the Busy Pathologist Needs to Know about Running a Clinical Laboratory."

Tufts/New England Medical Center, Department of Pathology, Boston, MA. March, 2006. "Value Added Services in Laboratory Medicine."

Molecular Pathology Meets the Business of Pathology, Spring Conference of the Illinois Society of Pathologists, Oak Brook, IL. April, 2006. "Value Added Clinical Pathology Services: What they Are and Barriers to their Implementation."

College of American Pathologists Annual Meeting, San Diego, CA, September , 2006. "Practice Management Institute, Service and Quality."

College of American Pathologists Annual Meeting, San Diego, CA. September, 2006. "Whatthe Pathologist Director Should Be Doing."

Clinical Laboratory Improvements Act Committee Meeting, Atlanta, GA. September, 2006. "Narrative Interpretations of Complex Clinical Laboratory Evaluations".

Commencement address, Medical Professional Institute, Malden, MA. September, 2006.

The Ohio Society of Pathologists, Fall Meeting, Columbus, OH. October, 2006. "What We Are Doing or Should Be Doing in Clinical Pathology."

Visiting Professor, Ball Memorial Hospital, Department of Pathology, Muncie, IN. December, 2006. "Value-Added Services in Laboratory Medicine" and "Finding Your Place in the World of Pathology: Mentorship for Residents."

Visiting Professor, Case Western University Medical Center, Department of Pathology, Cleveland, OH. January, 2007. "Patient-Specific Expert Driven Narratives in Clinical Pathology– It's Time Has Come" and "Finding Your Place in the World of Pathology".

University Palermo, School of Medicine. Palermo, Italy. March, 2007. "Value Added Services in Clinical Pathology: A New Role for the Clinical Pathologist."

The Annual Israel Michaelson Distinguished Lectureship, The University of Tennessee Health Science Center, Department of Pathology and Laboratory Medicine, Memphis, TN. June, 2007. "What We Really Need to be Doing as Pathologists in Laboratory Medicine."

75th Annual Meeting of the American Society for Clinical Laboratory Sciences, Closing Keynote Address, San Diego, CA. July, 2007. "Going Beyond the Performance of the Laboratory Test: What the Patients Need that Only You Can Give Them."

College of American Pathologists National Meeting, Chicago, IL. October, 2007. Moderator and Speaker at Half-Day Workshop entitled "Practice Management Institute: Adding Value– The Indispensable Pathologist".

UCLA Medical Center, Department of Pathology Grand Rounds, Los Angeles, CA. October, 2007. "Providing Patient-Specific Narrative Reports for Complex Clinical Laboratory Evaluations: Why Can't We Do What the Radiologists and Anatomic Pathologists Do?"

Lab Institute 2007 G2 Reports, 25th Annual Meeting. Growing your Lab at the New Frontiers. Arlington, VA, October, 2007. "Current Critical Issues: Molecular Diagnostics Testing."

Opening Keynote Address, Business & Financial Strategies for Molecular Diagnostics, Sponsored by Washington G-2 Reports, Cambridge, MA. May, 2008. "Molecular Diagnostics: Growing at New Frontiers".

College of American Pathologists National Meeting, San Diego, CA. September, 2008. Moderator and Speaker at Half-Day Workshop entitled "Practice Management Institute: Adding Value– The Indispensable Pathologist".

Tennessee Chapter, American Society of Clinical Laboratory Scientists, Nashville, TN September, 2008. "Advising Clinicians on Test Selection and Result Interpretation".

13th Annual Anatomic Pathology Informatics (APIII), Pittsburgh, PA. October, 2008. "Why Not New Delhi? Is the Local Pathologist Dispensable?"

Keynote Address for the XXXVIII Mexico Congress on Clinical Pathology, Acapulco, Mexico. October, 2008. "The Indispensable Role of the Clinical Pathologist."

Cleveland Clinic, Pathology Grand Rounds as Visiting Professor, Cleveland, OH. March, 2009. "Reshaping Laboratory Medicine."

Keynote Address for American Society for Clinical Pathology Leadership Exchange, Philadelphia, PA. March, 2009. "Expanding your Role in Patient Care."

Keynote Address for Clinical Laboratory Scientists of Alaska, Anchorage, AK. May, 2009. "Going Beyond the Laboratory Test: What Patients Need that Only You Can Provide."

American Association of Clinical Chemistry National meeting, Chicago, IL. July, 2009. "The Indispensable Laboratory Director."

American Association of Clinical Chemistry Awards Banquet Speaker, Upstate New York section, Verona, NY. October, 2009. "The Role of the Laboratorian in Assisting Physicians in Test Selection and Result Interpretation."

Visiting Professor, Ball Memorial Hospital, Department of Pathology, Muncie, IN. January, 2010. "The Rapid Transformation of Diagnostic Services and the Creation of the Diagnostic Management Team."

Visiting Professor, St. Francis Hospital, Medical Grand Rounds, Hartford, CT. February, 2010. "Cost Effective Strategies for Clinical Laboratory Testing."

American Pathology Foundation Spring Conference, Las Vegas, NV. March, 2010. "Clinical Pathology: Overcoming Barriers to Mainstream Implementation."

Keynote Address for American Society for Clinical Laboratory Scientists– Tennessee Annual Conference, Nashville, TN. April, 2010. "Why Doctors Find it Difficult to Use the Clinical Laboratory Effectively."

FDA Symposium, Silver Spring, MD. May, 2010. "Cost Effective Laboratory Testing: New Programs to Help the Physician Choose the Right Tests and Correctly Interpret the Test Results."

American Association for Clinical Chemistry 2010 Annual Meeting, Anaheim, CA. July, 2010. "Interpretive Comments from the Clinical Laboratory: Essential for Patient Safety. Clinical Consultation by Laboratory Directors: Impact on Patient Safety and Outcome."

APIII and Lab Info Tech Summit 2010, Boston, MA. September, 2010. "Barriers to Clinical Lab Interpretive Reporting."

College of American Pathologists Annual Meeting, Chicago, IL. September, 2010. "Practice Management College: Adding Value – the Indispensable Pathologist."

Washington G-2 Reports, 28th Annual Lab Institute, Arlington, VA. October, 2010. "Getting the Best Results: an Evolving Role for Pathologists and Lab Directors."

Clinical Laboratory Improvements Act Advisory Committee (CLIAC) Meeting,Atlanta, GA. March, 2011. "Update on the activities of the Clinical Laboratory Integration into HealthCare Collaborative."

46th Academy of Clinical Laboratory Physicians and Scientists National Meeting, St. Louis, MO. June, 2011. "Managing Send out Costs – What Have You Tried, What Works and What Doesn't"

Keynote Address for Annual Meeting of the American Society for Clinical Laboratory Scientists (with Dr. James Meisel), Atlanta, GA. July, 2011. "Getting to the Right Diagnosis with Laboratory Tests as Fast as Possible – The Challenges and Essential Role of the Medical Technologist in the New Healthcare Environment."

American Association of Clinical Chemistry National Meeting (with Drs Julie Taylor, James Meisel, and Paul Epner), Atlanta, GA. July, 2011. "Opportunities for Improvement in Physician's Utilization of Laboratory Testing for Better Patient Outcomes."

College of American Pathologists National Meeting. Dallas, TX. September. 2011. "The Best of Futurescape" and "Antiplatelet Agents and Anticoagulants."

APIII and Lab Info Tech Summit 2011, Pittsburgh, PA. October, 2011. "Improving Laboratory Test Selection and Results Interpretation."

Diagnostic Error in Medicine 2011, 4th International Conference, Chicago, IL, October, 2011. "Error in Test Selection and Result Interpretation: A Major Source of Poor Patient Outcome."

Academy of Pathology and Laboratory Medicine of Puerto Rico Fall Meeting, Caguas, Puerto Rico. October, 2011. "Coagulation Disorders: Thrombosis", "Coagulation Disorders: Bleeding", "Consultation by Clinical Pathologists on Laboratory Test Selection and Results Interpretation"

2011 American College of Veterinary Pathologists/American Society for Veterinary Clinical Pathology Concurrent Annual Meetings. Nashville, TN. December, 2011. "Diagnostic Management Team for Coagulation Disorders: The Expert is Always Available."

Yale University School of Medicine, Department of Laboratory Medicine, Visiting Professor. January, 2012. "Improving Test Selection and Test Result Interpretation."

G2 Intelligence, Pathology Institute, 2012. Fort Lauderdale, FL. February, 2012. "It Is a New World for Pathology Organizations: Is Your Practice Advising Treating Physicians on Laboratory Tests Selection and Results Interpretation?"

POSTDOCTORAL RESEARCH FELLOWS SUPERVISED

Ann Marie Connor, M.D.	1986-1987 Young Investigator Award of the Academy of Clinical Laboratory Physicians and Scientists (1987)
E. Elizabeth Furth, M.D.	1987-1989 Awarded NIH Postdoctoral Fellowship Grant for 1988-89
Catherine S. Manno, M.D.	1987-1988
Laszlo Muszbek, M.D., Ph.D., D.Sc.	1987-1988, 1991, 1993, 1995 (Visiting Scientist on Sabbatical Leave)
Jun Teruya, M.D.	1989-1991
Youssef Hallaq, M.D.	1989-1991
Daniel Rubin, M.D.	1990-1991 Young Investigator Award of the Academy of Clinical Laboratory Physicians and Scientist
Claudia Villate, M.D.	1990-1991
Kathleen M. Doyle, Ph.D.	1991-1993
Zbigniew Szczepiorkowski, M.D., Ph.D.	1992-1995 Fellowship Award of the American Liver Foundation
Hazem Nouraldin, M.D.	1993-1994
Li Dan, Ph.D.	1994-1996
Elizabeth M. Van Cott, M.D.	1996-1997
Ewa Sicinska, M.D.	1996-1997
Rami Alharethi, M.D.	1997
Mohamed S. Kashalo, M.D.	1997-1999

Majed A. Refaai, M.D.	1997- 2003
Walter Zybko, Ph.D.	2000
Piyush Patel, M.D.	2001-2002
Lizzy Andrews, M.D.	2002-2003
Hasan Hasaba, M.D.	2002
Samir Aleryani, Ph.D.	2003-2004 (Fulbright Scholar)
Sadik Sharef, M.D.	2004
Ibrahim Batel, M.D.	2004
Miguel Adan, M.D.	2004
Bassel Ericsoussi, M.D.	2005–2006
Ragheed Alturkmani, M.D.	2005–2006
Hani Habal, M.D.	2006-2007
Bashar Ericsoosi, MD	2007
Kelly King, MD	2008 (Master of Science in Clinical Investigation Program)

RESEARCH STUDENTS SUPERVISED

Michael E. Arrasmith	1986-1987
Eric J. Hartman	1987-1988
Nicole D. Pilevsky	1988-1989
H Douglas Fleishman	1989-1990
Zbigniew Szczepiorkowski	1990
Ted Elvhage	1990
Ellen Villa	1991-1993
Kendrick Goss	1992-1993
Norbert Gorski	1992-1993
David A. Bird (Ph.D. program)	1992-1995
Salih Al-Salihi	1992-1995
Vickie C. Trace	1992-1994
David M. Dube′	1992-1994
Samir Lutf Aleryani	1992-1994
Mouris Saghir (Ph.D. program)	1993-1997

Christopher R. Morse	1993-1995
Agnieszka M. Heith	1993-1994
Ayman Kabakibi (Ph.D. program)	1994-1997
Paul Cannistraro	1994
Magda Szyfelbein	1994
Liana Vesga	1994
Thomas G. Bernhardt	1994-1995
Alexis F. Teplick	1995
Saloua Mokrim	1995
Arina Hadziselimovic	1996
Charlton K. Byun	1996
Emily Blodget	1997-1998
Elizabeth Gemba	1997
Azra Nanji	1997
Michael Walden	1998-1999
Rose Dhaliwal	1998-2000
Catherine Best (Ph.D. program)	1998-
Ali Hasaba (Ph.D. program)	1998-2002
Raneem O. Salem (Ph.D. program)	1998-
Britt L. Soderberg	1998-2000
Rhoni Patel	1998-1999
Simone Bethge	1999-2000
Veronia Proios	1999-2000
Steven J. Kirchner	1999
Katherine Marshall	1999
Joshua W. Russo	1999-2000
Katherine M. Szyfelbein	1999
Bryan R. Foster	2000
Miho Teruya	2000-2001
Ami Teruya	2000
Heather Magner	2000
Zia Khan	2000-2001
Joseph J. Bedway, Jr	2003
Rabie Alturkmani (Ph.D. program)	2002-2007
Khaled Alhomsi (Ph.D. program)	2002-2006
Juanito Savaille	2005-2007
Ryan Harrington	2007
Waddah Katrangi (Ph.D. program)	2008-
Brian McKenna	2008
Sarah Njoroge (Ph.D. program)	2008-
Rachel Lippert	2008
Twila Mason	2008
Obi Umunakwe (Ph.D. program)	2009

AWARDS, HONORS

1973	Phi Beta Kappa, Bucknell University

1974	Magna Cum Laude, Bucknell University
1974	Phi Sigma Award for Research in Biology, Bucknell University
1985-86	Faculty Honor Roll for Excellence in Teaching, University of Pennsylvania School of Medicine
1986-87	Louis R. Dinon Award for Excellence in Teaching, University of Pennsylvania School of Medicine
1987-88	Sheryl N. Hirsch Award of the Lupus Foundation of Philadelphia (included $10,000 for research support)
1987-88	Medical Student Government Basic Science Teaching Award, University of Pennsylvania School of Medicine (vote of graduating senior class selecting one basic science instructor)
1988-89	Christian R. and Mary F. Lindback Distinguished Teaching Award, University of Pennsylvania School of Medicine (two medical school faculty selected annually)
1989	University of Pennsylvania School of Medicine Commencement Speaker (one faculty member selected by vote of graduating class of 1989)
1992	Distinguished Service Award in Clinical Pathology bestowed by the Hungarian Society of Clinical Pathology
1996	Harvard Medical School Faculty Award for Distinguished Teaching to First Year Medical Students (Typically one awardee for each of the four years of medical school)
1996	Award for Distinguished Teaching to Pathology Residents, Massachusetts General Hospital
1997	The Stanley Wyman Award for Excellence in Teaching, Department of Medicine, Massachusetts General Hospital
1998	Award for Distinguished Teaching to Pathology Residents, Massachusetts General Hospital
1998	A. Clifford Barger Excellence in Mentoring Award, Harvard Medical School (Four awardees selected among hundreds of nominated faculty members)

1999	Award for Most Distinguished Teacher in the Preclinical Years, Harvard Medical School (One awardee by the vote of the graduating class)
1999	Award for Distinguished Teaching to Second Year Medical Students, Harvard Medical School
2000	"Mover and Shaker" Award, 5-7 leaders in the Laboratory Industry annually selected by a leading publication in the field.
2000	Award for Distinguished Teaching in the Preclinical Years, Harvard Medical School (Four awardees by the vote of the graduating class)
2000	Advocacy Award from the College of American Pathologists for Advocating Issues Beneficial to Patients, Clinicians and Pathologists
2001	Quest Distinguished Visiting Professorship of Pathology at the Johns Hopkins University School of Medicine
2002	Emma Sadler Moss Lectureship, Louisiana State Medical Center
2002	The John Figgs Jewett, MD, Memorial Lectureship, Massachusetts Medical Society for Maternal & Perinatal Welfare
2002	The Fourth Annual Frank M. Townsend MD Lecture, University of Texas, San Antonio
2002, 2003, and 2004	Invited by the Nobel Committee of the Karolinska Institute to nominate candidates for the Nobel Prize in Physiology or Medicine
2003	Award for Teaching in Pathology to Second Year Medical Students, Harvard Medical School
2004	Harry J. Sacks, MD Lectureship, Cedars-Sinai Hospital, Los Angeles, CA
2004	Abraham J. Gitlitz Memorial Lecture at 124th Annual Meeting of Association of Clinical Scientists, Houston, TX.
2004	Gerald T. Evans Award from the Academy of Clinical Laboratory Physical & Scientists (ACLPS) for distinguished service in the field of Laboratory Medicine, Presented at the 39th meeting of the society in Denver, CO.
2004	The Dr. R.E. Bell Memorial Lectureship, University of Alberta, Edmonton, Alberta, Canada.

2004	Recognition by Centers for Disease Control and Prevention as Founding Partner for Institute for Quality in Laboratory Medicine
2004	Nominee, Prize for Excellence in Teaching (Years 1 & 2), Harvard Medical School
2004	Award for Excellence in Teaching to Second Year Medical and Dental Students, Harvard Medical School and Harvard School of Dental Medicine
2005	Award from the Institute for Quality in Laboratory Medicine (IQLM) of the Centers for Disease Control and Prevention (CDC) for "Improved Clinical Integration" of Laboratory Medicine Services. One of 10 major awards in 2005 by the IQLM.
2005	Semi-finalist award for Massachusetts Institute of Technology 50K Entrepreneurship Competition (innovation team name is MedPacks).
2005	The Ward Burdick Award from the American Society of Clinical Pathology (ASCP) for Distinguished Service to Clinical Pathology.
2005	Award for Distinguished Teaching in the Preclinical Years (1 of 2 awardees selected by the graduating class), Harvard Medical School.
2005	J. Heinrich Joist First Memorial Lecture, St. Louis University Coagulation Consultants Symposium.
2005 - 2006 2006 - 2007 2007 - 2008 2008 - 2009 2009 - 2010 2010 - 2011	Elected to Best Doctors for expertise as a clinician in coagulation disorders (Physicians identify fellow physician experts for Best Doctors, Inc.)
2006 and 2007	Boston Magazine's Best of Boston Doctors (Top 100-200 local physicians among >5000 licensed doctors)
2006	Laboratory Public Service National Leadership Award from the Lab Institute at the 24th Annual Meeting of the Washington G2 Reports
2007	The Israel Michaelson Distinguished Lectureship, University of Tennessee Health Science Center, Memphis, TN
2009	American Association of Clinical Chemistry, National Award for Outstanding Contributions in Education
2009	The Norman P. Kubasik Lectureship Award, American Association of Clinical Chemistry Upstate New York section.

TEACHING EXPERIENCE

Medical School Biochemistry, teaching assistant, Johns Hopkins University, 1977-1980.

Medical School Pathology, lecturer, Washington University, 1983-1985. "Laboratory Diagnosis of Disease."

Laboratory Medicine, *Coursemaster* of third year medical student course in Laboratory Medicine and lecturer, Washington University, 1984-1985. Lecturer on "Cardiac Enzymes."

Medical School Pathology 100, lecturer, University of Pennsylvania, 1986-1989. "Hemostasis."

Medical School Pathology 200, lecturer, University of Pennsylvania, 1985-1989. "Laboratory Analysis of Cerebrospinal Fluid", "The Diagnosis of Myocardial Infarction", "Coagulation Factor Disorders", "Deep Vein Thrombosis and Pulmonary Embolism." Medical Student Introduction to Clinical Medicine, lecturer, University of Pennsylvania, 1985-1989, "Laboratory Diagnosis of Disease".

Clinical Coagulation, 20 lectures repeated three times/year to pathology residents and hematology fellows, Hospital of the University of Pennsylvania, 1985-1989.

Pathology 700, *Coursemaster* of one-month elective in Laboratory Medicine in which students rotate through each of the individual clinical laboratories, University of Pennsylvania, 1986-1989.

Medical School Biochemistry, Section leader of 4-session mini-course on Biochemical Aspects of Diabetes, University of Pennsylvania, 1988.

Pathology 305, *Coursemaster* of two-week elective entitled "The Effective Use of Laboratory Tests," University of Pennsylvania, 1989.

Clinical Coagulation, 5 - 10 lectures repeated 3 times/yr to pathology residents and hematology fellows, Massachusetts General Hospital, 1990-2007.

Laboratory Medicine, *Coursemaster* of third year medical student course in Laboratory Medicine for students on Internal Medicine Rotation and Lecturer, Massachusetts General Hospital, 1990-. Lectures on "Clinical Coagulation Testing" and "Cardiovascular Risk".

Laboratory Medicine Resident Lecture Series, Initiated and developed as *Coursemaster* the complete training program for residents in Clinical Pathology at the Massachusetts General Hospital. Areas of specialization include: 1) Clinical Chemistry and Immunology; 2) Microbiology; 3) Hematology/Coagulation/Blood Transfusion, 1990-.

Medical School Pathology, First Year Curriculum, Lecturer, Harvard Medical School, 1991-1992. "Prostaglandins, Thromboxanes, and Leukotrienes".

Clinical Hemostasis, 9 Lecture Course to Graduate Students of Northeastern University, 1992, 1994, 1996.

Medical School Pathology, Second Year Curriculum, Lecturer, Harvard Medical School, 1993. "Interpretation of Laboratory Tests."

Medical School Pathology, Lecturer, HST Program at Harvard Medical School, 1995- "Acute Inflammation" (1995-2007), "Thrombosis and Atherosclerosis (2004-2007)".

Immunity, Microbes, and Defense, First Year Curriculum, Lecturer, Harvard Medical School, 1994-1995. "Cell Injury" and "Acute Inflammation." Organizer of laboratory sessions in Clinical Pathology.

Introduction to Pathology, First Year Curriculum, Lecturer (4 lectures), Harvard Medical School, "Cell Injury," (1996-2003), "Inflammation" (1996-2006). Organizer (with a 1 hour introductory lecture) of laboratory session on "Diagnosis Using Clinical Laboratory Test Results," and "The Role of the Physician in Laboratory Medicine."

Clinical Commons, Third/Fourth Year Curriculum, Lecturer, Harvard Medical School, 1996-2003). "The Appropriate Use of the Clinical Laboratory."

Mechanisms of Diseases Processes, Pathology 210 for Graduate Students, Lecturer, Harvard Medical School, 1997. "Cell Injury" and "Inflammation."

Laboratory Medicine Elective for 3rd/4th Year Medical Students, on Selection and Interpretation of Laboratory Tests. Harvard Medical School, Coursemaster and Course Founder, 1997-.

Internal Medicine Resident Lecture Series, Lecturer, 1998-2007. "Bleeding Disorders" and "Thrombotic Disorders." 2006 – 2007 "Anticoagulants."

Current Concepts in Clinical Pathology, Harvard Continuing Medical Education Course, Lecturer and Course Co-Director, 2003 and 2005.

MGH Surgery Rotation for Harvard Medical Students, Lecturer, 2003 –2007. "Bleeding and Thrombotic Disorders."

Laboratory Medicine Resident Career and Management Conference, Lecturer, Massachusetts General Hospital. "How to Give a Talk" (2000-2007), and "Performing Laboratory Outreach Testing" (2000-2007), and "How to be a Laboratory Director" (2005-2007).

Laboratory Medicine Resident "Outs" Conference (2 – 4 presentations/yr), Massachusetts General Hospital, 2002 - 2007.

Harvard Health Sciences & Technology Program Biochemistry Course, Lecturer, 2003 - 2007. "Fatty Acids & Their Metabolites."

Harvard Health Sciences & Technology Program Elective in Pathology, Lecturer, 2005. "Laboratory Medicine and the Use of the Clinical Laboratory."

Scientific Symposiums, Course director and lecturer, 2005. Updates in Laboratory Medicine, Hilton Head, SC. Five presentations on clinical coagulation, lipid metabolism, and alcohol metabolism.

Applying Anatomic and Clinical Pathology to Reach a Diagnosis. Harvard Continuing Medical Education Course. Lecturer and course co-director. 2007.

Disease, Diagnosis, and Therapeutics, Vanderbilt University School of Medicine. Pathology group co-leader. 2008 – Present.

Pathology 351. Vanderbilt University School of Medicine. Interdisciplinary graduate program for PhD students. Lecturer (2008-11) and section leader (2008, 2010, and 2011) (cystic fibrosis) and 2009 (thrombosis).

Coagulation & Transfusion Medicine for Anesthesia Residents, Vanderbilt University Hospital. 2008 - 2010.

The Capstone Course: Basic Science for 4th Year Medical Students. Section Leader for "Thrombosis," with a presentation to entire class and organizer of 13 breakout sessions. Vanderbilt University School of Medicine, 2009 - Present.

Vanderbilt University School of Medicine Emphasis Program. (All medical students must perform a research project for graduation through this program): Advisor to 10 students and co-leader of laboratory based research section. 2009 – Present.

Molecular Foundations of Medicine. Lecturer, 2010. "Fatty Acid Metabolism," "Fatty Acids & Disease," "Phospholipids and Cell Signaling." 2010.

PUBLICATIONS

Articles

1. Laposata, M., S.M. Prescott, T.E. Bross, and P.W. Majerus. 1982. Development and characterization of a tissue culture cell line with essential fatty acid deficiency. Proc. Natl. Acad. Sci. USA 79, 7654-7658.

2. Laposata, M., D.K. Dovnarsky, and H.S. Shin. 1983. Thrombin-induced gap formation in confluent endothelial cell monolayers in vitro. Blood 62, 549-556.

3. Laposata, M., E.L. Reich, and P.W. Majerus. 1985. Arachidonoyl-CoA synthetase: separation from nonspecific acyl-CoA synthetase and distribution in various cells and tissues. J. Biol. Chem. 260, 11016-11020.

4. Majerus, P.W., E.J. Neufeld, and M. Laposata. 1985. Mechanisms for eicosanoid precursor uptake and release by a tissue culture cell line, in Inositol and Phosphoinositides: Metabolism and Regulation, Bleasdale, J.E., J. Eichberg, and G. Hauser, eds. 443-457.

5. Brass, L.F., M. Laposata, H. Singh, and S. Rittenhouse. 1986. Regulation of the phosphoinositide hydrolysis pathway in thrombin-stimulated platelets by a pertussis toxin-sensitive guanine nucleotide-binding protein. J. Biol. Chem. 261, 16838-16847.

6. Capriotti, A.M. and M. Laposata. 1986. Identification of variables critical to reproducible delipidation of serum. J. Tissue Cult. Methods. 4, 219-222.

7. Laposata, M. and P.W. Majerus. 1987. Measurement of icosanoid precursor uptake and release by intact cells. Methods in Enzymology. 141, 350-355.

8. Laposata, M., S.L. Kaiser, E.L. Reich, and P.W. Majerus. 1987. Eicosadiynoic acid: a non-toxic inhibitor of multiple enzymatic steps in the production of icosanoids from arachidonic acid. Prostaglandins. 33, 603-615.

9. Lowe, J.B., M. Sacchettini, M. Laposata, J.J. McQuillan, and J.I. Gordon. 1987. Expression of rat intestinal fatty acid-binding proteinin escherichia coli. J. Biol. Chem. 262, 5931-5937.

10. Brass, L.F. and M. Laposata. 1987. Diacylglycerol causes Ca release from the platelet dense tubular system: comparisons with Ca release caused by inositol 1,4,5-triphosphate. Biochem. Biophys. Res. Commun. 142, 7-14.

11. Hicks, D.G., A.M. Connor, and M. Laposata. 1987. Laboratory diagnosis and monitoring of disseminated intravascular coagulation. Lab. Med. 18, 585-589. (Reprinted in the Italian journal Biochimica Clinica 13, 30-34, 1989.)

12. Laposata, M., C.M. Krueger, and J.E. Saffitz. 1987. Selective uptake of [3H]arachidonic acid into the dense tubular system of human platelets. Blood, 70, 832-837.

13. Laposata, M., S.L. Kaiser, and A.M. Capriotti. 1988. Icosanoid production can be decreased without alterations in cellular arachidonate content or enzyme activities required for arachidonate release and icosanoid synthesis. J. Biol. Chem. 263, 3266-3273.

14. Connor, A.M. and M. Laposata. 1988. A rapid assay for thromboxane production by platelets and its use in assessing prior aspirin ingestion. Am. J. Clin. Path. 89, 216-221.

15. Capriotti, A.M., E.E. Furth, M.E. Arrasmith and M. Laposata. 1988. Arachidonate released upon agonist stimulation preferentially originates from arachidonate most recently incorporated into nuclear membrane phospholipids. J. Biol. Chem. 263, 10029-10034.

16. Furth, E.E. and M. Laposata. 1988. Mass quantitation of antagonist induced arachidonate release and icosanoid production in a fibrosarcoma cell line: Effect of time of agonist stimulation, amount of cellular arachidonate, and type of agonist. J. Biol. Chem. 263, 15951-15956.

17. Laposata, M., M. Minda, A.M. Capriotti, E.J. Hartman, and R.V. Iozzo. 1988. Reversible phenotypic modulation induced by deprivation of exogenous essential fatty acids. Lab. Invest. 59, 838-847.

18. Tate, G.A., B.F. Mandell, R.A. Karmali, M. Laposata, D.G. Baker, H.R. Schumacher Jr., and R.B. Zurier. 1988. Suppression of monosodium urate crystal induced inflammation by diets enriched with gamma linolenic and eicosapentaenoic acids. Arth. Rheum. 31, 1543-1551.

19. Muszbek, L. and M. Laposata. 1989. Glycoprotein Ib and glycoprotein IX in are acylated with palmitic acid through thioester linkages. J. Biol. Chem. 264, 9716-9719.

20. Hartman, E.J., S. Omura, and M. Laposata. 1989. Triacsin C: A differential inhibitor of arachidonoyl-CoA synthetase and nonspecific long chain acyl-CoA synthetase. Prostaglandins. 37, 655-671.

21. Remaley, A.T., Kennedy, J.M., and M. Laposata. 1989. Evaluation of the clinical utility of platelet aggregation studies. Am. J. Hematol. 31, 188-193.

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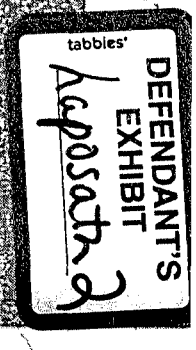
LAPASO



DIAGNOSIS OF CHILD ABUSE

- The medical literature contains many case reports in which child abuse was overdiagnosed in children with hemorrhagic coagulopathies
- A major concern is that overdiagnosis may be more common than is currently believed because of the high prevalence of von Willebrand's disease, which may be on the order of 1% in the general population

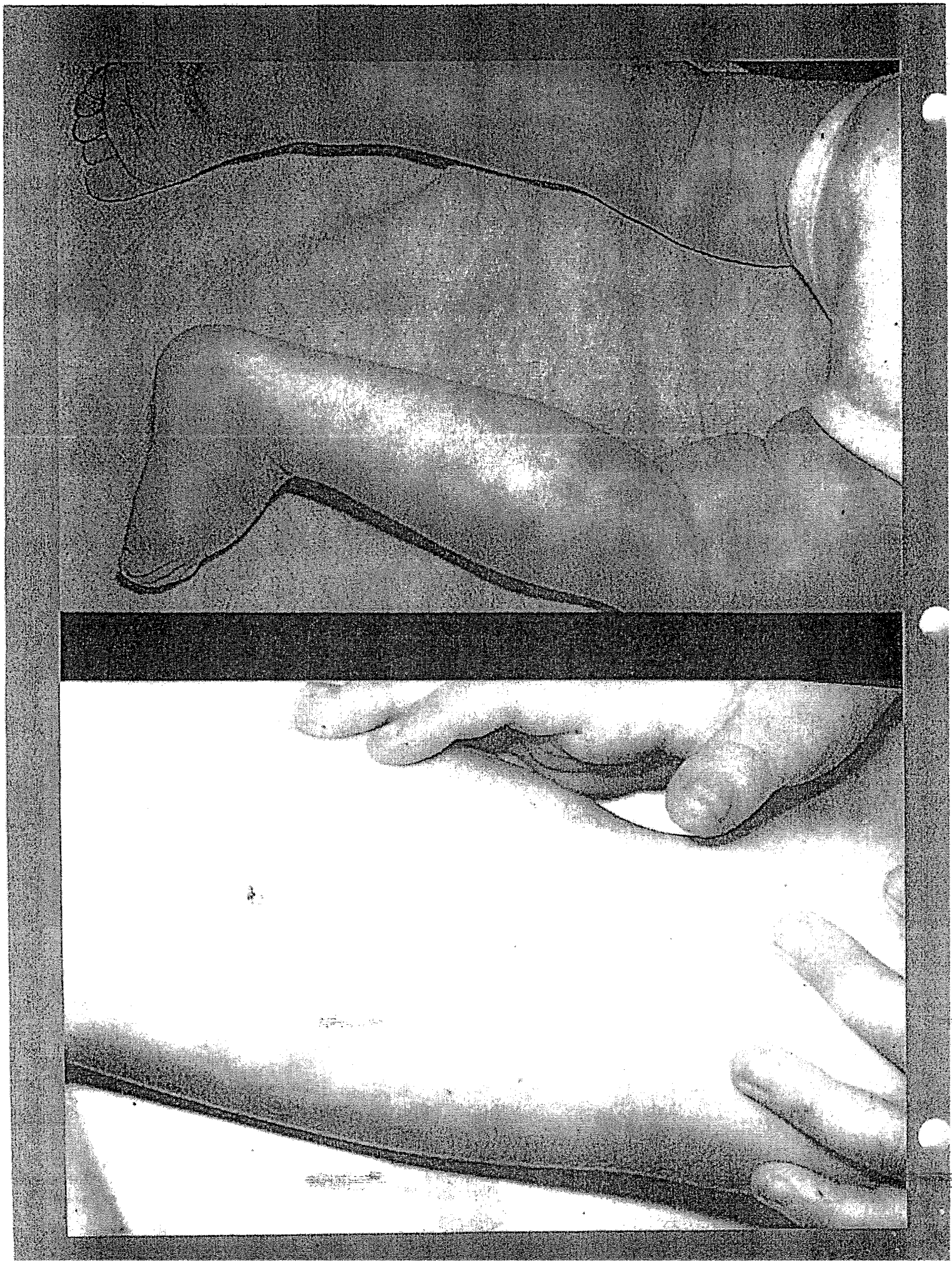
SYMPTOMS THAT SUGGEST CHILD ABUSE AND NONINFLICTED ENTITIES THAT MIGHT CAUSE THEM

Physical Symptom	Possible Noninflicted Cause
Bruises	von Willebrand disease
	Hemophilia A and B
	Idiopathic thrombocytopenic purpura
	Thrombocytopenia with lymphoblastic leukemia
	Vitamin K deficiency
	Purpura fulminans
	Meningitis with disseminated intravascular coagulation
	Hemorrhagic disease of the newborn
	Henoch-Schönlein purpura
	Ruptured subarachnoid vascular formation
	Blue spots malformation

Am J Clin Pathol 2005;123(Suppl 1):S119-S124

COMPARISON SLIDES OF CHILD ABUSE VS. COAGULOPATHY –

Which case is abuse and which case is a child with a bleeding disorder who experienced a minor injury?



DIAGNOSIS

Vascular Disorder

Female - Age 10 Months

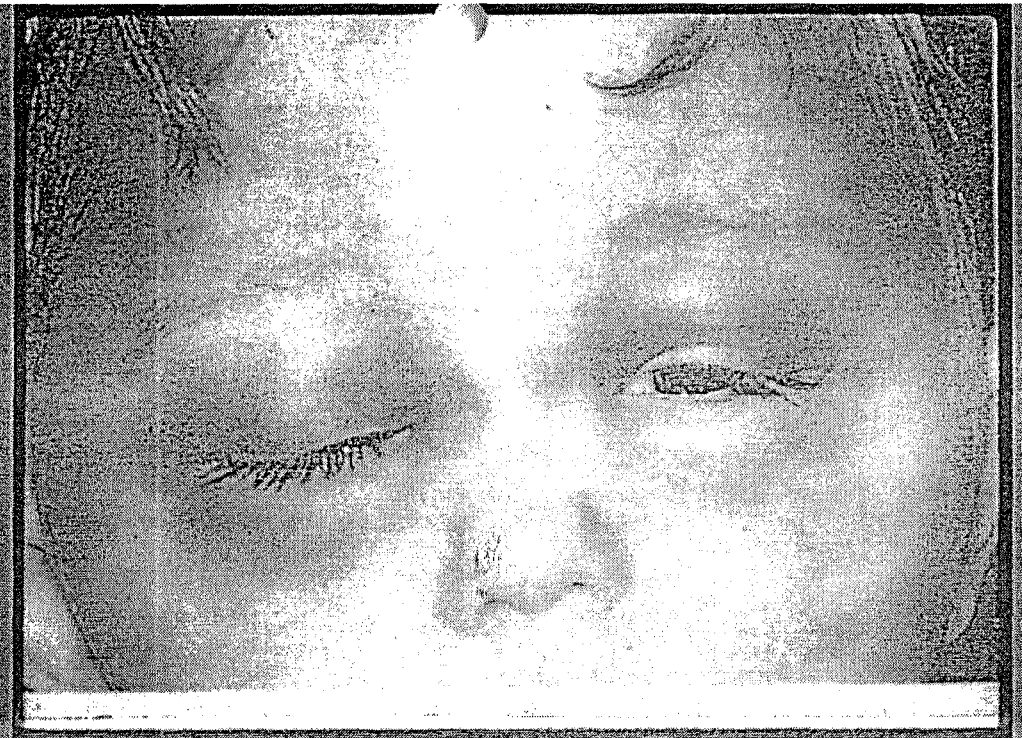
Extensive bruising on legs due to Henoch-Schönlein purpura

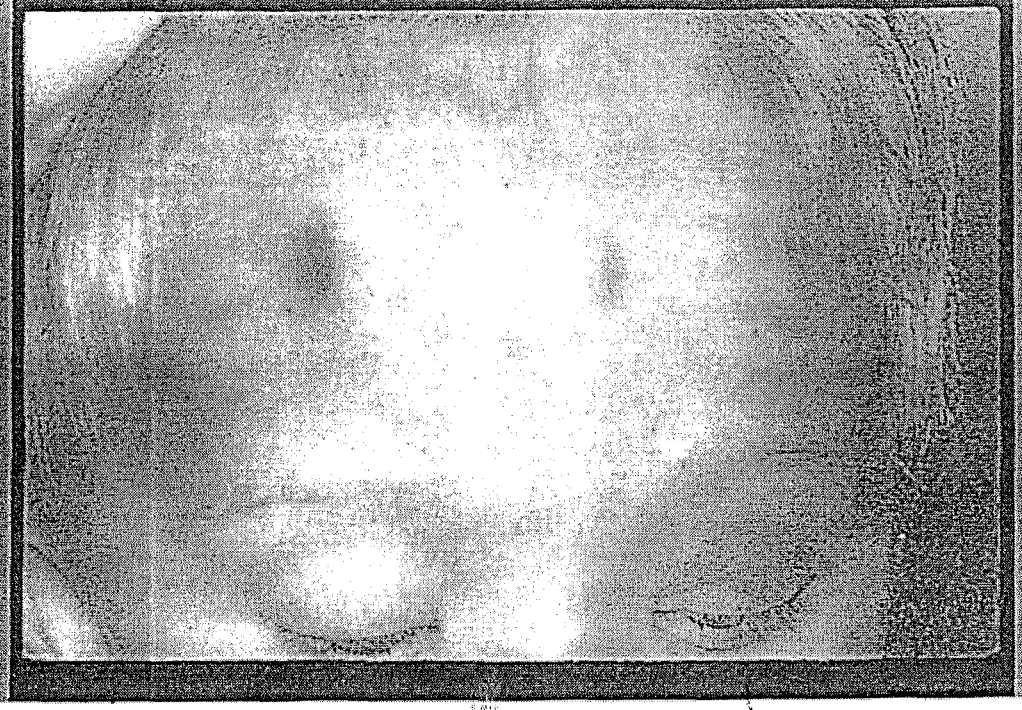
Child Abuse

Male - Age 4 Years

Cluster of roughly circular bruises on the inner aspect of the lower leg from grip marks

Physical Signs of Child Abuse, Saunders, p 32, p 108, 1996





DIAGNOSIS

Child Abuse

Male - Age 4 Years

Unexplained black eyes and multiple burns with swelling of the forehead and bilateral peri-orbital hematoma

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Coagulopathy

Male - Age 5 Years

Extensive large purple bruise in a well cared for child with a diagnosis of idiopathic thrombocytopenic purpura

Physical Signs of Child Abuse, Saunders, p 92, p 105, 1996

Michael Laposata, M.D., Ph.D.

Director of Clinical Laboratories
Massachusetts General Hospital
Professor, Harvard Medical School

LAPASOTA

COAGULATION TESTS

- ☐ Platelet Specific PLA-1 Antigen (526)
- ☐ Platelet Factor 4 (504)

Protein C

- ☐ *Activity (035)* ☐ *Antigen (036)*
- ☐ *Antigen/F VII Ratio (067)*

- ☐ Protein C Inhibitor (PAI-3)* (717)

Protein S

- ☐ *Activity (088)*
- ☐ *Antigen Total (038)* ☐ *Antigen Free (087)*
- ☐ *Antigen/F VII Ratio (059)*

Protein C and S

- ☐ *Activity (149)* ☐ *Antigen (142)*
- ☐ *Antigen/F VII Ratio (032)*
- ☐ *Activity and Antigen (204)*

- ☐ Proconvertin Prothrombin Assay (084)
- ☐ Prothrombin Consumption (PF3) (503)
- ☐ Prothrombin Fragment 1+2 (718)
- ☐ Prothrombin Time (080)
- ☐ Prothrombin Time Mixing Study (116)
- ☐ Reptilase Time (610)
- ☐ Stypven Time (611)
- ☐ Thrombin Time (807)
- ☐ Thrombin Time Mixing Study (813)
- ☐ Thrombin-ATIII Complex (714)
- ☐ Thrombus Precusor Protein* (209)
- ☐ Tissue Factor Pathway Inhibitor Ag* (147
- ☐ Tissue Plasminogen Act Antigen (125)
- ☐ Tissue Thromboplastin Inhibition (804)

von Willebrand Factor

- ☐ *Activity (114)* ☐ *Antigen (113)*
- ☐ *Multimers (117)*

- ☐ *F VIII Human (Bethesda) (701)*
- ☐ *F VIII Porcine Screen (703)*
- ☐ *F IX (Bethesda) (704)*

☐ Fibrin Monomer (202)

Fibrinogen

- ☐ *Activity (200)* ☐ *Antigen (199)*

☐ Fibrin(ogen) Degradation Products (201)

☐ Fibrinopeptide A (086)

Fletcher Factor

- ☐ *Prekallikrein Assay (121)*
- ☐ *Prekallikrein Screen (120)*

☐ Heparin Adsorption of Plasma (135)

Heparin Anti-Xa Assay

- ☐ *Unfractionated (600)*
- ☐ *LMWH (602)*

☐ Heparin Cofactor II* (133)

Heparin-Induced Antibody

- ☐ *Antibody* (522)* ☐ *Antibody Titer* (528)*

☐ Heparin Solution Quantitation (139)

☐ Hexagonal Phospholipid Neut. (144)

☐ High Mol Wt Kininogen Assay (123)

☐ Homocysteine (Serum) (727)

☐ Homocysteine (Urine) (729)

☐ Kaolin Clotting Time (056)

☐ Lipoprotein(a)* (715)

☐ Plasminogen Activator Inhibitor-1 (126)

☐ Plasminogen Activator Inhibitor-2* (140)

☐ PIVKA-II* (726)

Plasminogen

- ☐ *Activity (400)* ☐ *Antigen (408)*

☐ Platelet Neutralization Procedure (805)

Platelet Antibody

- ☐ *Direct (523)*
- ☐ *Screen (520)* ☐ *Platelet Specific (524)*

- ☐ Activated Protein C Resistance (716)
- ☐ alpha-2-Antiplasmin Assay (039)
- Anticardiolipin Antibody
  - ☐ *IgG, IgM (034)* ☐ *IgA (164)*
- ☐ Antiphosphatidylserine (153)
- Antithrombin
  - ☐ *Activity Plasma (030)*
  - ☐ *Antigen Plasma (033)*
  - ☐ *Activity Serum (031)*
- ☐ APTT (040)
- ☐ APTT Mixing Study (806)
- ☐ beta-Thromboglobulin (085)
- ☐ C4b Binding Protein* (160)
- ☐ Cryofibrinogen (203)
- D-Dimer
  - ☐ *Quantitative(405)*
  - ☐ *Semiquantitative (404)*
- ☐ Dilute Russell's Viper Venom Test (057)
- ☐ Euglobulin Lysis Time (401)
- Factor Activities
  - ☐ *F II (100)*
  - ☐ *F V (101)* ☐ *F X (105)*
  - ☐ *F VII (102)* ☐ *F XI (106)*
  - ☐ *F VIIa* (activated Factor VII) (111)*
  - ☐ *F VIII (103)* ☐ *F XII (107)*
  - ☐ *F IX (104)* ☐ *F XIII* (108)*
- Factor Antigens
  - ☐ *F VII* (112)* ☐ *F IX (205)*
  - ☐ *F X* (206)*
- ☐ Factor V Mutation (Leiden) (719)
- ☐ Factor VIII Concentrate Quantitation (058)
- Factor Inactivators
  - ☐ *Inhibitor/Inactivator Screen (700)*
  - ☐ *F V (Bethesda) (706)*
  - ☐ *F VIII Porcine (Bethesda) (702)*

1 Check in Box for Prolonged PTT Panel
Initiates Use of This Test Selection Algorithm

Prolonged PTT Evaluation

Degrade heparin in sample and repeat PTT - if the PTT normalizes, heparin is the cause

↓

PTT mixing study (1:1 mix of patient:normal plasma)

↙ PTT Normalizes

↓

Factor deficiency- measure factors VIII, IX, XI, and XII

↘ PTT remains prolonged

↓

Inhibitor, most commonly Lupus anti-coagulant; may be a Factor VIII inhibitor if PTT mixing study first normalizes and then becomes prolonged

↓

Perform tests for specific inhibitors suggested by results of PTT mixing study

MGH experience with detectable errors in test selection by clinicians

Test selection mistakes in coagulation by MGH and non-MGH clients in January 2003 are only 2-3 per week and include –

Ordering Factor V instead of APC resistance to screen for Factor V Leiden

Ordering Factor X instead of Anti-factor Xa or chromogenic Factor X

Ordering tests for both bleeding and thrombosis when only one condition is present

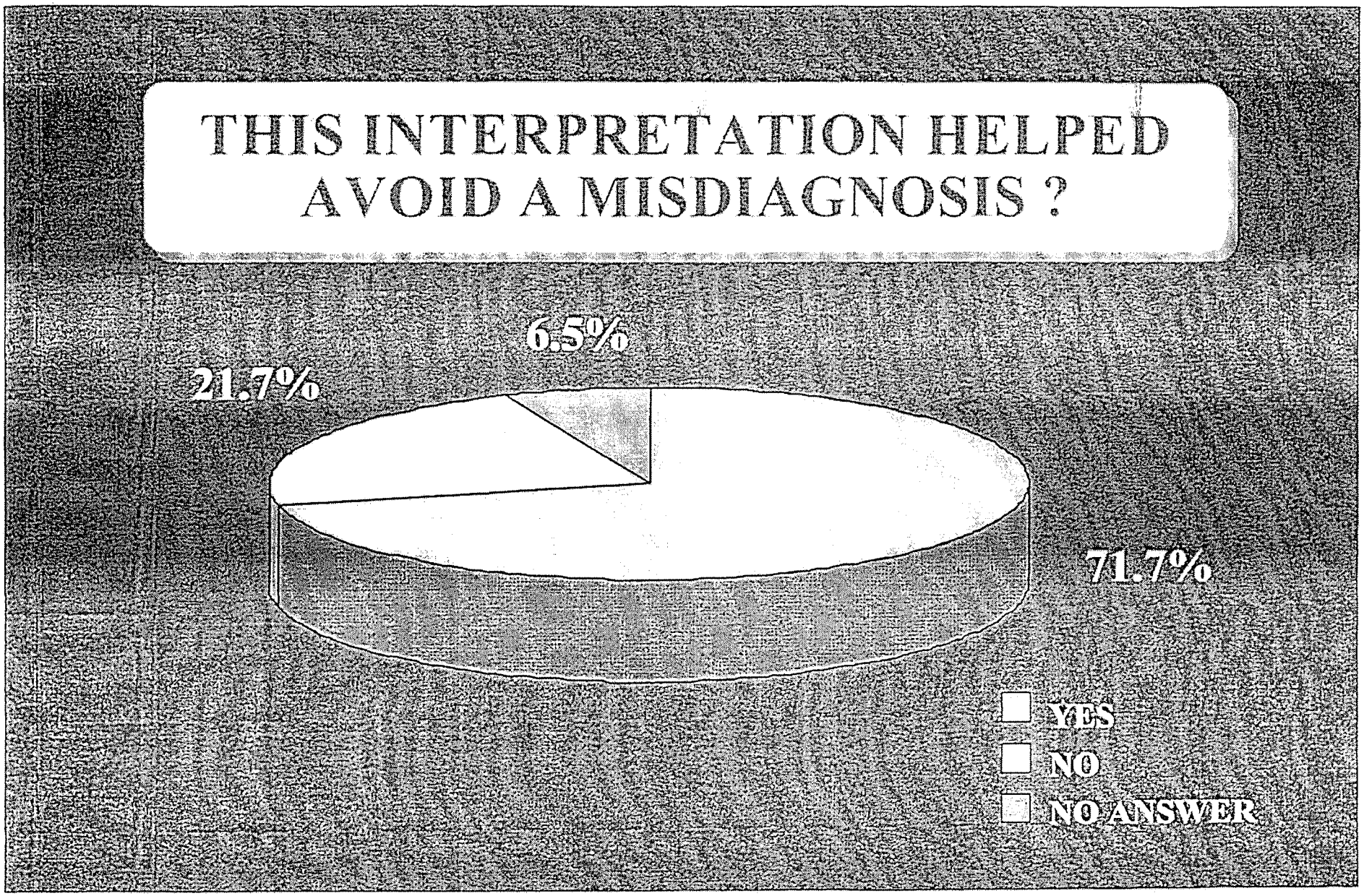
1996 Survey of MGH physician experience with narrative interpretations of complex laboratory evaluations in coagulation

Ordering physicians sent a narrative interpretation of one their own cases

Clinicians asked to respond to several questions about the interpretation

46 0f 100 surveys returned

THIS INTERPRETATION HELPED AVOID A MISDIAGNOSIS ?

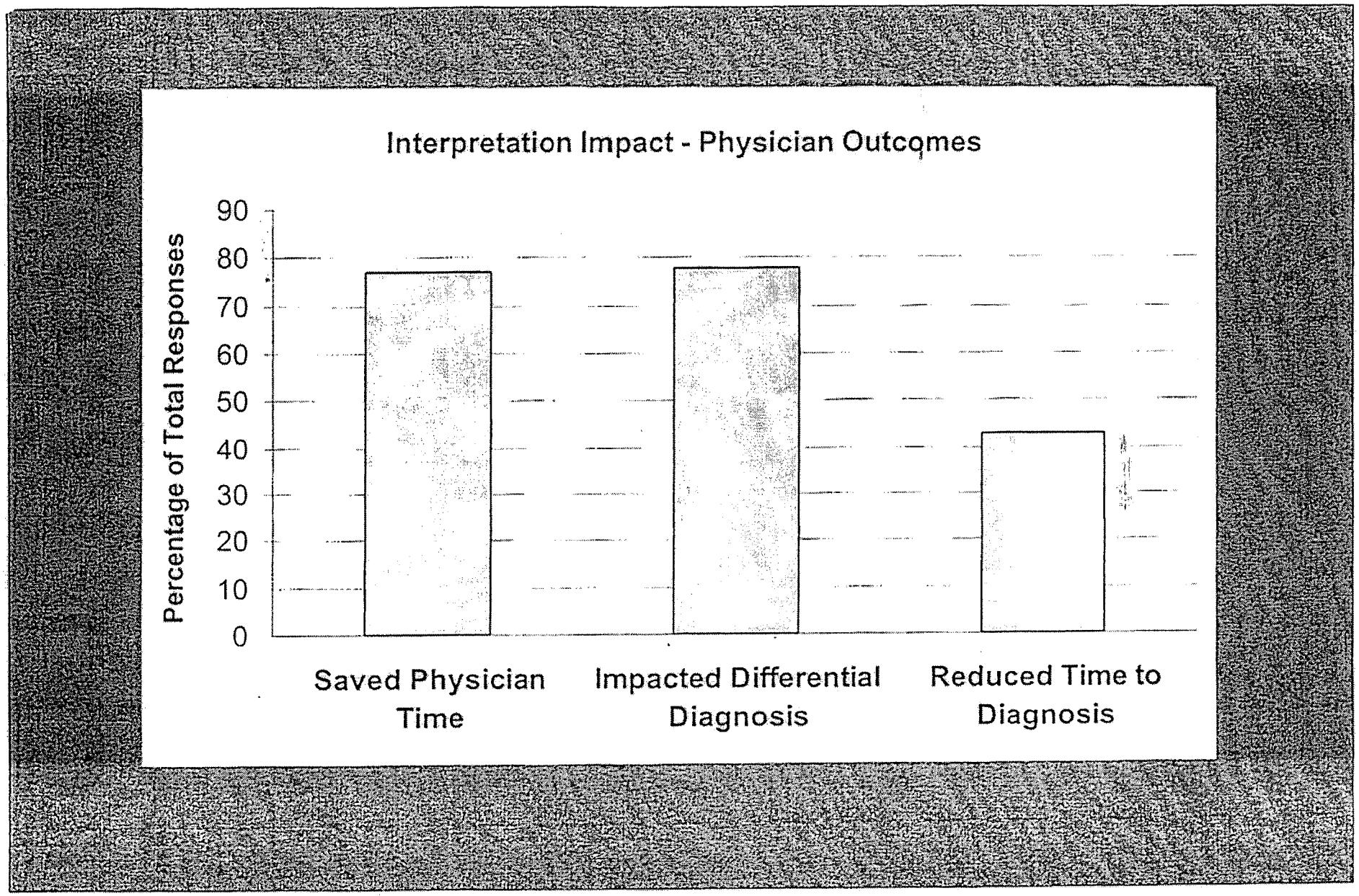


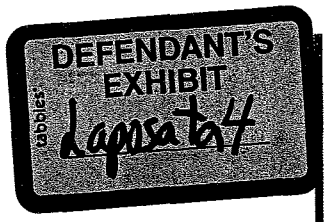
2000 Survey of MGH physician experience with narrative interpretations of complex laboratory evaluations in coagulation

Ordering physicians electronically sent a narrative interpretation of one their own cases

Clinicians asked to respond electronically to several questions about the interpretation

100 of 100 surveys returned


Interpretation Impact - Physician Outcomes
Percentage of Total Responses
90
80
70
60
50
40
30
20
10
0
Saved Physician Time
Impacted Differential Diagnosis
Reduced Time to Diagnosis



Children With Signs of Abuse

When Is It Not Child Abuse?

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Key Words: Child abuse; Hemophilia; von Willebrand disease; Misdiagnosis; Coagulopathies; Shaken baby syndrome; Idiopathic thrombocytopenic purpura; Bruising; Bleeding disorder; Medical error

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Abstract

Child abuse is a problem that is frequently underdiagnosed. Recognition that underdiagnosis of abuse exists has produced a high zeal for identifying cases of child abuse, which has inevitably produced cases of overdiagnosis. Overdiagnosis of child abuse is as catastrophic as underdiagnosis. In this case, a family member is often accused of injuring or killing a child at a time when the loss is felt most deeply. This review focuses on 1 specific presentation of child abuse—the child with bruises and bleeding. Many children and adults have coagulation or vascular disorders that predispose them to bruise or bleed excessively with minor trauma. It is very easy for a health care worker to presume that bruising and bleeding is associated with trauma, because the coagulopathies that may explain the findings are often poorly understood. The clinical cases reviewed in this article show the need for an extremely thorough analysis for an underlying bleeding disorder in the bruised or bleeding child being evaluated as a possible victim of child abuse.

Correctly distinguishing between accidental and nonaccidental injuries in children has critical ramifications. An overview of the literature regarding injuries in children indicates that many underlying diseases often mimic child abuse and that although there are some published guidelines to help clinicians evaluate social conditions that might suggest child abuse,[1] there are not standard published guidelines to evaluate a wide range of clinical conditions that mimic and likely rule out child abuse. Certainty in these cases is a difficult goal; however, overdiagnosis and underdiagnosis of child abuse have devastating effects, not only for the child, but also for the entire family. Understanding the various ways these diagnoses can be reached incorrectly is critical to gaining better outcomes. Following is a review of many cases described in the literature, and 2 others in our case experience not yet published, that were misdiagnosed as child abuse, with information on how the correct diagnoses were missed. Because bruising is among the most common reasons a child may be evaluated for child abuse, a suggested standard evaluation for coagulopathies when a child has bruising and is being evaluated as a potential abuse victim is also presented.

Sadly, many forms of child abuse exist. The National Clearinghouse on Child Abuse and Neglect Information, an office of the US Department of Health and Humans Services, categorizes abuse as follows: neglect, physical, sexual, and other (which includes verbal and emotional abuse). This review involves cases of suspected physical abuse and focuses on bleeding and bruising. *Physical abuse* is defined as "inflicting a non-accidental physical injury upon a child. This may include burning, hitting, punching, shaking, kicking, beating, or otherwise harming a child."[2] In 1997, a special report from the Federal Interagency Forum on Child and Family Statistics presented statistics indicating that in 1993, 381,700 (or 5.7 per 1,000) children were

abused physically out of approximately 1.6 million children who were abused or neglected.[3]

We found it impossible to find statistics on the number of child abuse cases that are overdiagnosed or underdiagnosed. Anecdotal evidence appearing in the form of case reports is the only form of information indicating that both types of outcomes occur. The path to correct diagnosis is not easy. The sheer size of the medical knowledge base necessary to rule out other noninflicted possibilities and the nature of the legal system create impediments. In addition, the suspicion aroused by some presenting symptoms, in conjunction with "early evidence of psychosocial problems" in the family, may mislead a clinician into "protecting" the child and making "an erroneous early conclusion which may later adversely affect the physician's ability to intercede for the benefit of the patient and family."[4]

The broad categories of symptoms observed in an emergency department or in a physician's office that might indicate physical abuse include burns, broken bones, and/or bleeding and bruising. Unfortunately for the clinician evaluating these types of symptoms there are many noninflicted causes, ranging from the common to the very uncommon, that must be ruled out.

Several signs and symptoms that can be produced by assault along with selected disorders that can mimic them are shown in **Table 1**. For bone fractures, a review of the literature showed the following disorders can mimic child abuse: osteogenesis imperfecta, metabolic bone disease, congenital insensitivity to pain, metabolic bone disease, multifocal infection, infantile cortical hyperostosis, clavicle injured at birth, scoliosis, osteomyelitis, congenital hydrocephalus, Caffey disease, and osteoporosis.[5-7] Impetigo can mimic cigarette burns. Impetigo is common, and cigarette burns are not. Scalding, dermatitis, chilblains, drug eruption, mechanical abrasion, and accidental exposure to commercial grade vinegar are reported in the literature as being mistaken for child abuse.[5]

Bleeding and bruising can be especially complicated to understand when there is suspicion of physical abuse. Certainly it is true that a child with a bleeding disorder also might be abused physically; however, a child with bruising needs to be evaluated carefully so that any question of abuse is considered in the context of a bleeding disorder that might be present. Bleeding disorders are common, affecting more than 1% of the population.[8] They often are inherited and misunderstood. If a genetic component exists with a mild bleeding disorder, the family is likely to consider excess bleeding (frequent nosebleeds, for example) normal and might not provide this information to a clinician seeking family history. Such information, if understood, could direct the clinician to evaluate the child for such a hemorrhagic disorder.

Table 1

Symptoms That Suggest Child Abuse and Noninflicted Entities That Might Cause Them[5-7]

Physical Symptom	Possible Noninflicted Cause
Bone fractures	Osteogenesis imperfecta
	Metabolic bone disease
	Congenital insensitivity to pain
	Metabolic bone disease
	Multifocal infection
	Infantile cortical hyperostosis
	Clavicle injured at birth
	Scoliosis
	Osteomyelitis
	Congenital hydrocephalus
	Osteoporosis
Burns and scalds	Impetigo
	Dermatitis
	Chilblains
	Fixed drug eruption
	Mechanical abrasion
	Accidental exposure to commercial grade vinegar
Bruises	von Willebrand disease
	Hemophilia A and B
	Protein S and C deficiencies
	Idiopathic thrombocytopenic purpura
	Thrombocytopenia with lymphoblastic leukemia
	Vitamin K deficiency
	Purpura fulminans
	Meningitis with disseminated intravascular coagulation
	Hemorrhagic disease of the newborn
	Henoch-Schönlein purpura
	Ruptured subarachnoid vascular formation
	Blue spots malformation

A myriad of coagulopathies exist that can mimic child abuse. Unfortunately, cases are found in the literature involving families that have undergone the severe torment of being mislabeled as child abusers. von Willebrand disease affects approximately 1% to 2% of the population. Hemophilia A and B affect approximately 1 in 10,000 individuals.[8] Both of these diseases have been mistaken as child abuse. Idiopathic thrombocytopenic purpura, thrombocytopenia with lymphoblastic leukemia, vitamin K deficiency, purpura fulminans, meningitis with disseminated intravascular coagulation (DIC), hemorrhagic disease of the newborn, Henoch-Schönlein purpura, and ruptured subarachnoid vascular malformation also appear in the literature misdiagnosed as child abuse.

Review of Cases of Bleeding and Bruising Masquerading as Child Abuse

Each of the cases that follow were found to not be child abuse because subsequent questioning, after the family was facing prosecution, revealed the true cause of the signs and symptoms.

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von Willebrand Disease

A 3-month old was brought to the hospital after 2 days of evaluation, first at the pediatrician's office and then at another hospital, with seizures, bilateral retinal hemorrhages, subdural and subarachnoid hematomas, a history of easy bruising, with no apparent bruising at the time of admission anywhere on the trunk, back, or arms. The father reported that he had dropped the child while feeding her a bottle the night before and caught her and pulled her upward sharply before she struck the floor.

An initial review for coagulopathies was performed. von Willebrand disease, thrombocytopenia, and disorders associated with a prolonged prothrombin time (PT) and prolonged partial thromboplastin time (PTT) were ruled out. The physician interpreting the test results was unaware that the trauma of a fall, such as the one described by the father, could increase a low value for von Willebrand factor and for ristocetin cofactor well into the normal range.

The father was arrested for shaking his child and sent to trial. The mother was charged with neglect for leaving the child with the father, who had no violent or untoward history. The child was put into foster care and suffered continued bruising. At 6 months, the child was admitted to another hospital by the foster parent; the child had meningitis and a subdural hematoma. The bleeding history triggered tests for coagulopathies after the infection subsided. The child was diagnosed with moderately severe von Willebrand disease.

The prosecutor and states' witnesses, with virtually no expertise in coagulation disorders, insisted that the presence of a moderately severe bleeding disorder in the absence of bruises on the trunk, arms, or shoulders did not rule out shaken baby syndrome. The prosecutor argued that the literature was devoid of cases citing misdiagnosis of child abuse with underlying von Willebrand disease in a child with the exact constellation of presenting symptoms found in this child. The prosecutor further argued that retinal hemorrhages are pathognomonic of shaken baby syndrome. Cases of spontaneous bleeding in the head or retina with von Willebrand disease were considered completely irrelevant. The father was convicted and incarcerated. The child has largely recovered. Treating physicians at the hospital where the meningitis and von Willebrand disease were diagnosed thought that the clinicians who diagnosed shaken baby syndrome were overzealous in their pursuit of the abuse diagnosis.

It is worth reviewing the specifics of shaken baby syndrome because it is largely a diagnosis determined by particular presentations of bleeding. Caffey[9] introduced the whiplash shaken baby syndrome, now referred to as shaken baby syndrome, as a diagnosis in 1972. The disproportionate size of the infant head and weak neck muscles allow for the signs and symptoms of this condition. This syndrome generally refers to a constellation of symptoms that includes subdural and/or subarachnoid hemorrhage and bilateral retinal hemorrhages.[10]

Two issues must be considered when making this diagnosis. First, the bilateral retinal hemorrhages might not be indicative of shaken baby syndrome directly, as previously thought, but might be a result of increased intracranial pressure.[10] Increased intracranial pressure can result from hemorrhage from major trauma to the head but also from other causes. The minor trauma reported by the father in this patient with von Willebrand disease could explain the results. The slow pooling of blood in the subdural hematoma in this case was highly consistent with the presence of von Willebrand disease, which would permit oozing of blood into the subdural hematoma, increased intracranial pressure, and resultant retinal hemorrhages.

Diseases exist in which noninflicted events have generated unilateral and bilateral retinal hemorrhages. These diseases include retinopathy of prematurity, Coats disease, anoxia, cytomegalovirus, herpes simplex, endocarditis,[11] X-chromosome–linked juvenile retinoschisis,[12] aplastic anemia,[13] and von Willebrand disease.[14] It is especially troubling that the concept persists that retinal hemorrhages are pathognomonic of shaken baby syndrome, because with clinicians who believe this to be true, the patient and his or her family have almost no opportunity to pursue the diagnosis of an underlying disease.

A similar case involved a 1 year-old child who reportedly was playing with his 2 older siblings on a bed and fell head first a few feet onto a hardwood floor. The child suffered a subdural hematoma and subsequently developed bilateral retinal hemorrhages. Despite having cared for his children alone, daily, in the evenings while the mother was working for several years, it was determined the father had assaulted the child, and he was accused of attempted murder. The initial evaluation for a bleeding disorder did not include any testing for von Willebrand disease. Evaluation of the child (twice) and his 2 older siblings (once) later revealed a diagnosis of von Willebrand disease in the child and in both siblings. When these test results were obtained, at least 6 months after the event, the charges against the father were dropped. It should be noted that the first evaluation for von Willebrand disease of the child with trauma was performed at a time when the child had rhinorrhea and an elevation of acute phase reactants, one of which is von Willebrand factor. In this evaluation, the child had values that were essentially normal. If it had not been recognized that the von Willebrand factor level was likely to be much lower at baseline, the repeated study that established the diagnosis would not have been done.

Hemophilia

In 2003, a 7-month-old child was brought to the hospital by his single mother. The child was reported to have hit his

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head on a wall after a fall from his cradle while under the care of a sitter 2 weeks earlier. On the day before admission, the child became somnolent and had 2 episodes of vomiting. A computed tomography scan showed disjunction of the left lambdoid suture, a left parieto-occipital epidural hematoma, and a suggestion of bifrontal cortical atrophy. The child underwent a left parieto-occipital craniotomy. During surgery, a large epidural hematoma was drained and a left occipital fracture was found. No fractures were found on radiographs of the long bones. A diagnosis of child abuse was registered. Two weeks later, the child was brought back to the hospital for dehiscence of the surgical wound from drainage of a large subgaleal hematoma. While the child was still hospitalized, another subgaleal hematoma formed at the same site. Coagulation testing was performed, hemophilia A was diagnosed, and child abuse was excluded.[15]

Wheeler and Hobbs[5] reported a case of a 3-year-old Asian child who was referred to the hospital for excessive bruising. The child subsequently was diagnosed with hemophilia A.[5]

Schwer et al[4] described a case of a 10-month-old child with severe bruising over all portions of his body and a healing clavicle fracture. The family had no explanation for the bruising and fracture. Child abuse was suspected until the PTT was found to be abnormal. The child abuse diagnosis was dropped when hemophilia was diagnosed.[4]

Idiopathic Thrombocytopenic Purpura

in 1997, Harley[16] reported a case of a 2-year-old with a 2-day history of unexplained bruising. A teacher reported the child's bruises to the police, and the child was taken by the police to an emergency department where no tests were done. The child was put into protective custody. The following day, the child was seen by another physician who observed petechiae scattered all over the body and multiple bruises ranging up to 6 cm. A CBC count was ordered. Idiopathic thrombocytopenic purpura was diagnosed, and the child was sent home after being stabilized at the hospital.

Late Hemorrhagic Disease of the Newborn

A 10-week-old child was brought to the emergency department, comatose and hypotensive, with a bulging fontanelle, large bruises over her buttocks and thighs, bilateral multiple hemorrhages, severe cerebral edema, subdural and subarachnoid bleeding, and an extensive gluteal intramuscular hematoma; brainstem reflexes were absent. She also had an elevated PT and a low factor VII level. She was diagnosed with nonaccidental injury. The coagulopathy initially involving the low factor VII level was attributed to the severe head injury because such an injury can produce DIC that lowers the factor VII level. The initial conclusion was that the child was physically assaulted. However, as further medical history was obtained, it was realized the child had never received vitamin K prophylaxis as a newborn, which also can result in a low factor VII level. Autopsy confirmed late hemorrhagic disease of the newborn.[17]

Vitamin K Deficiency

A 4-month-old brought to the emergency department was pale, emaciated, and irritable with severe developmental delay, recurrent episodes of vomiting and diarrhea associated with frequent changes of formula, and multiple bruises. Laboratory analysis showed DIC with sepsis and vitamin K deficiency. The child received vitamin K, and the DIC resolved as the sepsis was treated effectively. A casein hydrolysate–sucrose formula was introduced, and the child's general condition improved markedly. The parents were perceived to be poorly educated, in poverty, inexperienced as parents, with family environmental isolation. Child neglect was diagnosed. By chance, a salty taste from the child's skin was noted. A diagnosis of cystic fibrosis with vitamin K deficiency secondary to the pancreatic and gastrointestinal disturbances of cystic fibrosis was made, and the child neglect diagnosis was abandoned.[18]

Thrombocytopenia With Lymphoblastic Leukemia

A 2-year-old child was found unresponsive by her mother, who with her boyfriend, rushed the child to the hospital where she died. The child had numerous bruises of various ages on her back and extremities, and the police were notified. Petechiae were present on her face, chest, abdomen, and labia majora. Focal hemorrhages existed on the anal mucosa. A previous police report existed from an anonymous caller noting the child had been seen at a restaurant with numerous bruises. It was thought that the boyfriend was responsible for the significant bruising. Autopsy revealed that the child had undiagnosed, untreated lymphoblastic leukemia, and the bruising and hemorrhages were explained by the low platelet count associated with her leukemia.[19]

Henoch-Schönlein Purpura

A case report appeared in 1998 by Daly and Stegel[20] of a 3-year-old brought to an emergency department with multiple bruises on the buttocks and lower extremities. The child lived with her mother, sibling, and mother's boyfriend. The mother had a history of substance abuse. The sibling had cerebral palsy and severe developmental delay, allegedly from shaken baby syndrome. With the exception of a 2-day history of nausea and vomiting the week before, the medical history of the child was unremarkable. Swelling and tenderness were apparent over the left eye and on the right knee. The patient was believed to have nonaccidental injuries, and child protective services was contacted. The child was put into the care of a relative. During the next 2 days, more

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ecchymoses appeared, and this time some of the purpuric lesions were palpable. Henoch-Schönlein purpura was diagnosed. The child was returned to the mother, and more lesions continued to appear. The child was returned to the hospital for further evaluation, and Henoch-Schönlein purpura was diagnosed again. The author of the case report was impressed by the hospital's persistent efforts to prove child abuse on the second visit, despite a clear diagnosis that explained the bruising.[20]

Ruptured Vascular Malformation

In some cases, the cause of bleeding is not related to anything in the flowing blood, but instead to a blood vessel that has ruptured. An example of noninflicted injury masquerading as shaken baby syndrome appeared in a 1995 report by Weissgold et al.[21] This case involved a child with an acute intracranial hemorrhage and diffuse cerebral edema, coupled with optic nerve sheath hemorrhages. The parents were perceived to be remarkably stoic throughout the admission and death of the child, and because of this, the pediatrician became suspicious that they had abused their child. When the child died, prosecutors were anxious to charge the parents criminally for shaken baby syndrome but waited for the completion of an autopsy. The autopsy demonstrated an unusual vascular malformation that had ruptured.[21]

Legal System

If the clinician has been unable to rule out noninflicted sources of injury to a child with the expertise available at the time, "the dye becomes set and all the processes connected with the state system through which children are protected follows."[22] It is crucial, therefore, that the process of evaluation be as complete as possible and include a requirement to rule out other entities, which might not be within the expertise of the evaluating physician or local experts, that can mimic child abuse.

At the point the case is reported, the legal and medical systems merge in an effort to sort out the evidence as fairly as possible, with maximal "protection" given to the child. Unfortunately, the 2 systems were not designed to work together all that well. Many issues related to jurisprudence inhibit the sharing of information, while the medical community optimizes clinical outcome by information sharing. The problem becomes apparent in the evaluation of child abuse when the treating physician is unable to discuss the case with experts brought by the defense who indeed might have specialized knowledge not available to the physician making the diagnosis of child abuse.

Because several clinical entities that can mimic child abuse are uncommon and many more are rare, the likelihood that a primary care physician or a local specialist could miss one of these diagnoses is not small. Worse still, the practices in the courtroom to challenge the testimony of the opposing witness in an effort more to win the case than to reveal the true circumstances make many expert physicians, who would provide the best insights, unwilling to step forward. Juries for any case are not composed of medical experts, and their ability to determine the credibility of one physician over another often depends highly on the skills of particular attorneys involved. In addition, the expert testimony the defendant can obtain often is very dependent on his or her financial resources. A defendant of little means may be unable to bring forward a convincing and credible expert, let alone multiple experts, to testify on his or her behalf. Therefore, the ability to sort out medical evidence in a courtroom, while certainly possible, has serious limitations.

Conclusion

Coagulopathies can mimic child abuse not only by producing easy bruising, but also by allowing small bleeding episodes to become large ones, suggesting to a treating physician that massive force was applied to create the bleeding. Although children with bleeding disorders indeed might be abused, many articles describing cases of child abuse never mention that coagulopathies were ruled out, and in many other reports, the evaluation for coagulopathies is superficial, leaving much room for an undiagnosed bleeding disorder. Even if abuse might have occurred and been documented by other clinical signs, in the presence of a coagulopathy, it might well be that the force applied to the child was not as excessive as the hemorrhaging would seem to indicate. Antiplatelet and anticoagulant medications also can exacerbate bleeding in a patient with a mild or an undiagnosed bleeding disorder. For example, the use of aspirin in patients with mild von Willebrand disease can result in significant bleeding.

Given the inherent challenge of differentiating child abuse from other noninflicted injury, hospitals should consider establishing guidelines for appropriate evaluation of suspected cases of bruising and bleeding in children, including consideration of noncriminal causes. Careful and structured testing for disorders that can cause bleeding and bruising should be established. In addition, a careful medication history to identify drugs that impair hemostasis, including aspirin and nonsteroidal anti-inflammatory drugs, is essential in the evaluation of a bruised child.

A suggested list of tests to be considered at the time of admission would include PT, PTT, platelet count, fibrinogen, von Willebrand factor, ristocetin cofactor, platelet aggregation studies by one of several available methods, and assays

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for factors II, V, VII, VIII, IX, X, and XI to identify mild factor deficiencies that do not prolong the PT or the PTT but might predispose to bleeding. In all of the cases described in this review, one or more of the aforementioned tests at one time or another in the course of the patient's illness revealed the underlying diagnosis. Other tests for more rare bleeding disorders also could be considered if the circumstances merit further evaluation. In the case of factors that are acute phase reactants, the information should be very clear to the treating physician, so a test for these factors is delayed or reordered to obtain the correct answer.

Finally, another important reason that such tests must be performed on admission of the child with bruising or bleeding is that the child might die, and at this point, the cause of the bruising can rarely, if ever, be established. Although many in the court system might believe that the most definitive way to determine cause of death is autopsy, for coagulopathies, which require circulating, unclotted plasma from a living patient for diagnosis, this statement is not correct. Without a substantial evaluation for coagulopathies while the patient is alive, the worst-case scenario is that the family cannot get the truth and might be forced to live with the incorrect perception of guilt and possible punishment, in addition to the death of their child.

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STATE OF ILLINOIS
IN THE CIRCUIT COURT OF THE EIGHTEENTH JUDICIAL CIRCUIT
DUPAGE COUNTY

Randy Liebich, Petitioner	)	Cir. Ct. No. 02-CF-654
v.	)	Post Conviction No.
People of the State of Illinois	)	

AFFIDAVIT OF DARINKA MILEUSNIC-POLCHAN, M.D., Ph.D.

1. My name is Darinka Mileusnic-Polchan. I am the Chief Medical Examiner for Knox County and Anderson County, Tennessee. I am also an Assistant Professor in the Department of Pathology, University of Tennessee Graduate School of Medicine, Knoxville, Tennessee. I am board certified in anatomic and forensic pathology.

2. I obtained my M.D. from University of Rijeka Medical School, Rijeka, Croatia in 1981. I did postgraduate study in Clinical Pharmacology at the University of Rijeka from 1988-1990. In 1999, I obtained a Ph.D. in Neuroscience from Loyola University, Maywood, Illinois. I also completed a residency in Anatomic Pathology/Neuropathology at the Department of Pathology, Loyola University Medical Center. I did a fellowship in Forensic Pathology at the Office of the Medical Examiner Chicago, Cook County, Illinois from 1998-1999 and remained as a full-time Assistant Medical Examiner at the Cook County Medical Examiner's Office until May 2002. My curriculum vita is attached as Ex. 1.

3. I performed an autopsy on Steven Quinn on February 12, 2002 at the Cook County Medical Examiner's Office. At that time, I had accepted a position as Deputy Medical Examiner for Knox County, Tennessee and was finishing my duties in Chicago.

4. In February 2012, I reviewed the autopsy slides and medical records for Steven Quinn. These included a surgical report that establishes that the massive subdural hemorrhage that had reportedly been evacuated at Rush Hospital did not exist, laboratory tests confirming pancreatitis shortly after admission to Mt. Sinai on February 8, 2002, and new stains of the autopsy slides that establish that the child's injuries preceded February 8.

Conclusion

5. The autopsy slides confirm that Steven Quinn had myocarditis (damage to the heart), which would have reduced his circulation and increased his vulnerability to trauma or infection. This is a new finding that has not been previously addressed.

6. The slides confirm that the child had a healing hematoma in the area outside the pancreas that is at least 10 days old and is most likely 2-3 weeks old. This places it in the time period February 1 or earlier. I do not believe this injury was addressed at the trial.

7. The slides, including the new stains, confirm that the remaining abdominal injuries occurred before February 8. Since myocarditis and the peripancreatic hematoma would have made the child susceptible to trauma or infection, these injuries would not require major trauma and are consistent with a push, shove or inappropriate punishment.

8. There is no evidence of head trauma occurring on February 8 and no evidence of inflicted head trauma occurring earlier. The forehead bruise is older (*i.e.*, occurred before February 8) and is of unknown significance, and the large subdural hemorrhage reportedly evacuated at Rush Hospital did not exist.

9. The findings that brought the child to medical attention on February 8 – pancreatitis and a hypoxic-ischemic brain (a brain that lacked oxygen) – are a natural progression of the earlier injuries. They do not suggest trauma occurring on the day of hospital admission.

10. The majority of the lines and marks that appeared during the hospitalization likely reflect pancreatitis and disseminated intravascular coagulation (DIC). The exception is the bruising on the back, which is likely associated with the earlier abdominal injuries.

Autopsy

11. The Cook County investigative report stated that trauma was noted at Mt. Sinai Hospital on 2/8. According to the information provided, the child arrived at Rush Hospital at 8 pm with the diagnosis of head trauma and was found to have a "Massive Subdural Hemorrhage" and marks on the body. The subdural hemorrhage was evacuated on 2/8 and life support was removed on 2/11. The mother reported that the child did not have a significant medical history, was not on any medications, and had a runny nose for a few weeks with no other cold symptoms. Ex. 2. The brief hospitalization report stated that the child died from head trauma. Ex. 3.

12. At autopsy, it was difficult to assess the head injuries since the child had neurosurgery on the right side, reportedly to evacuate a large subdural hemorrhage, causing artifacts. There was also an older bruise, a subgaleal hemorrhage and a 30 g (2 TB) subdural hemorrhage on the left. There was diffuse subarachnoid hemorrhage, and subdural hemorrhage along the spinal cord. The brain was severely swollen (edematous) with herniation.

13. The neuropathological examination found extensive hypoxic ischemic changes (changes due to lack of oxygen) with superimposed respirator changes and extensive necrosis. There was 7 cm contusion necrosis (tissue death) in the left cerebral hemisphere (cortex and subcortical white matter). The blood vessels were distended and congested, and

there were numerous petechiae throughout the white matter. The base of the brain was almost completely necrotic.

14. There were many marks (lines and contusions) on the body but no fractures or other bony abnormalities on examination or x-ray.

15. Although the investigative reports did not mention abdominal injuries, the autopsy revealed 7 inches of ischemic necrotic bowel (bowel that died from lack of oxygen) with a small (.1 inch) bowel perforation, peritonitis, peripancreatitis, pancreatitis, and a subcapsular liver hematoma.

16. My report on the tissue slides states that the pancreatic injuries were subacute (five days or more); the intradural and subdural hemorrhages were approximately day 5; the intramuscular and subcutaneous hemorrhages were approximately day 5; and the small intestinal wall hemorrhage, transmural inflammation and perforation were subacute (five days or more). Since the days are measured in 24 hour increments from the removal of life support, this placed the injuries in the period from noon on February 6 to noon on February 7. Some of the findings looked older; none looked more recent.

Trial

17. Before trial, I spoke with Dr. Teas, a forensic pathologist retained by the defense, who had dated most of the injuries to five days or possibly longer and wanted to confirm that this was consistent with my findings. I told her that this was consistent and that it was in my opinion unlikely that the injuries had occurred three days before death.

18. Although it is routine to order medical records, I do not believe that I received the medical records on this case before completing the report and leaving for Tennessee. I did not have an opportunity to review the slides, photographs or medical records before testifying at trial in 2004.

19. When I returned to Illinois for the trial, the prosecutor urged me to place the injuries three days before death or to testify that this was possible. I made clear that this was very improbable given the stage of healing and made clear that my best estimate of timing was five days or slightly longer. The prosecutor understood my position and did not question me on the timing of the injuries.

20. During my testimony, I was shown hospital photographs that I had not seen previously. In these photographs, some of the marks seen in the hospital had disappeared by the time of autopsy while other marks that were not seen in the hospital appeared at autopsy. This suggests that some of the marks may have been associated with DIC.

21. I was concerned when I learned that Mr. Liebich was convicted of assaulting and murdering Steven on February 8. Given the pathology, it was improbable that any injuries occurred on February 8. Instead, the child's collapse appeared to be the end result of a process that began days earlier.

New information

22. In February 2012, I received additional information on this case, including medical records and stained slides. The new information clarifies the progression of the medical findings and confirms that the child's collapse was due to injuries occurring before February 8.

23. My conclusion that the child suffered a severe head injury was based largely on the investigative report, which stated that a "massive" subdural hemorrhage had been evacuated at Rush. Massive subdural hemorrhages usually represent ruptured bridging veins (the relatively large veins that drain the brain) and are almost always caused by significant impact (accidental or abusive).

24. In February 2012, I learned that the investigative report was incorrect. The medical records confirm that the child was taken to surgery for evacuation of a large subdural hemorrhage but that little or no subdural hematoma was found during surgery. The relevant portion of the surgery report states:

Pre-operative diagnosis:	Subdural hemorrhage
Post-operative diagnosis:	No subdural hematoma found

The text of the report states that when the surgeon opened the skull, he found that the brain was under tremendous pressure, and he elected to perform a decompressive craniectomy (removal of a portion of the skull to relieve the pressure). In so doing, the surgeon "found there was no large subdural blood accumulation. Although there was some subdural blood, it did not appear to be as severe as it appeared to have been on the CT scan that the patient brought in from Mt. Sinai Hospital." The report states that "[i]n summary, this patient had a severely swollen brain with a large amount of subarachnoid hemorrhage and a small thin subdural hemorrhage."

25. This information is critical since thin hemorrhages in children may be secondary to a wide array of natural causes and medical events, including infection in other parts of the body.

26. The laboratory reports reviewed in February 2012 further confirmed that the child had pancreatitis on admission. I am quite certain I did not receive these reports before the trial as I would have noted and remembered the extraordinarily high levels of amylase and lipase present on arrival at Mt. Sinai. Since pancreatitis was a late development, with most of the pancreas spared, this gives us a benchmark for timing the other medical findings, which were older.

27. The lab reports also show that the child's platelets dropped rapidly after admission. When confronted with injury from trauma or infection, the body sends platelets to attempt to "plug" or repair the damage. Once the platelets are used up, the body loses

control of its ability to regulate bleeding and clotting, resulting in hemorrhage, thrombosis and/or easy bruising. This is known as disseminated intravascular coagulation (DIC).

28. In light of the new information, I recently reviewed the hospital and autopsy photographs as well as the autopsy slides (discussed below). The autopsy report and photographs identify more than 40 areas of discoloration (contusion or bleeding under the skin). At trial, these were largely attributed to trauma. At the time, it was my assumption that most of these marks were associated with abdominal injuries occurring approximately five days before the removal of life support. However, the hospital records and trial testimony indicate that most of the contusions were not present on hospital arrival. Based on this information, most of these discolorations were likely due to DIC, pancreatitis and/or hospital interventions.

29. The only contusions that are in my opinion most likely associated with trauma are the circular marks down the spine and a larger contusion to one side. These are not in a location that I associate with DIC, pancreatitis or hospital intervention, and it is my opinion that they are likely associated with the older abdominal injuries. Although these marks were not noted on admission, it can be difficult to identify bruising on African-American children, and it is not clear that the child's back was examined thoroughly before the CT scan. These marks would have increased in size and deepened in color during the hospitalization as a result of DIC.

Slide review

30. I also reviewed the autopsy slides on February 16, 2012 in light of the new information, including slides that had been stained with iron or Masson stains after the trial.

31. To time injuries, forensic pathologists look under the microscope for the types of cells that are sent to heal injuries. These cells appear and disappear in an established order. The first cells to appear are neutrophils, followed by macrophages (histiocytes) and then by fibroblasts. In assessing timing, the pathologist looks for the presence, absence and proportion of these cells. While this timing is imprecise, it is the gold standard for timing and is far superior to the naked eye. It is particularly helpful in allowing us to place findings in order, *i.e.,* to determine which findings are older and which are newer.

32. Since injuries continue to bleed and repair until life support is removed, the presence of fresh blood or neutrophils does not determine the age of the original injury. For that, it is necessary to identify the oldest part of the injury and to determine whether older healing cells are present even in areas of what might appear to the naked eye to be fresh bleeding.

33. For more precise timing, one can stain the tissue slides. Iron stains turn the hemosiderin (iron) in cells blue. Since it takes some time for hemosiderin to develop, slides that have significant areas of blue staining are at least three days old, with more intense and larger areas of staining indicating longer time periods. The Masson stain similarly turns

collagen (the substance found in scars) blue, making it more visible. Areas that appear bright blue with the Masson stain indicate injuries that are at least five days old.

34. Stains are usually ordered when there is disagreement on timing. In this case, there did not appear to be any disagreement on timing since the defense pathologist reached conclusions similar to my own. At trial, I learned for the first time that the prosecutors disagreed with the timing on pathology. While I agreed that some of the bleeding and deterioration would have occurred during the hospital stay, I could not agree that the injuries that began the process occurred three days before the death.

35. My recent review of the autopsy slides confirms that the child had myocarditis (damaged heart cells) and an older pancreatic injury (at least 10 days old) that would have made him more vulnerable to trauma or infection. There is also evidence of a traumatic event occurring approximately 5 days before the removal of life support, culminating in the 2/8 collapse. There are no indications of trauma on the day of admission.

Brain slides

36. The brain slides show a hypoxic ischemic brain consistent with the 2/8 collapse, with superimposed respirator brain and DIC. A few areas suggest slightly older injuries.

37. The 2/16/12 slide review established the following:

Brain slide 1 (possibly brainstem). This slide shows a hypoxic ischemic necrotic (dead) brain. The damage is several days old and is consistent with the 66 hours between hospital admission and removal of life support (respirator brain).

Brain slide 2 (basal ganglia). This slide shows a hypoxic-ischemic brain with some glial reaction. There is global damage (anoxic encephalopathy). This is consistent with the 2/8 collapse and life support.

Brain slide 3 (cortex). This slide shows hypoxia with red neurons, advanced edema and breakdown around the blood vessels. This is likely a reaction to inadequate circulation and poor oxygenation. This is consistent with life support.

Brain slide 4 (cortex/white matter/corpus collosum). This slide shows hypoxia ischemia and mild gliosis consistent with the period spent on life support. There are no visible axonal spheroids that would suggest trauma.

Brain slide 5 (cerebellum). This slide, which is from the back of the brain, is completely necrotic (dead). There is global hypoxia and the granular cells have died, which takes at least 3 days. Since the cerebellum and hippocampus are particularly susceptible to lack of oxygen, this part of the brain may have been affected for up to a week. This may present as lethargy as the brain begins to suffer from lack of proper circulation.

Brain slide 6 (cerebellum). In some areas, the tissue is completely necrotic but other areas are preserved or show early hypoxia-ischemia. There is no evidence of trauma.

Brain slide 7. There is fresh thrombosis (abnormal clotting) in the vessels consistent with disseminated intravascular coagulation (DIC).

Brain slide 8 (possibly temporal cortex). This slide shows thrombosis with minimal subarachnoid hemorrhage.

Brain slide 9. There are thrombosed vessels with surrounding hemorrhage. There is also subarachnoid hemorrhage.

Brain slide 10 (basal ganglia). There is thrombosis with leaky vessels, possibly from increased intracranial pressure.

Brain slide 11 (cortex). There is substantial hemorrhage in one of the lobes in the cortex, going deep into the white matter and extending through the full thickness of the cortex. There are red neurons, possibly thrombosed vessels and subarachnoid hemorrhage. There is some fibrin with fibroblasts, and the proportion has shifted from neutrophils to macrophages. On day 3, one usually sees neutrophils with some lymphocytes and macrophages. This slide has fewer neutrophils and more macrophages than one would expect from an injury that is 3 days old and suggests that this process may have started closer to day 5. This slide is complicated by the artifacts of the surgical procedure (craniectomy with removal of the bone flap) and dura resection, which allowed the brain to swell through the cranial defect and eventually infarct.

Although this slide was initially a candidate for trauma, on review it is not possible to determine whether this represents a contusion (caused by outside force) or infarction (caused by internal obstruction or deterioration). An infarction that surrounds a thrombosed vein may be confused with contusion. The picture is further clouded by the presence of a coagulopathy (bleeding/clotting disorder), most likely DIC. On balance, the most that can be said is that this may represent thrombosis or hypoxia ischemia occurring in the 3-5 day range.

Brain slide 12. This slide shows a hypoxic ischemic brain.

Brain slide 13 (hippocampus/temporal lobe). There is clot in the vessels and a small amount of hemorrhage consistent with hypoxia ischemia and DIC.

Brain slide 14. There are thrombosed vessels and focal subarachnoid hemorrhage with considerable artifact (air bubbles).

Brain slide 15. This slide shows a hypoxic ischemic brain that is degrading.

Brain slide 16 (brainstem/little cerebellum/junction of pons). This slide shows clustered thrombosed vessels in a peculiar formation. Since there doesn't appear to be anything unusual in the brainstem, this likely reflects the way the slide was prepared.

Brain slide 17 (dura). There is a subdural hemorrhage with no neutrophils, some lymphocytes, many macrophages, some fibroblasts and beginning fibrin. This looks older than 3 days and more likely began closer to 5-7 days before removal of life support.

Brain slide 18 (dura). As the brain swells, it squeezes the sinuses (the large veins that drain the brain), causing insufficient circulation and thrombosis. This slide shows a thrombosed superior sagittal sinus with neutrophils (particularly at the periphery), lymphocytes, macrophages and some fibrin. The significant number of neutrophils suggests that it is approximately 3 days old but the fibrin suggests it may be a little older (3-5 days). This slide also shows an earlier bleed/clot in the peripheral areas of the dura, with lymphocytes, histiocytes, fibroblasts, fibrin and a thin layer of hemorrhage with no neutrophils. This looks about 5-7 days old. On its own, this injury is minor.

Brain slide 19 (spinal cord). The spinal cord is hypoxic-ischemic with no signs of trauma. There is some subarachnoid and subdural hemorrhage, likely secondary to the brain findings.

38. *Conclusion.* The slides show a hypoxic ischemic brain consistent with the 2/8 collapse, respirator brain and/or DIC, with no indicators of head trauma. There are some suggestions of earlier findings in the dura and cortex.

Other slides

39. The remaining slides confirm that the child had two pre-existing conditions, myocarditis and a hematoma with scarring in the area outside the pancreas (at least 10 days old). The bulk of the findings were in the 5-7 day range.

Slides 1 and 2 (heart). These slides show myocarditis (damaged heart cells). Since myocarditis is typically multifocal, it can be missed if multiple sections are not taken. Slide 1 has two tiny foci of myocarditis that could easily be missed were it not for the more extensive damage in slide 2. Since myocarditis cannot be repaired, these tiny areas would have developed scars over time.

Slide 2 shows myocarditis with multiple foci and dying muscle in the left ventricle. There are white blood cells (mainly lymphocytes) between the muscles, and the muscle fiber is almost disintegrating. In some areas, the muscle fiber is being replaced by scar tissue with fibroblasts. Since it takes at least a week for fibroblasts to replace muscle, this is a reaction to something that happened at least a week earlier and probably longer.

Based on slide 2, I would have no hesitation signing this out as a death from myocarditis if this were the child's only finding. The damage suggests that the child would have been ill for several days to a week or so before his collapse. If he was too young to verbalize,

he might have simply appeared less active and slower than usual (e.g., talking less and moving more slowly).

Myocarditis is similar to an autoimmune reaction and can be insidious in children. It is often preceded by an ordinary virus, such as an upper respiratory infection or gastrointestinal flu approximately two weeks earlier. In some children, the body does not recognize the alterations in the heart caused by the virus and attacks itself days to weeks later. The child then becomes sick again, this time from the damaged heart. For example, in one of my recent cases, a nine year old girl and other family members had a viral infection from which the other family members recovered. The nine year old was "up and down" for a week or two, and became sick again on a Friday. On Sunday, she went to church but was not herself, didn't eat much, and was sensitive to light. On Monday, her mother went to see if she felt well enough to go to school and found her dead. The autopsy established that she died from myocarditis.

To determine the source of the myocarditis, one needs good information on the weeks before the collapse. Specifically, one would want to know if the child, his family or other children in daycare had an upper respiratory or gastrointestinal infection, possibly with diarrhea. If the alterations in the heart elicit an immune reaction, which typically occurs a week or two after the infection, the child may be sickly or lethargic for days to weeks as the heart became less and less effective. Myocarditis can cause ischemia in other parts of the body, including the bowel, making it more susceptible to injury or rupture.

Myocarditis may also be secondary to sepsis (infection that enters the bloodstream). In this case, however, a review of the slides does not suggest sepsis. In any event, if the myocarditis were related to sepsis, one would see neutrophils rather than scarring. In this case, the heart was reacting to a subacute (older) insult.

Slide 4 (bowel). There is considerable hemorrhage in the mesentery with some reaction. From the outside in, the bowel consists of serosa, muscle and mucosa, with serosa on the outside and mucosa on the inside. There is hemorrhage under the serosa and limited mucosal inflammation in one area. There is not much ischemia. There are many plump macrophages with some lymphocytes and no neutrophils. The fibroblasts are moving towards a scar. Since there are no neutrophils and the lymphocytes are more numerous than the fibroblasts, I would place this at approximately day 5. An iron stain shows more iron than one would expect on day 3, confirming this timing.

Slide 5 (spleen). This does not look like sepsis as there are not enough neutrophils.

Slide 6 (liver). Liver damage is first seen in the area where the lobes come together. This slide shows some thickening of the venules with perivenular reaction. There is some reactive change in the portal spaces that may be attributable to inflammation or beginning sepsis. There are possibly minor changes from compromised circulation but they are insufficient to kill the liver cells. There is a small subcapsular hemorrhage with many neutrophils, some lymphocytes and a few macrophages. These changes are occurring at the edge of the liver and are difficult to date.

Slide 7 (pancreas). There are some inflammatory cells in and around the pancreas but most of the pancreas is intact. One portion of the slide shows fibrinous necrosis of a vessel that has a clear thrombus with rare neutrophils, some lymphocytes and predominantly macrophages. There is relatively little organization and only a small amount of hemorrhage. There is necrosis and hemorrhage in the surrounding fat. The cells that secrete amylase and lipase are breaking down, which would cause them to release amylase and lipase into the bloodstream. There is no suggestion of trauma. The vessels appear thrombosed with surrounding reaction/inflammation (neutrophils, macrophages and lymphocytes). This may have started on day 3 or a little earlier.

Slide 9 (thymus). This slide shows nonspecific reaction.

Slides 10 -14 (lung). Two of the five lung slides show segmental pneumonia in the lower lobes, which is where pneumonia typically first appears. This is consistent with respirator pneumonia (pneumonia that develops during life support) and is insufficient for sepsis. Slide 10 shows early bronchopneumonia consistent with respirator pneumonia; most of the lung is clear. Slide 11 shows segmental rather than lobar pneumonia, consistent with respirator pneumonia; there is no indication of sepsis. Slide 12 shows minor focal bronchopneumonia with some congestion and edema; most of the lung is clear. Slide 13 shows segmental bronchopneumonia with many neutrophils; much of the area is clear. Slide 14 shows minor focal bronchopneumonia with edema and congestion, with no signs of sepsis; most of the lung clear.

Slide 15 (stomach). The gastric mucosa has undergone autolysis.

Slide 16 (pancreas/peripancreas). There is hemorrhage around the pancreas with some neutrophils and what appears to be a capsule with fibroblasts and possible scarring. Masson staining confirms a hematoma that is almost encapsulated and has a scar on the periphery from a previous insult. There are fibroblasts but no macrophages or neutrophils. Typically, fibroblasts start around day 5, dominate on day 7 and then take over; macrophages start disappearing around day 10. This area of damage appears to be at least 10 days old and is possibly 2-3 weeks old.

Slide 17 (thyroid). There is nothing notable in the thyroid.

Slide 18 (skin). Since there are not many hairs in this segment, it is not part of the scalp. There is deep bruising with organization and no neutrophils. This injury is most likely about 5-7 days old. Since the slides are not labeled, it is not possible to determine the source with certainty. This slide may have been taken from the back.

Slide 19 (skin). This slide shows a hair shaft and follicles. It is likely from the forehead as there are not many follicles, and it may be from the bruise on the left forehead. In the central area, there is a hematoma with some bleeding and a lot of surrounding reaction. There are no neutrophils, few lymphocytes (which typically exit around 3-5 days), some

macrophages and many fibroblasts. I would estimate the age at 5-7 days. Given the amount of iron in the macrophages in the stained slide, it is probably closer to a week old.

Slide 20 (area of bowel perforation). There is inflammation and necrosis involving the entire wall. At the periphery, there are macrophages, lymphocytes, fibroblasts and a great deal of fibrin. In the middle, there are more neutrophils, particularly on the surface. Since the healing and repair process continues until the child dies, neutrophils will appear until life support is removed. The oldest part of the damage appears to be approximately 5 days old.

This picture is clouded by myocarditis. When the child is ill, everything in the body suffers, including the bowel. Since an ill child can act up, a parent may also be more inclined to slap or punish. Since the injured organs are anchored (rather than floating), this type of abdominal injury does not require a great deal of trauma and is seen in children who hit the handlebars of a bicycle or are restrained by an adult seatbelt. These injuries are also consistent with being slapped or punched in the stomach or pushed from the back, causing the child to hit a wall or furniture. It is not possible to determine whether the small perforation was caused by infection/ischemia or external force.

Slide 21 (mesentery). These findings do not seem acute and appear to have occurred around day 5.

Slide 22 (diaphragm). There is considerable hemorrhage around the diaphragm. The diaphragm separates the abdomen from the chest and is a major muscle used in breathing and respiration. There are some neutrophils, many macrophages and some fibroblasts. This amount of reaction is consistent with day 5 or slightly longer and is inconsistent with day 3. This hemorrhage suggests that a traumatic event occurred around day 5.

Slides 23-25, 27 (mesentery). There are thrombosed vessels consistent with DIC and reaction, with no evidence of trauma. These findings likely occurred around day 3.

Slide 26 (dura). There is a thin intradural/subdural hemorrhage with reaction. This is consistent with day 3. This finding also coincides with the surgical intervention (craniectomy).

Slide 28 (testicle). There is no hemorrhage or trauma to the testicle.

Slide 29 (bowel). This slide shows an ischemic bowel in the area of perforation. The bowel wall has been almost entirely replaced by transmural inflammation (inflammation that extends through the entire thickness of the bowel). There is hemorrhage but the perforation does not appear complete. Since an ischemic bowel is susceptible to injury and can rupture with the insertion of a penrose drain, the perforation seen at autopsy may be an artifact of treatment.

Slide 30 (bowel). The area adjacent to the perforation is less involved. There is some ischemia with hemorrhage on the serosa (outer lining) with macrophages and many fibroblasts. This is approximately day 5 and is inconsistent with day 3.

Slide 31 (colon). The colon is not involved.

Slide 32 (bowel). In this slide from the area of perforation, the bowel has lost its integrity, which means that it can perforate or fall apart without trauma. There is necrosis through the serosa, muscle and mucosa with hemorrhage through all three layers. There are newly recruited neutrophils, many macrophages and fibroblasts. Since this is a continuing process, neutrophils will be recruited until the child is removed from life support. It is not possible to determine what was caused by trauma and what was caused by DIC since the original findings are overrun and obscured by DIC.

Slides 33-34 (bowel). These slides show a necrotic bowel with hemorrhage likely associated with DIC.

Slides 35-36 (adrenal gland). There is thrombosis consistent with DIC. There is no hemorrhage, suggesting that the child was not septic.

Slide 37 (liver). There is a large subcapsular hemorrhage with inflammatory cells at the edges. There are no neutrophils, some macrophages and some lymphocytes. This suggests day 5. An iron stain is consistent with this timing.

Slides 38-39 (kidneys). There is some thrombosis with no hemorrhage.

Hot dog incident

40. It is my understanding that Mr. Liebich described a hot dog/choking incident occurring approximately three hours before hospital admission. I do not know the role of this incident. However, a sick child is unlikely to be able to swallow and could easily choke, further reducing the oxygen supply and possibly triggering the collapse. This incident is also consistent with seizure.

41. I am told that, according to Mr. Liebich, Mr. Liebich attempted to clear the child's airway and that the child bit down on his finger when he did so. Mr. Liebich reportedly slapped the child's cheek to get him to release the finger. This would not cause head injury.

Progression of injuries

42. The new information makes it possible to provide more accurate information on timing. It is particularly helpful in determining relative timing, *i.e.*, which findings came first and which came later.

43. The slide review confirms that the child had myocarditis (damage to the heart), most likely caused by a virus 2-3 weeks earlier. This would have damaged his circulation and

made him vulnerable to traumatic or hypoxic ischemic injury. He also had a healing hematoma outside the pancreas that is at least 10 days old and likely 2-3 weeks old.

44. Approximately five days before death, the child developed an ischemic bowel with liver involvement and hemorrhage in the diaphragm. While there are natural causes for the abdominal findings, the bruises on the lower back are suspicious for trauma given their location and extent. Given the pre-existing conditions, it would not have required much force to cause these findings.

45. There is no evidence of head trauma and no evidence of any trauma occurring on the day of collapse. Instead, the events on the day of admission represent a natural progression of the earlier injuries, which culminated in pancreatitis and a hypoxic-ischemic brain. The majority of the findings on the scalp, skull, meninges and brain were the consequences of surgical intervention on the evening of the 8th.

I swear under penalty of perjury that the foregoing is true and correct.

MD PhD

Darinka Mileusnic-Polchan, M.D., Ph.D.

Date: April 04, 2012

Linda L. Hall

CURRICULUM VITAE

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EDUCATION:

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Not Applicable.

Graduate/Medical School:
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Postgraduate Study in Clinical Pharmacology, Medical School, University of Rijeka, Rijeka, Croatia, 9/1988-9/1990

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Internship:
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Anatomic Pathology/Neuropathology, Department of Pathology, Loyola University Medical Center, Maywood, Illinois, 7/1995-7/1998

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Scholarship, Neuroscience Graduate Program, Loyola University Graduate School, 8/1992-6/1995

Scholarship Award for Italian Language Studies, Centro Culturale e Linguistico, Rimini, Italy, 7/1991

Scholarship Award for Masters Degree in Clinical Pharmacology, Medical School University of Rijeka, Croatia, 9/1988 - 9/1990

Merit Award as Top-Ranking Student Class of 1986, Medical School University of Rijeka, Croatia, 9/1984 - 9/1985

BOARD CERTIFICATION:

American Board of Pathology Certification in Anatomic Pathology, 10/2000

American Board of Pathology Certification in Forensic Pathology 9/2001

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American Academy of Forensic Science	since 01/1999
National Association of Medical Examiners	since 01/1999
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PRACTICE/PROFESSIONAL EXPERIENCE:

Chief Medical Examiner, Knox County Office of the Medical Examiner, Knoxville, Tennessee, 5/2008 – present

Chief Medical Examiner, Anderson County, Tennessee, 7/2008-present

Acting Chief Medical Examiner, Knox County Office of the Medical Examiner, Knoxville, Tennessee, 1/2008 – 5/2008

Assistant Chief Medical Examiner, Knox County Office of the Medical Examiner, Knoxville, Tennessee, 6/2002 – 1/2008

Assistant Medical Examiner, Cook County Office of the Medical Examiner, Chicago, Illinois, 8/1999 - 5/2002

CERTIFICATES, AWARDS, COMMITTEES AND OFFICES HELD:

Four Year Accreditation of the Regional Forensic Center by the National Association of Medical Examiners, July 2010 – July 2014.

Member of the Search Committee for the Department Chair of Pathology, University of Tennessee Graduate School of Medicine, May 2011 – present.

Member of the Dissertation Committee for Donna McCarthy, a Dissertation Presented for the Doctor of Philosophy Degree: "Using Biological Evidence to Assess Affinity: A Re-evaluation of Selected Mississippian Sites in East Tennessee," Department of Anthropology, The University of Tennessee, Knoxville, Tennessee, March 2011.

Commendation for Exemplary Service Presented by the District Attorney General, 8th Judicial District, State of Tennessee, Lafollette, Tennessee, November 2010.

Certificate of Completion of the Advanced HAZMAT Life Support Course, Knox County Health Department, Administered by AHLS International Office, Knoxville, Tennessee, October 2008.

Certification of Completion of the Mass Fatalities Incident Response Planning Course, the National Mass Fatalities Institute, Oak Ridge, Tennessee, September 2007.

Member of Pathology-Biology Scientific Program, Abstract Review Committee and Moderator for Pathology-Biology Section, American Academy of Forensic Sciences 58th Annual Meeting, Seattle, Washington, February 2006.

Member of Performance Improvement Committee, University of Tennessee Medical Center, 1/2004 - present

Member of National Association of Medical Examiners Ad Hoc Committee on Position Papers, 1999 - 2003

Member of Child Fatality Review Team, Knox County Health Department, Knoxville, Tennessee, 8/2002 - present

Member of the Dissertation Committee for Mariateresa Anne Tersigni, a Dissertation Presented for the Doctor of Philosophy Degree: "Serial Long Bone Histology: Inter- and Intra-Bone Age Estimation," Department of Anthropology, The University of Tennessee, Knoxville, Tennessee (graduated in 2005)

Member of the Dissertation Committees for Rebecca Wilson and Sandy Cridlin, Department of Anthropology, The University of Tennessee, Knoxville, Tennessee

RESEARCH AND OTHER EXTERNAL SUPPORT:

Collaborative Project, Department of Pathology, University of Tennessee Medical Center and Department of Anthropology (Murray Marks, Ph.D.), University of Tennessee: Fetal Osteology Project, 2006 - present.

Collaborative Project, Department of Pathology, University of Tennessee Medical Center and Department of Anthropology (Murray Marks, Ph.D.), University of Tennessee: Bone Healing in Child Abuse, 2005 – present.

Collaborative Project, Department of Pathology, University of Tennessee Medical Center and Department of Anthropology (Murray Marks, Ph.D.), University of Tennessee: Histological Evaluation of Postmortem Changes in Human Soft Tissues, 2004 - present.

Collaborative Project, Cook County Office of the Medical Examiner and Northwestern University, Department of Psychology: Prediction of Violent Crime Among Juvenile Delinquents, a Longitudinal Study, 2001 - 2002.

Collaborative Project, Cook County Office of the Medical Examiner and Loyola University Medical Center, Department of Pathology: Molecular Pathology of Myocarditis in Cases of Sudden, Unexpected Death, 2001 -2002.

Collaborative Project, Cook Co. Office of the Medical Examiner and Northwestern University, Department of Immunology: Relationship Between Bronchial Asthma and Drugs of Abuse, 2001.

Resident Research Proposal: Escape from p53-Mediated Apoptosis in High-Grade Gliomas, in Collaboration with Dr. J.M. Lee, Department of Pathology, and Drs. E. Melian and C. Scharf from Radiation Oncology, Loyola University Medical Center, 1998.

Loyola University Medical Center and VA Hines Brain Bank Project, under Dr. J.M. Lee's Directorship, 7/1995 - 7/1998.

SYMPOSIA, PRESENTATIONS, LECTURES AND TEACHING:

National Forensic Academy: Selected Topics in Death Investigation; Part I: The Role of the Medical Examiner in Drug Abuse Deaths; Part II: Abusive Head Trauma of Childhood, Knoxville, Tennessee, October 2011.

Third Annual Regional Forensic Center Conference: Forensic Connections - Toxicology, Part I and Part II, Knoxville, Tennessee, September 2011.

Knoxville Area Health Information Management Association Annual Meeting, Key Note Speaker, Knoxville, Tennessee, August 2011.

East Tennessee Prescription Drug Summit (Organized by the National Forensic Academy): Prescription Drug Issues in the United States and Greater Knoxville Area, Knoxville, Tennessee, July 2011.

National Forensic Academy Best Practices Symposium; Case Study: Christian/Newsom Double Homicide, Nashville, Tennessee, December 2010.

Tennessee Donor Services Regional Summit: Collaboration Between Medical Examiner's Office, Funeral Homes and Donor Services, Knoxville, Tennessee, October 2010.

National Forensic Academy: Selected Topics in Death Investigation; Part I: The Role of the Medical Examiner and Natural Deaths in Adults; Part II: Child Abuse, Knoxville, Tennessee, October 2010.

Second Annual Regional Forensic Center Conference: Coordination of Death Investigation; Forensic Pathology II: Blunt Head Injuries and Child Abuse II, Knoxville, Tennessee, October 2010.

National Forensic Academy: Selected Topics in Death Investigation: The Role of the Medical Examiner and Natural Deaths in Adults, Knoxville, Tennessee, February 2010.

First Annual Regional Forensic Center Conference: Coordination of Death Investigation; Forensic Pathology I: Basic Autopsy Service and Child Abuse, Knoxville, Tennessee, October 2009.

Federal Bureau of Investigation Child Abduction Rapid Deployment Team Training Conference: Forensic Pathology of Trauma, Knoxville, Tennessee, June 2008.

Federal Defender Services of Eastern Tennessee Regional Meeting: Forensic Pathology Pearls, Knoxville, Tennessee, November 2007.

University of Tennessee Law School and District Attorney General: Advanced Trial Practice Class, Law School's Advocacy Center, Knoxville, Tennessee, September 2006.

Panel Member and Presenter for Physical Anthropology Session Entitled Working With Family Members of Decedents: A Discussion of Techniques for Forensic Scientists, 58th Annual Meeting, American Academy of Forensic Sciences, Seattle, Washington, February 2006.

Sevier County Law Enforcement In-service Certification School: "Forensic Pathology Basics," Sevierville, Tennessee, November 2005.

Knoxville Bar Association, Continuing Legal Education: Mock Trial Seminar, Knoxville, Tennessee, October 2005.

The University of Tennessee Graduate School of Medicine Department of Continuing Medical Education - The 2005 John E. Sullivan, DDS Memorial Endowed Lecture: "Forensics of Substance Abuse," Knoxville, Tennessee, April 2005.

The University of Tennessee Knoxville, Forensic Anthropology Center and Federal Bureau of Investigation Sponsored Annual Human Remains Recovery School: "Introduction to Forensic Pathology," Knoxville, Tennessee, March 2003; March 2004; March 2005.

Forensic Pathology Basics: Collaboration Between Forensic Pathologists and Forensic Anthropologists, Forensic Anthropology Lecture Series for Graduate Anthropology Students, University of Tennessee, Knoxville, Tennessee, August 2004.

The State of Tennessee Annual Public Defender Investigator Training Conference: "Forensic Pathology Basics," Glenstone Lodge, Gatlinburg, Tennessee, October 2003.

Northwestern University, Forensics Professional Development Program: "Asphyxial Deaths," Chicago, Illinois, April 2002.

Forensic Pathology Workshop at "The Changing Face of Medicine" Student National Medical Association Region II Conference, Oak Brook Marriott, Oak Brook, Illinois, November 2001.

Series of Lectures on Forensic Pathology and Medicine for Pathology Residents, Loyola University Medical Center, Maywood, Illinois, Scholastic Year 2001/2002

Pathology Grand Rounds for Loyola University Medical Center, Department of Pathology: "An Introduction to Forensic Pathology," Maywood, Illinois, March 2001.

Human Anatomy and Physiology Society and the Chicago Area Anatomy and Physiology Society Regional Conference: Lecture Entitled "The Disease Process," Triton College, River Grove, Illinois, February 2001.

Forensic Pathology Basics, Lectures for 2nd Year Medical Students, Loyola University Chicago, Stritch School of Medicine, Maywood, Illinois, April 2000.

Trial Practice Courses of the Kent Law School I.I.T., Chicago, Illinois, March 1999/2000.

Trial Practice Course of the Chicago Bar Association, Chicago, Illinois, February 2000.

Juvenile Law Class, Kent Law School: Child Abuse and Shaken Baby Syndrome, Chicago, Illinois, April 2000.

Instructor in Forensic Pathology to Forensic Pathology Residents and to Visiting Medical Students and Resident Physicians at the Cook County Medical Examiner's Office, Chicago, Illinois, August 1999-May 2002.

Illinois Registry of Anatomic Pathology: "Primary Peritoneal Malignant Mixed Müllerian Tumor," Chicago, Illinois, April 1998.

Illinois Registry of Anatomic Pathology: "Congenital Neuroblastoma: Primary Diagnosis in Placenta," Chicago, Illinois, April 1997.

Illinois Society of Cytology: "Collision Tumors: Diagnosis by Fine Needle Aspiration Biopsy," Chicago, Illinois, May 1997.

Symposium on Sports Medicine: "Pharmacological and Clinical Implications of Beta Blockers," Crikvenica, Croatia, June 1989.

ABSTRACTS:

Evans, SR, **Mileusnic-Polchan, D.** Patterns of Breaks in Umbilical Cords by Different Mechanisms, Platform Presentation, Annual Meeting of the National Association of Medical Examiners, Cleveland, Ohio, October 2010.

Hendrickson, BW, **Mileusnic-Polchan, D.** Polysplenia and Associated Cardiac and Visceral Malformations in a Case of Heterotaxy with Fetal Demise, Poster Presentation, University of Tennessee Medical Center Pathology Resident Research Conference, Knoxville, Tennessee, June 2010.

Evans, SR, **Mileusnic-Polchan, D.** Patterns of Breaks in Umbilical Cords by Different Mechanisms, Slide Presentation, University of Tennessee Medical Center Pathology Resident Research Conference, Knoxville, Tennessee, June 2010.

Bethard, JD, Marks, MK, **Mileusnic-Polchan, D.** Aquatic Taphonomy in a Lacustrine Environment: A Case Study from Southeastern Tennessee, Poster Presentation, 61st Annual Meeting, American Academy of Forensic Sciences, Denver, Colorado, February 2009.

Bruker, CT, Googe, PB, **Mileusnic-Polchan, D**. Acrodermatitis Enteropathica: Is It an Underreported Cause Of Morbidity and Mortality in Infancy in The United States? Poster Presentation, University of Tennessee Medical Center Pathology Resident Research Conference, Knoxville, Tennessee, June 2008.

Zezulak, AC, **Mileusnic-Polchan, D**. A Case Report of Acute Splenic Sequestration Crisis as a Complication of Sickle Cell Anemia, Poster Presentation, University of Tennessee Medical Center Pathology Resident Research Conference, Knoxville, Tennessee, June 2008.

Mock, AR, Mash, D, **Mileusnic-Polchan, D**. Case Report: Dopaminergic Overdrive in Cocaine-Induced Excited Delirium, Poster Presentation, University of Tennessee Medical Center Pathology Resident Research Conference, Knoxville, Tennessee, June 2008.

Trammell, LH, Marks, M, Klippel, WE, **Mileusnic-Polchan, D**. Cranial Histomorphology: Species Identification and Age Estimation, Poster Presentation, 60th Annual Meeting, American Academy of Forensic Sciences, Washington DC, February 2008.

Mock, AR, **Mileusnic-Polchan, D**. Electromuscular Incapacitating Devices in Excited Delirium, Slide Presentation, University of Tennessee Medical Center Pathology Resident Research Conference, Knoxville, Tennessee, June 2007.

Marks, M, **Mileusnic-Polchan, D**. Histopathology of Antemortem Infant Bone Fracture: Estimation of Time Since Insult, Slide Presentation, 59th Annual Meeting, American Academy of Forensic Sciences, San Antonio, Texas, February 2007.

Marks, M, Tersigni, MA, **Mileusnic-Polchan, D**. Antemortem vs. Perimortem Infant Rib Fracture: The Histological Evidence, Poster Presentation, 58th Annual Meeting, American Academy of Forensic Sciences, Seattle, Washington, February 2006.

Mileusnic-Polchan, D, O'Connor, S. Child Abuse by Another Child: Can It Happen? Slide Presentation, 57th Annual Meeting, American Academy of Forensic Sciences, New Orleans, Louisiana, February 2005.

Mileusnic, D, Donoghue, ER. Lucid Interval Revisited: Delayed Onset of Unconsciousness in an Impacted Infant, Slide Presentation, 56th Annual Meeting, American Academy of Forensic Sciences, Dallas, Texas, February 2004.

Mileusnic, D, Lee, JM. Upregulation of CNS Neurokinin-3 Receptors in Alzheimer's Disease Brain and Following Amyloid-β 25-35 Injections in Young Male Fisher-344 Rats, Slide Presentation, 29th Annual Meeting, Society for Neuroscience, Miami, Florida, October 1999.

Mileusnic, D, Denton, JS, Donoghue, ER. A Review of 38 Cases of Strangulation for the Year 1996 in Cook County, Illinois, Slide Presentation, 33rd Annual Meeting, National Association of Medical Examiners, Minneapolis, Minnesota, October 1999.

Mileusnic, D, Lee, JM. Differential Effects of Aging on the Neurokinin B System: Neurons versus Astrocytes, Slide Presentation, 27th Annual Meeting, Society for Neuroscience, New Orleans, Louisiana, October 1997.

Mileusnic, D, Jensen, J, Pierce, K, Reyes, C. Collision Tumors, Poster Presentation, Southern Medical Association, 91st Scientific Assembly, Charlotte, North Carolina, October 1997 (Abstract Published in Southern Medical Journal 90:S94; 1997.)

Mileusnic, D, Lorens, JB, Lorens, SA, Lee, JM. Characterization of the Neurokinin-3 Receptor in Human Brain, Poster Presentation, Annual Meeting of the American Association of Neuropathology, Vancouver, Canada, 1996.

Mileusnic, D., Magnuson, DJ, Hejna, MJ, Lorens, JB, Lorens, SA, Lee, JM. Neurokinin-3 Receptor Distribution in Rat and Human Brain: An Immunohistochemical Study, Poster Presentation, 25th Annual Meeting, Society for Neuroscience, San Diego, California, November 1995.

Mileusnic, D, Magnuson, DJ, Lorens, SA, Lee, JM. Preprotachykinin B [Neurokinin B (NKB)] Immunoreactive Neuronal Processes in Human Brain, Poster Presentation, 23rd Annual Meeting, Society for Neuroscience, Washington DC, November 1993.

Mileusnic, D, Simonic, A. Effects of Nicotine and Lecithin on Spontaneous Motor Activity in Rats, Poster Presentation, Annual Meeting of the Croatian Pharmacological Society, Zagreb, Croatia, June 1990.

Simonic, A, **Mileusnic, D.** Effects of Physostigmine and Neostigmine on Spontaneous Motor Activity in Rats, Poster Presentation, Annual Meeting of the Croatian Pharmacological Society, Zagreb, Croatia, June 1990.

PUBLICATIONS, BOOKS AND BOOK CHAPTERS:

Evans, SR, **Mileusnic-Polchan, D.** Patterns of Breaks in Umbilical Cords by Different Mechanisms, Accepted to the American Journal of Forensic Medicine and Pathology, June 2011.

Lochmuller, CM, Marks, MK, **Mileusnic-Polchan, D**, Cogswell, SC. Misidentification of a Transverse Occipital Suture as a Persistent Mendosal Suture, Journal of Pediatrics [Epub ahead of print]; 2011.

Marks, MK, Marden, K, **Mileusnic, D.** Forensic Osteology of Child Abuse, Chapter in Hard Evidence: Case Studies in Forensic Anthropology, Second Edition by Dawnie Steadman, Pearson Education, Inc., December 2008.

Teplin, LA, McClelland, GM, Abram, KM, **Mileusnic, D.** Early Violent Death in Delinquent Youth: A Prospective Longitudinal Study, Pediatrics 115:1586-1593; 2005.

Mileusnic, D. Lucid Interval and Delayed Onset of Unconsciousness Resulting in Sudden Death Following Abusive and Accidental Head Trauma of Childhood, In Preparation.

Uschuplich, V, Johnson, M, **Mileusnic, D.** Pathologic Quiz Case: Progressive Fatal Encephalopathy in Immunosupressed Patient with History of Discoid Lupus Erythematosus, Archives of Pathology and Laboratory Medicine 128:e109-111; 2004.

Denton, JS, **Mileusnic, D.** Delayed, Sudden Death in an Infant Following Accidental Fall: A Case Report with Review of the Literature, The American Journal of Forensic Medicine and Pathology 24:371-376; 2003.

Mileusnic, D, Donoghue, ER, Lifschultz, BD. Pathological Case of the Month: Sudden Death in a Child as a Result of Pancreatitis During Valproic Acid Therapy, Pediatric Pathology and Molecular Medicine 21:477-484; 2002.

Tatum, AJ, **Mileusnic, D**, Lifschultz, BD, Donoghue, ER, Greenberger, PA. Clinical, Pathologic and Toxicologic Findings in Asthma Deaths in Cook County, Illinois, Allergy and Asthma Proceedings 22:285-291; 2001.

Hermann, ME, **Mileusnic, D**, Jorden, M, Kalelkar, MB. Sudden Death in an Eight Week Old Infant with Beckwith-Wiedemann Syndrome, The American Journal of Forensic Medicine and Pathology 21:276-280; 2000.

Mileusnic, D, Magnuson, DJ, Hejna, MJ, Krause, EJ, Lorens, JB, Lorens, SA, Lee, JM. Neurokinin-3 Receptor Distribution in Rat and Human Brain: An Immunohistochemical Study, Neuroscience 89:1269-1290; 1999.

Mileusnic, D, Magnuson, DJ, Hejna, MJ, Lorens, JB, Lorens, SA, Lee, JM. Age and Species Dependent Differences in the Neurokinin B System in Rat and Human Brain, Neurobiology of Aging 20:19-35; 1999.

Thomas, C, **Mileusnic, D**, Carey, RB, Kampert, M, Anderson, D. Fatal Chaetomium Cerebritis in a Bone Marrow Transplant Patient, Human Pathology 30:874-879; 1999.

Mileusnic, D. Neurokinin B System in the Rat and Human Brain: Interspecies Differences, Effects of Aging and Changes in Alzheimer's Disease. Dissertation Submitted to the Faculty of the Graduate School in Candidacy for the Degree of Doctor of Philosophy, Loyola University, Chicago.

COMMUNITY WORK:

Volunteer Work for the Car Safety Committee, Farragut Primary School, Knoxville, Tennessee, August 2005-April 2010.

5. The autopsy identified three types of injuries: abdominal injuries; head injuries; and bruising. The abdominal findings consisted of an ischemic necrotic bowel (approximately 7 inches) with a very small (.1 inch) bowel perforation; peritonitis (inflammation/infection in the peritoneal cavity surrounding the abdominal organs); peripancreatitis and pancreatitis (inflammation or infection surrounding and entering into the pancreas); and a healing liver injury.

6. There were two medical findings in the head: a hypoxic-ischemic brain, *i.e.*, a brain that had been deprived of oxygen for an extended period, and relatively small subdural/subarachnoid hemorrhage.

7. The external injuries consisted of a wide variety of marks (bruises, lines, etc.), most notably on the back.

8. Given the complexities of the case, my initial focus was on determining the timing of the various findings based on the microscopic slides of the tissues. Pathologists time injuries based on the presence or absence of various types of "healing" cells that can be seen under a microscope. The progression of healing is established in the literature. To heal an injury, the body sends neutrophils to the injured area, followed by macrophages and fibroblasts. If the pathologist sees only hemorrhage or neutrophils, the injury is new, with the time measured in hours. If there are macrophages and granulation tissue with few if any neutrophils, the injury is at least 5 days or more since it takes that long for the fibroblasts to reach that stage of healing (granulation tissue).

9. In looking at timing, it is critical to look at the oldest part of an injury. Once healing occurs, the delicate vessels in the granulation tissue can rebleed easily. Injuries that do not heal can also expand, as occurred in this case. Thus, the presence of fresh blood does not tell you that the injury is new. Instead, to determine when the injury occurred, one must look at the oldest area of the injury.

10. The healing in the initial slide review established that Steven's injuries occurred approximately five days before he was removed from life support. Since Steven was taken off life support at February 11 at noon, this meant that the injuries most likely occurred by noon on February 6 or earlier. It is also possible that the injuries occurred early on February 7. Some portions of the injuries appeared to be even older, closer to the seven day range.

11. Before giving my opinion, I obtained the handwritten slide review notes by Dr. Mileusnic, the forensic pathologist who conducted the autopsy at the Cook County Medical Examiner's office. These notes indicated that Dr. Mileusnic dated the injuries at approximately five days, or two days before Mr. Liebich cared for the child. I also spoke with Dr. Mileusnic, who confirmed her written opinion.

12. After I gave my opinion, Mr. Ruggiero, the prosecutor, called me. In our conversation, he inquired about the part of the autopsy report in which Dr. Mileusnic had opined that the injuries were 5 days old. I pointed out pages in the autopsy report where the timing was

mentioned. At the time, it was my impression that Mr. Ruggiero had not been aware that the medical examiner had timed the injuries to a period before Mr. Liebich cared for the child.

13. When I was consulted, I advised Mr. Holman and Mr. Casey that they needed to thoroughly understand how pathological timing is done, and I further advised them to go to Tennessee to review the slides with Dr. Mileusnic so they could understand the basis for her opinion. This would also refresh her memory since she had left the Cook County Medical Examiner's Office and would not have access to the slides or other materials. Since the medical records that I received were incomplete and in some cases illegible, I asked them to subpoena the records directly from the hospitals. Finally, I advised that they needed to establish that the child had been symptomatic in the days before his collapse, as evidenced by his weight loss and the Tylenol in his system. In my opinion, it would not be possible for a Court to consider the issues without complete information.

Verdict

14. When I learned after trial that Mr. Liebich had been convicted of causing the injuries despite pathological evidence establishing that the injuries occurred before the child was in his care, I advised the Court that the amount of reaction and healing in the tissues was inconsistent with injuries occurring on February 8 and that the child's 4-5 pound weight loss between his November 2001 checkup and hospital admission further confirmed that this process began well before February 8.

15. In February 2012, I learned that the Court relied on my testimony to place the injuries within the time period that Mr. Liebich cared for the child. In her judgment, the Court correctly states that I timed the injuries to 5 days, plus or minus 1 day, based on my review of the slides. However, the Court went on to say that this timing places the injuries between February 4-8 or February 5-9 based on the healing shown on the slides. This was not my testimony and does not follow from the timing that I provided. Since the body does not recognize calendar days, pathological timing is measured in hours, with a day being a full 24 hour period.

16. Since Steven was removed from life support at noon on February 11, the 5 days of healing shown on the slides goes back to at least noon on February 6. With one day on either side, the timing for the injuries goes from noon on February 5 to noon on February 7. As I made clear in my testimony, the healing shown on the slides was highly unlikely to have occurred after noon on February 7 and likely occurred much earlier. The court's dating appears to have been a mathematical miscalculation.

17. I also just learned that Mr. Casey suggested in his closing argument that I was not sure whether I saw older injuries, *i.e.,* injuries occurring before February 8. This was not my testimony. While pathological timing is not precise and in medicine one can rarely say "never," my testimony was that the healing and reaction seen in the slides represented injuries that occurred on or before the morning of February 7. This testimony was based on established pathological principles.

18. In the verdict, the Court relies heavily on the testimony of the neurosurgeon, Dr. Munoz, who timed the injuries based on the color of the blood seen at surgery. What is seen visually and what is seen under the microscope can, however, be quite different. The principles are the same in forensic and clinical pathology. Gross examination of blood or tissue is not a reliable method to assess healing or timing or pathology. A surgeon who takes a tissue sample from a breast does not rely on a visual review to determine whether cancer is present. Instead, the tissue is sent to a pathologist who examines it under the microscope. The same is true in forensic pathology. Often an injury that looks fresh visually may prove to be days, weeks or even months old when the oldest portion of the injury is viewed under the microscope.

19. Since Steven continued to deteriorate in the hospital, I agree that Dr. Munoz would have seen fresh blood and a very swollen brain at surgery. I also agree with the treating doctors that Steven would not have been able to eat, drink or behave normally in the condition he was in when he arrived at the hospital since by then he had acute pancreatitis and a hypoxic brain. However, this does not indicate or suggest when this process began or what started it. For that, it is necessary to review the pathology, which in this case confirmed that the process had been ongoing for more than a day before hospital admission.

20. I have been told that the Court emphasized that the damage to the bowel, pancreas and liver represented a straight line of force. While these organs are in the same area, the organs and injuries are not in a straight line.

New information: slides

21. Since the court's verdict was contrary to my review of the slides, I immediately double checked my work. I first took photomicrographs of the slides to a professional conference and asked several other forensic pathologists to review the slides and date the injuries. All of the reviewing pathologists found that the key injuries were at least five days old, with some suggesting that they were even older.

22. I also had some unstained slides that I had not used before the trial since the medical examiner and I agreed on timing. Additional testing is usually done only when there is disagreement. Iron and Masson stains are two of the more common stains used to assist in determining timing. When a slide is stained with iron stain, hemosiderin from breakdown of red blood cells shows up as bright blue. While scattered iron may appear by 72 hours, significant areas or clumps of iron do not appear before 72 hours. The Masson stain makes collagen easier to see and appreciate by turning it bright blue. The presence of collagen indicates that scarring has begun.

23. Since the trial, I have had stains done at my own expense to see if this would shed any further light on timing. The iron and Masson stains have confirmed that the injuries were at least five days old and that some were even older.

24. I have reviewed some of the photographs of these slides with Mr. Escuder, Mr. Liebich's counsel. As I am leaving for India tomorrow, I do not have time to prepare a presentation on specific slides but I am willing to do so on my return or at an evidentiary hearing. I can, however, identify a few of the slides that confirm older injury.

25. On my initial review, slide #4 (bowel) showed many macrophages and very few neutrophils. Fibroblasts were attempting to heal the injury and beginning to form a scar. This placed the injuries at approximately 5 days, plus/minus 1 day. Recent iron staining shows significant iron, confirming that the injury was approximately 5 days old or even older.

26. On my initial review, slide #16 (pancreas) showed a large area of normal pancreatic tissue with an older injury in the surrounding connective tissue with fibroblasts and well-established granulation tissue (scarring). There was some continuation of healing, with a few neutrophils. This placed the injury at more than 5 days. Recent Masson staining shows clear blue staining (collagen or scarring), indicating that the area of granulation tissue is approximately 7-10 days old.

27. On my initial review, slide #19, which appears to be forehead skin, had many fibroblasts and macrophages but no neutrophils. This placed the injury at approximately 5 days. The recent iron stain shows very heavy iron.

28. Iron and/or Masson stains on other slides, including slides # 29 and #33 (bowel) and #37 (liver) produced similar results, confirming that the significant injuries were 5 days old or older.

New evidence: coagulopathy

29. I have recently learned that, based on the laboratory tests, Dr. Michael Laposata, a national expert on coagulation, has confirmed that Steven had pancreatitis and disseminated intravascular coagulation (DIC) at the time of hospital admission.

30. Since DIC causes spontaneous bruising or bruising from trivial trauma, including medical intervention and treatment, DIC would provide a satisfactory explanation for many of the bruises and lines that appeared on Steven's body after hospital admission. Other marks attributed by the State to trauma, including the Cullens sign (a bruise like mark around the umbilicus), are simply indicia of pancreatitis and were recognized as such in the hospital records. The only marks that are concerning to me as an indicator of trauma are the bruises on the lower part of the child's back, which could represent a push or shove, resulting in a crush injury.

31. DIC may also explain the head findings, which consisted of a small subdural or subarachnoid hemorrhage and a hypoxic-ischemic brain. Both of these findings can be caused by a coagulopathy (bleeding/clotting disorder) such as DIC. Since DIC causes thrombosis as well as hemorrhage, the CT scan should be re-read by a qualified pediatric radiologist, with particular emphasis on possible thrombosis.

32. Based on the slides and history, it is possible to suggest the most likely progression of Steven's medical findings. Based on currently available information, it appears that the child had an ischemic bowel beginning at least five days before death, leading to peritonitis, pancreatitis, a small perforation and DIC, with rapid deterioration shortly before presentation to the hospital. The hypoxic-ischemic brain, small subdural hemorrhage and many of the bruises and lines on the child's body are likely secondary to infection and DIC.

33. It is unfortunate that the abdominal CTs and treatment were cancelled since this would have provided more accurate information on the progression of the injuries and may have improved the chances for survival.

Changes in the medical literature

34. There have been major changes in the medical literature since Steven's death in 2002 and the trial in 2004. In the early 2000s, it was widely believed that swollen brains were caused by the traumatic tearing of axons (the nerve fibers that connect the cells of the brain) throughout the brain and that subdural hemorrhages were caused by the traumatic rupture of the bridging veins that connect the brain to the superior sagittal sinus (the large vein that drains the brain). It was further believed that such traumatic tearing would require a major force, often described as equivalent to a major motor vehicle accident or fall from a multistory building.

35. In 2001, a position paper published in the journal of the National Association of Medical Examiners (NAME), the professional organization for forensic pathologists, adopted these hypotheses and suggested that the force was caused by violent shaking. (1). This position paper was not approved by the reviewers and was accompanied by an editorial caveat intended to make clear that it was not an official NAME position paper but rather represented the views of the authors. Despite these red flags, this paper became the foundation of many criminal prosecutions.

36. While this case was not a shaken baby case, the Rush diagnosis included shaken baby syndrome and the state's key trial witnesses relied heavily on the underpinnings of this theory, as set forth above.

37. Soon after the NAME paper was published, a series of research and review papers established that many of the assumptions in this paper were incorrect. Perhaps most important, neuropathological research papers published in 2001 confirmed that the brain swelling in infants was hypoxic-ischemic rather than traumatic and is also found in natural deaths. (2, 3) The same papers found that the subdurals seen in allegedly abused infants are small and thin, and are similarly seen in natural deaths. (2, 3) A third paper, also published in 2001, found that short falls can produce the same findings, disproving the former belief that it required the force of a fall from a multistory building. (4) A 2003 paper published in the NAME journal found that there was no scientific or evidence-based research support for the shaken baby hypothesis. (5). The current consensus is that there are numerous accidental and natural causes for the medical findings previously attributed to shaking or abuse, and that

such findings may be secondary to other injuries or illnesses. (6, 7, 8, 9). There is also considerable consensus that children may have lucid intervals (periods of normality or relative normality) of up to 72 hours after a head injury that ultimately proves fatal. (10, 11)

38. In October 2006, the 2001 NAME position paper expired and the NAME annual meeting included papers with titles such as "The Use of the Triad of Scant Subdural Hemorrhage, Brain Swelling, and Retinal Hemorrhage to Diagnose Non-Accidental Injury Is Not Scientifically Valid." (12)

39. In 2009 and 2010, new research confirmed that the small subdurals seen in allegedly abused children are too small to represent traumatic bridging vein rupture (13) and that retinal hemorrhages are related to brain swelling and life support, rather than the traumatic rupture of retinal veins. (14) Other research has further supported the role of hypoxia. (15)

40. There have been similar changes in our understanding of abdominal injuries. Forensic medicine has long recognized that slow collapse from abdominal injuries is common. This is a well-known phenomenon in children who hit the handlebars of bicycles or are impacted by a seatbelt and who present with abdominal injuries a day or more after the event. (16). In her judgment, the Court stated that there is no lucid interval concept or theory with abdominal injuries and that there is an immediate onset of symptoms. It is my understanding that this was based on the testimony of a pediatric intensivist. In 2006 and 2009, however, the leading textbooks written or edited by child abuse pediatricians confirmed that abdominal injuries may progress slowly. (17, 18)

41. In view of these major changes in the literature, the courts are beginning to review child cases from the late 1990s and early 2000s that were based on misunderstandings of the progression of injury and disease in children. (19, 20, 21) While Mr. Liebich was not accused of shaking Steven, the prosecutors and the prosecution witnesses relied heavily on the tenets of shaken baby syndrome to support their claims, including the outdated beliefs that swollen brains and subdural hemorrhages represent torn axons and ruptured bridging veins, requiring the force of a multistory fall or major motor vehicle accident, and that there are no lucid intervals for head or abdominal injuries.

Conclusion

42. The microscopic slides, including the new stains, establish that the abdominal injuries were five days old or older, putting them outside the period that Mr. Liebich cared for the child. Based on newly available information, including the new literature, it is likely that the small intracranial hemorrhage, the hypoxic-ischemic brain and many of the marks and bruises identified at autopsy were secondary to hypoxia, septicemia, peritonitis (abdominal infection/inflammation) and DIC rather than trauma.

I swear under penalty of perjury that the foregoing is true and correct.

Shaku S. Teas, M.D.

Date: 2/25/12

Rosemary F. Conway

"OFFICIAL SEAL"
ROSEMARY F. CONWAY
NOTARY PUBLIC STATE OF ILLINOIS
My Commission Expires 05/05/2014

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(6) Carole Jenny, Presentation, *The Mechanics: Distinguishing AHT/SBS from Accidents and Other Medical Conditions*, slide 11, New York City Abusive Head Trauma/Shaken Baby Syndrome Training Conference (Sept. 23, 2011) *at* http://www.queensda.org/SBS_Conference/SBC2011.html.

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(12) Scientific Program, 40th Annual Meeting, National Association of Medical Examiners, San Antonio, TX (Oct. 13-18, 2006).

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(15) Marta C. Cohen, Alan Sprigg & Elspeth Whitbey, *Subdural Hemorrhage, Intradural Hemorrhage and Hypoxia in the Pediatric and Perinatal Post Mortem: Are They Related? An Observational Study Combining the Use of Post Mortem Pathology and Magnetic Resonance Imaging*, 200 FORENSIC SCI. INT. 100 (2010).

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(19) Deborah Tuerkheimer, *The Next Innocence Project: Shaken Baby Syndrome and the Criminal Courts,* 87 WASH. UNIV. L. REV. 1 (2011).

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(21) State v. Edmunds, 746 N.W. 2d 590, 596 ¶ 15 (2008).

STATE OF ILLINOIS
IN THE CIRCUIT COURT OF THE EIGHTEENTH JUDICIAL CIRCUIT
DUPAGE COUNTY

Randy Liebich, Petitioner	)	Cir. Ct. No. 02-CF-654
v.	)	Post Conviction No.
People of the State of Illinois	)	

AFFIDAVIT OF GEORGE R. NICHOLS II, M.D.

I, George R. Nichols, II, under oath and penalty of perjury, state as follows:

1. My name is George R. Nichols, II. After obtaining my M.D. from the University of Louisville School of Medicine in 1972, I did a residency in anatomic and clinical pathology at the University of Louisville and affiliated hospitals followed by a fellowship in forensic pathology at the Institute of Forensic Medicine, Cincinnati, Ohio. I am board certified in anatomic, clinical and forensic pathology.

2. I was the Chief Medical Examiner for the Commonwealth of Kentucky from 1977-1997. I have held teaching and academic appointments since 1974 and am currently a Clinical Professor, Section of Forensic Pathology and Clinical Forensic Medicine, Department of Pathology and a Clinical Associate Professor, Department of Pediatrics, at the University of Louisville School of Medicine. I was on the staff in the Department of Pathology, Kosair Children's Hospital, Louisville, Kentucky from 1995-2007 and am currently emeritus staff at the same institution. I am affiliated with Commonwealth Medical Legal Services, Inc., 6013 Brownsboro Park Boulevard, Suite D, Louisville, Kentucky 40207. My C.V. is attached.

3. I have reviewed the following records relating to the fatal injuries of Steven Quinn (DOB 4/17/99; DOD 2/11/02):
   (a) Records of the Office of the Medical Examiner, including the autopsy report; the neuropathology, radiology and surgical pathology reports; the certificate of death; and the investigation report.
   (a) 61 autopsy slides labeled 202-2-02.
   (b) Medical records for Steven Quinn, including Mt. Sinai Hospital records; EMS transport report; Rush-Presbyterian records; and pediatric records. I also reviewed Hinsdale Hospital birth records for Kenyatta Brown and baby girl Brown.
   (c) Hospital and autopsy photographs.
   (d) DCFS records.
   (e) Police reports.

(f) Report of Elizabeth Gilles, M.D.
(g) Report of Shaku Teas, M.D.

4. The contents of this report are based on extensive training and experience in clinical and forensic pathology. My opinions are expressed with reasonable medical and/or scientific certainty or probability.

5. Based on my review of the above materials, with emphasis on the glass microscopic tissue slides and a recent review of the photographs, I conclude:

(a) The child had intra-abdominal blunt force injury that was present *at least* 5 days prior to death.
(b) The child had a hypoxic-ischemic brain and intracranial hemorrhages of undetermined significance, by history and radiologic examination, with extensive operative trauma and resulting anatomic distortion.
(c) Cutaneous (skin) blunt force injury of differing ages.

6. Abdominal injuries. There is acute inflammation in the pancreas associated with hemorrhage in the mesentery occurring at least five days before the removal of life support. The area of necrosis (dead tissue) in the small bowel occurred 5-7 days before removal of life support (slide 33). Other slides show healing responses of 5 days or longer. It is my opinion that the abdominal injuries were caused by blunt force trauma occurring at least 5-7 days before removal of life support.

7. Brain findings. The brain findings reflect hypoxia-ischemia (lack of oxygen to the brain) with progressive cerebral edema. These findings are a delayed reaction to the abdominal injuries and have no independent significance given the extent of the surgery and the time on life support (respirator brain).

8. Cutaneous injuries. The marks in the area of the spine and lower back are consistent with being hit on the back or with falling on the back after being hit, pushed or shoved, and are likely associated with the abdominal injuries. Although they were not noted on hospital presentation, the hospital staff often does not examine the back thoroughly or at all on admission since the priority is to stabilize and treat the child. The absence of a report of an accidental injury of this nature suggests that the injuries were inflicted.

9. The skin slides (slides 18 and 19), one of which appears to be from the forehead area, show reactions approximately 5-7 days old. From the currently available information, it is not possible to determine the areas from which these were taken or their significance.

10. The other marks and lines on the child's body appear consistent with normal childhood bruising, abdominal infection, hospital interventions and/or a secondary coagulopathy.

11. Conclusion. Steven Quinn died from abdominal injuries inflicted at least five days before removal of life support. Based on the histology, it is not possible that the injuries were inflicted three days before removal of life support.

I am giving this affidavit of my own free will. No promises or threats were made to me in exchange for making the statements contained herein. If called to testify, I would testify consistent with this affidavit.

George R. Nichols II, M.D.

Date: March 27th, 2012

Subscribed and sworn to before me this 27th day of March, 2012, by George R. Nichols, II.

My commission expires: 5/4/2012

Thomas O Nichols
Notary Public
State of Kentucky
County of Jefferson

Revised 01/01/11

CURRICULUM VITAE

George R. Nichols II, M.D.

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Louisville, Kentucky 40206

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Louisville, Kentucky 40207
(502) 899-9837 or 899-9838
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E- mail: ***REAPERGRN@AOL.COM***

Date and Place Of Birth: November 22, 1946
Louisville, Kentucky

Marital Status: Married – Janell Seeger, M.D.
Children: Ian, Dillon and Jordan

Education:

1964 – 1968 University of Louisville
Louisville, Kentucky
Degree: B.A. – American History

1968 – 1972 University of Louisville
Degree: M.D.
School of Medicine

Residency

1972 – 1976 Anatomic and Clinical Pathology
University of Louisville
Affiliated Hospitals
Louisville, Kentucky

Fellowship

1976 – 1977	Forensic Pathology Institute of Forensic Medicine Toxicology and Criminalistics Cincinnati, Ohio

Certification

1977	American Board of Pathology, Anatomic & Clinical Pathology
1978	American Board of Pathology, Forensic Pathology

Licensure

1973	Kentucky # 16801
1976	Ohio #39388
1987	Indiana # 01036519 (Lapsed as of 06/30/2003)

ACADEMIC APPOINTMENTS

1974 – 1976	Pathology Instructor (Teaching Resident) University of Louisville School of Medicine
1977 – 1978	Instructor Department of Pathology University of Louisville School of Medicine
1978 – 1983	Assistant Clinical Professor Department of Pathology University of Louisville School of Medicine
1979 – 1985	Associate Member Department of Toxicology & Pharmacology University of Louisville School of Medicine
1981 – 1982	Instructor National College of District Attorney Houston, Texas

1983 – 1991	Associate Clinical Professor Department of Pathology University of Louisville School of Medicine
1981 – 1997	Section Chief, Forensic Pathology and Director of Forensic Pathology Training Department of Pathology University of Louisville School of Medicine
1997 – Aug 1, 2002	Director, Clinical Forensic Medicine Department of Pathology University of Louisville School of Medicine
1991 – Present	Clinical Professor Section of Forensic Pathology And Clinical Forensic Medicine Department of Pathology University of Louisville School of Medicine
1996 – Present	Clinical Associate Professor Department of Pediatrics University of Louisville School of Medicine
Academic Address:	Division of Forensic Pathology University of Louisville 810 Barret Avenue – 7th Floor Louisville, Kentucky 40204 (502) 852-5587 (502) 852-1767 FAX *E- mail:* ***REAPERGRN@AOL.COM***

Non-Academic Appointments

1974	Fellow, American Cancer Society
1975 –1976	Chief Resident Department of Pathology University of Louisville School of Medicine

1976 – 1977	Deputy Coroner Hamilton County, Ohio
1977 – 1997	Chief Medical Examiner Commonwealth of Kentucky Retired September 1, 1997
1977 – 2005	Medical Director of Laboratories International Clinical Laboratories Smith Kline Clinical Laboratory Smith Kline Beecham Clinical Laboratories Quest Diagnostics
1983 – 2006	President and Medical Director Laboratory Physicians, Inc. 2307 Greene Way Louisville, Kentucky 40220 (502) 491-3484
Sept. 1997-Dec 31, 1997	Consulting Forensic Pathologist Office of the Chief Medical Examiner Louisville, Kentucky
1997-Present	President Commonwealth Medical Legal Services, Inc. Brownsboro Office Park 6013 Brownsboro Park Boulevard, Suite D Louisville, Kentucky 40207 (502) 899-9837
1998-2004	Medical Consultant Attorney General Commonwealth of Kentucky Medicaid Fraud and Abuse Unit
1999-2005	Consultant in Laboratory Medicine AdminaStar of Kentucky (Medicare carrier) 9001 Linn Station Road Louisville, KY 40223

Memberships

1972 – 1976	House Staff Association University of Louisville Affiliated Hospitals President, 1974-75

1974 – 1975	Board of Governors Louisville General Hospital
1974 – Present	Jefferson County Medical Society
1974 – Present	Kentucky Medical Association Board of Delegates – 1975-79 Committee on Community and Rural Health – 1984-86
1976 – Present	American Society of Clinical Pathologists Fellow – 1980
1976 – Present	National Association of Medical Examiner Board of Directors 1982 – 1992, 1994 – 1995
1977 – Present	Kentucky Society of Pathologists President - 1983
1977 – Present	American Academy of Forensic Sciences Fellow – 1991
1983 – 1988	Board of Directors Kentucky Alliance for Exploited and Missing Children
1983 – Present	American Association of Clinical Anatomists
1984 – 1985	Commission Member, Kentucky State Child Sexual Abuse and Exploitation Prevention Board
1987 – 1995 and 1998 – Present	Board of Directors Children First Louisville, Kentucky
1990 – Present	College of American Pathologists – Fellow 1990
1991 – Present	Association for Advancement of Automotive Medicine
1996-2003	Jefferson County Task Force to Prevent Domestic Violence Chairman, Domestic Violence Fatality Review Committee

AWARDS

1977	Outstanding Accomplishment in Medical-Legal Issues. Louisville Bar Association
1987 and 1991	Kentucky Attorney General's Certificate of Merit
1990	Individual Achievement Award, Kentucky Organ Donor Affiliates
1993	Outstanding Victim Advocacy, Kentucky Victim's Coalition
1993	Protect A Life Award, Office of Kentucky State Fire Marshall
1995	Silver "Telly" Award, Educational Video Foundation
1995	Vision Award, International Association of Forensic Nurses
1996	Certificate of Appreciation, Kentucky Coalition Against Rape and Sexual Assault
1996	Distinguished Alumnus Award, Alumni Association, School of Medicine, University of Louisville
1997	"The Wolf Award", in recognition of outstanding service in the Coroner/Medical Examiner program. Coroner's Association of Kentucky
1997	Ambassador for the Visually Impaired award presented by the Kentucky Lions Eye Foundation, Inc., the University of Louisville Ophthalmology & Visual Science and University of Louisville Lions Eye Bank
1997	Clinical Forensic Medicine Leadership Award Presented at the Fourth Annual Postgraduate Clinical Forensic Medicine Conference Louisville, Kentucky
1998	Outstanding Crime Victim Advocacy Presented by the Kentucky Victims' Coalition (KVC)

1998	1998 R. Dietz Wolfe Medical Educator Award Presented by the Louisville Consortium for Medical Education

Hospital Appointments:

1977 - 1983	Staff Pathologist Louisville General Hospital Louisville, Kentucky
1983-2008	Staff Pathologist University of Louisville Hospital Louisville, Kentucky
1993 – 1995	Consultant in Pediatric Pathology Department of Pathology Kosair Children's Hospital Louisville, Kentucky
1986-1992	Director of Autopsy Service Department of Pathology University of Louisville Hospital Louisville, Kentucky
1988 – 1995	Consultant in Laboratory Medicine Department of Pathology Louisville Veterans Administration Medical Center Louisville, Kentucky
1995 – 2007	Active Staff Department of Pathology Kosair Children's Hospital Louisville, Kentucky
2008 – Present	Emeritus Staff Department of Pathology Kosair Children's Hospital Louisville, Kentucky
2008 – 2009	Courtesy Staff University of Louisville Hospital Louisville, Kentucky

HONORS

1992 Alpha Omega Alpha, Honor Medical Society

FIELD OF SPECIFIC INTEREST

Mass Fatality Management, Hospital Associated Disorders, Medical Device Related Morbidity and Mortality, Child Abuse, Domestic Violence, Occupational Disorders, Clinical Forensic Medicine. Medical Negligence, Causation Determination.

SPEECHES AND INVITED LECTURESHIPS

Too numerous to list

RESEARCH

Living Forensic Pathology Program, Grant No. 5601-N15A-02/95

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Infrared Videography Grant #5210-N15A-1/95, 1995, $179,125

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Nichols GR, Davis J, Parolla AJ; "Dirty Diving: Sudden Death of a Scuba Diver in a Water Treatment Facility". American J Forensic Med Pathol. 1992

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Smock WS, **Nichols GR,** and Fuller PM, "Development and Implementation of the First Clinical Forensic Training Program, J of Forensic Sciences, Vol 38 (1993)

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BOOK CHAPTERS

Nichols GR. Investigation of Church Bus Collision with Fire and Mass Fatalities. Proceedings of the International Symposium on the Forensic Aspects of Mass Disaster and Crime Scene Reconstruction. U.S. Government Printing House, 1990.

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STATE OF ILLINOIS
IN THE CIRCUIT COURT OF THE EIGHTEENTH JUDICIAL DISTRICT
DUPAGE COUNTY

Randy Liebich, Petitioner	)	
v.	)	Cir. Ct. No. 02-CF-654
State of Illinois, Respondent	)	Post Conviction No.

Affidavit of Dr. Peter J. Stephens

I, Peter J. Stephens, being duly sworn, states as follows:

1. My name is Peter J. Stephens. My address is 100 Club Drive, Suite 135, Burnsville NC 28714 (tel. 828-682-7472). I am a Board Certified Forensic Pathologist with over thirty years experience in hospital (clinical) pathology and forensic pathology. I was Acting Iowa State Medical Examiner from 1984 to 1985 and a Deputy Iowa State Medical Examiner from 1985 to 1995. As such, I testified in cases of child abuse. In 1997 I was consulted in the index case of a series of misdiagnosed alleged "Shaken Baby" cases in Iowa which were subsequently agreed by numerous other forensic pathologists to be due to non-abuse related causes.

2. Since then, I have watched the evolution in Evidence Based Medical Science and the changes that it has brought to the study of pediatric head injury. Since my retirement from full time practice in 2001, I have remained active in this field and have been consulted in four to six similar cases per year. My curriculum vitae is attached.

3. I have reviewed the following information for Steven Quinn (DOD 2/11/02):
   a. Autopsy report, with slides, photographs, radiology report, ophthalmology report, histology notes, and police and hospital reports.
   b. Records for Mt. Sinai Hospital (2/8/02) and Rush Presbyterian Hospital (2/8/02 – 2/11/02).
   c. Photographs from Rush Presbyterian Hospital (2/9/02).
   d. Steven's pediatric records (no pregnancy or birth records available).
   e. Pregnancy and birth records for Steven's half-sister, Angelique.
   f. Trial testimony of Drs. Green, Boykin, Severin, Munoz, Mileusnic and Teas and nurses Letitia Beasley and Tammy Smith.
   g. Various materials from police investigation, DCF investigation, fact witnesses, and court rulings.
   h. The medical records are numbered Med 1-425 and are referenced

accordingly.

4. I hold the opinions expressed in this affidavit to a reasonable degree of medical certainty.

Conclusion

5. Mr. Liebich was convicted of beating his fiancé's son, Steven Quinn, between 10 a.m. and 4 p.m. on February 8, 2002, causing his death. Mr. Liebich and Steven's mother, Kenyatta Brown, took Steven to Mt. Sinai hospital after noticing abnormal breathing, arriving around 6 p.m. The first E.R. doctor did not initially see signs of abuse and thought he was ill. His condition continued to deteriorate, resulting in brain death later that evening. He was removed from life support and officially declared dead shortly after noon on February 11.

6. The medical records confirm that Steven had a severe abdominal infection (peritonitis), leading to systemic inflammatory response (SIRS), sepsis, septic shock and multi-organ failure involving the pulmonary, gastrointestinal, cardiovascular and central nervous systems. He also had hypoxic/ischemic encephalopathy (brain swelling due to lack of oxygen), probably secondary to the abdominal infection. The abdominal infection was confirmed at the hospitals by laboratory tests and serosangous drainage from the abdominal area. The autopsy report confirmed peri-pancreatitis (infection outside the pancreas), pancreatitis, liver damage and a small perforation in the small bowel.

7. Abdominal infections in a young age group are generally associated with impact, either accidental (often bicycle accidents with impact against the handlebars) or inflicted (*e.g.*, punch to the stomach). In this case, the younger age of this child (just under 3 years) and reported rough treatment by the mother suggests inflicted injury, but accidental causes cannot be excluded.

8. Regardless of cause, the pathology establishes that the abdominal infection was present at least 7-10 days before death (4-7 days before collapse and hospital admission). It is not possible that it began as late as February 8, 2002. This infection progressed until the child's collapse on February 8 and continued after hospitalization.

9. As determined at autopsy, the injuries to the brain were hypoxic-ischemic in nature (*i.e.*, due to lack of oxygen). This likely represented a natural progression of the abdominal infection, possibly triggered or aggravated by choking on a hot dog around 3 p.m. on February 8, as described by Mr. Liebich. Choking on hot dogs is a well-recognized cause of death in children under the age of four. While the initial choking event was

apparently resolved, this incident may have set off a chain of interacting hypoxic/ischemic events involving the abdomen and brain.

10. I do not see significant signs of trauma in the hospital or autopsy photographs. Many of the signs interpreted as trauma are well-known indicia of abdominal injuries or artifactual (*i.e.,* attributable to medical interventions). The only significant marks are a series of marks down the child's spine that were small at the first hospital but that grew in size at the second hospital. These cannot be definitively identified as to causality but may have been caused by a fall, accidental or from a push, or other types of pressure. They may also represent hemorrhage from the pre-existing abdominal infection.

11. At this point, it is not possible to determine the origin of the abdominal infection. Based on the evidence, the most one can say is that it began no later than February 6 (and likely earlier), progressing into sepsis, shock and multi-organ failure, as documented in the hospital records.

12. In diagnosing abuse, medical professionals look for disparities between the caretakers' accounts and the medical findings. In this case, the caretaker accounts of the child's reluctance to eat on February 7 and the choking incident on February 8 are consistent with the pre-existing abdominal infection, followed by hypoxia/ischemia.

13. Because it is not possible to determine the cause of the abdominal infection, I would classify the manner of death as undetermined. Given accounts of rough behavior by the mother, one could also reasonably conclude that the abdominal infection likely arose from inflicted trauma occurring a minimum of 5-7 days before death. The medical evidence of inflicted trauma is, however, weak.

Clinical history

14. In diagnosing the cause of death, it is necessary to review the child's medical records and clinical history, starting with prenatal records and continuing through autopsy. In a case that involves a progression or combination of findings, it is critical to determine when the process began, since this determination may have very different legal implications.

15. Prenatal and birth records. Steven's prenatal and birth records are not available. The birth records for Steven's half-sister, Angelique, who was born approximately 10 days before Steven's collapse, indicate that the mother, Kenyatta Brown, received virtually no prenatal care, was suffering from infection at the time of Angelique's birth, and had a history of depression, treated with hospitalization and prozac. Med 339-40, 364. The labs confirmed infection and anemia but were negative for illegal

drugs. (Med 385, 394, 396, 409, 418-22).

16. Pediatric records. Steven was born on April 17, 1999. Initially, he was in approximately the 50th percentile for weight, length and head circumference. Med 224-5. At his four month appointment (9/1/99), his head circumference was slightly over the 50th percentile but his height and weight had increased to well above the 95th percentile. This weight gain was considered excessive. Med 224-5, 227. Steven was seen for checkups and childhood illnesses, including rash, wheezing (treated with albuterol), congestion, conjunctivitis (pink eye), ear infection, possible speech delay, various bumps and rashes (forehead, neck, arms) and scalp lesions diagnosed as ringworm. It is unclear whether he continued to take albuterol for breathing difficulties. Med 219-271.

17. Steven weighed 31 pounds on February 13, 2001 and 35.5 pounds on November 6, 2001. Med 256, 261. On hospital admission on February 8, 2002, his weight was recorded as 14 kg or 30.8 pounds (50th percentile), 3 kg less than his ideal weight for height and nearly 5 pounds less than his recorded weight three months earlier. Med 64-65. There are two possibilities: either the hospital improperly recorded his weight, or Steven lost nearly 5 pounds between November 2001 and February 2002. Since an error of this magnitude is possible but unlikely, this weight loss suggests that Steven was severely dehydrated and/or ill for some time prior to hospital admission.

18. Caretaker reports. The records indicate that Steven was cared for largely by his grandmother and great-aunts, who reported no concerns, with visits to his mother and her boyfriend/fiancé, Randy Liebich. There were no reports of physical discipline or violence against the child by Mr. Liebich, and various witnesses (including Kenyatta's mother) confirmed that Mr. Liebich and Steven appeared to have a good relationship.

19. There are multiple reports of possible abuse by the mother, with one report of shaking and slapping approximately two days before the child's collapse. On the day before hospital admission, the mother described striking the child with a belt over his diaper, hand slaps on the buttocks and possible "muffs" to the head (poking with four fingers) following the child's refusal to eat dinner and continued crying. The trial court found that the mother's actions were non-abusive and that the witnesses who testified to mistreatment by the mother were not credible. The trial court did not find evidence of prior physical discipline or abuse by Mr. Liebich.

20. Symptoms prior to February 8, 2002. Abdominal injuries or infection may have no symptoms at all, or nonspecific symptoms such as lethargy or decreased appetite, for a substantial period of time prior to diagnosis or collapse. In this case, the autopsy slides confirm that the infection started

by February 6 and probably much earlier. While there is virtually no information on this time period, the information that is available confirms that the child was not feeling well by February 7, when he was sent to his room because he refused to eat his dinner (pork chops and mashed potatoes) and was disciplined for crying. The child also had low levels of acetaminophen (Tylenol or Tylenol equivalent) and salicylate (aspirin) in his system on hospital admission, suggesting that he had not been well. Med 25. It is unknown whether these lab reports reflected small doses given on the day of admission or the half-life of larger doses given earlier.

21. In a recent affidavit, Mr. Liebich indicates that Steven wasn't eating well, cried a great deal and was lethargic for at least the last few days of the February 2-8 visit, which Mr. Liebich attributed to jealousy of the new baby. The mother said he had a cold and always cried a lot. Mr. Liebich also states that Steven did not eat his dinner on February 7 and that his plate was still in the refrigerator, covered with plastic wrap, when he was taken to the hospital. The mother said that he initially refused his dinner but ate it later. Scene photographs may help resolve this issue.

22. February 8. Mr. Liebich reported that Steven ate cereal on the morning of February 8 but left the milk. He said he gave Steven a hotdog, juice and water at 3 pm. A police report indicates that there was a plate with a cut up hot dog in the kitchen, suggesting that the child did not eat much.

23. Mr. Liebich told the hospital staff and investigators that Steven choked on the hot dog at about 3 pm and that Mr. Liebich slapped him on the back to dislodge any food and put a finger in his mouth to make sure his airway was clear. In a later interview, Mr. Liebich said that Steven clamped down on Mr. Liebich's finger, and Mr. Liebich slapped or tapped him on the side of his head to get him to release his finger. He then found vomit in Steven's mouth, which he cleaned with a paper towel.

24. When Steven's mother returned from work after 4 p.m., Mr. Liebich and Steven's mother describe an abnormal breathing pattern. Steven's mother described finding some vomit in Steven's mouth, which she encouraged him to spit out.

25. When the abnormal breathing continued, Mr. Liebich and Steven's mother took him to Mr. Sinai. En route, they stopped at Mr. Liebich's work to say that they were taking Steven to the hospital. Mr. Liebich's employer described the child as blinking and having flu-like symptoms, with no noticeable signs of abuse. Laboratory tests taken shortly after arrival at Mt. Sinai confirmed that Steven had a serious abdominal infection.

Hot dog incident and abdominal infection

26. There are two known potential causes of Steven's collapse: the choking incident and the abdominal infection. Before addressing the hospital findings, I will briefly review each.

27. Hot dog incident. What Mr. Liebich described is a classic case of choking on a hot dog. A 1980 report in the Journal of the American Medical Association found that half of the fatalities of children who choked on food were attributable to choking on hot dogs. *Childhood asphyxiation by choking or suffocation,* JAMA 224 (12) (September 1980). The American Association of Pediatrics therefore recommends that hot dogs and carrots be quartered lengthwise and then sliced into small pieces for children under the age of 4. *See also* Bren, L., *Prevent Your Child from Choking,* U.S. Food and Drug Administration (2005) (choking on food may have delayed reaction; recommends keeping hot dogs away from children under the age of 4); Roche, J., *Dangerous Duo: Hotdogs and Toddlers,* Parenting Handout, City of Davis (Nov. 2001) (every week, children choke to death on seemingly innocent foods such as hot dogs and apples; circumference of hot dog is nearly the same size as a child's throat; when a round slice or bite of hot dog is swallowed without being chewed, the piece can lodge in the throat causing suffocation, sometimes within minutes; children under age 4 are most vulnerable).

28. Upper airway obstruction produces the abnormal breathing patterns and other symptoms described by Steven's mother and Mr. Liebich, *i.e.,* changes in consciousness, choking, difficulty breathing, wheezing, crowing, whistling or other unusual breathing noises. Complications include brain damage, breathing failure and death. *See, e.g.,* Acute Upper Airway Obstruction, Penn State Hershey Medical Center (on-line).

29. Since Steven was still breathing, albeit abnormally, on arrival at the hospital three hours later, it is obvious that the choking incident did not cause continuing total airway obstruction, which would have resulted in immediate death. However, it may have caused brief total airway obstruction and/or continuing partial airway obstruction, with stridor (abnormal breathing from airway obstruction) and hypoxia/ischemia (reduced level of oxygen to brain and other organs). *See* Leung and Cho, *Diagnosis of Stridor in Children,* Am. Family Physician (Nov. 1999) (stridor may be first sign of serious and even life-threatening disorder; possible causes include aspiration of food and underlying infection). It is also possible that a brief or partial airway obstruction aggravated the abdominal infection and set off a spiral of hypoxic/ischemic damage to the brain and other organs.

30. Abdominal injuries/infections. Abdominal injuries of the type identified at

autopsy typically arise from impact. In older children, injuries of this type are commonly caused by bicycle accidents, with impact against the handlebars. Symptoms can be immediate or delayed. For example, at a recent conference, a case was presented in which a 9 year old girl hit the handlebars of her bicycle, causing abdominal injuries. She was checked at the hospital and released, vomited that afternoon and the following morning, and was found unresponsive in bed approximately 24 hours after injury. The autopsy found a duodenal transaction with many other abdominal and head injuries, including a depressed skull fracture. Abstract G42, *Prolonged Survival Time Following Duodenal Transection in a Child with Abdominal Trauma,* AAFS (February 2009). Many similar reports are contained in the forensic literature.

31. Inflicted injury can produce the same results. Injuries to the abdomen can be caused by a punch or kick to the abdomen, or any type of crush injury. Absent witnesses or pattern injury, it is difficult if not impossible to determine whether an injury is accidental or inflicted. In this case, the balance may tilt towards inflicted injury because of the age of the child (just under 3) and reports of abusive behavior by the mother, who was caring for the child in the relevant time period. The trial court's determination that the mother was not abusive may tilt in the direction of accidental injury, even if inflicted by the mother.

32. Abdominal infections can also be natural in nature. While pancreatitis and other abdominal infections were once thought to be rare in children, they have been increasingly diagnosed in recent years, with increasing numbers attributed to infection rather than trauma. *See, e.g., Etiology and outcome of acute pancreatitis in infants and toddlers,* J Pediatrics 152(1):106-110 (2008) (87 children under age 3 diagnosed with acute pancreatitis, median age 20 months; pancreatitis associated with multisystem disease in 34% of cases, systemic infection in 16%, idiopathic in 15%, bilary disease in 9%, and trauma in 8%); *Pancreatitis in Children,* J Pediatr Gastroenterol Nutri 37(5):591-5 (2003) (214 cases with 11 deaths; pancreatitis attributed to systemic disease in 14% of cases, trauma in 14%, drug induced in 12%, bilary tract disease in12%, infectious in 8%, idiopathic in 8%); *Acute pancreatitis in childhood: analysis of literature data,* J Clin Gastroenterol 37(2):169-172 (2003) (acute pancreatitis of childhood carries significant morbidity and mortality; etiologies included idiopathic, trauma, structural anomalies, multisystem disease, drugs and toxins, viral infections, and hereditary or metabolic disorders).

33. Foreign objects such as fishbones or coins are also a relatively common cause of abdominal infection. While no foreign objects were found at autopsy, it is possible that an object was swallowed, remained in the bowel long enough to cause infection or the beginning of a perforation,

and was either eliminated prior to autopsy or not discovered at autopsy. *See* Byard & Cohle, Sudden Death in Infancy, Childhood and Adolescence, Cambridge Univ. Press (1994), Ch. 2, Accidental Death, *Foreign body impaction/migration,* pp 28-32 (peak range for choking on foreign bodies is 2-3 years; round food such as hot dogs are most frequently aspirated; symptoms may subside when foreign body moves into the bronchi; diagnosis may be delayed in 30% of cases; only 80% will have positive history for aspiration; chest x-ray may be normal and death may be delayed; with medical attention, foreign object may have been removed and autopsy may not reveal cause of death).

34. Irrespective of cause, abdominal infections may initially have either no symptoms or nonspecific symptoms, such as lethargy, irritability or lack of appetite. The extent and degree of the symptoms depends on the location and the onset of infection. For example, an abdominal injury may be symptom-free until it results in infection or the release of toxic substances into the abdominal cavity. Similarly, pancreatitis may have nonspecific symptoms for weeks or months prior to diagnosis. *See, e.g., Pancreatitis in Childhood: Experience with 15 Cases,* Arch Dis Childh 40:132-145 (1965) (symptoms ranged from intermittent or mild to severe, with duration of days to months); Byard and Cohle at 66-73 (consequences of abdominal trauma include sepsis caused by peritoneal contamination, a late complication, and organ dysfunction, which may be early or late; repeated abdominal trauma over weeks to months may have no signs or symptoms of ruptured viscera until shortly before death).

35. In the absence of abdominal imaging or lab results, abdominal infections, particularly pancreatitis, may be diagnosed by external signs, including the Bryant sign (swollen and discolored testicles), the Cullens sign (redness around the umbilicus), the Gray Turner sign (bruising on the hip or flank) or the Fox sign (bruising on the thighs). These marks can be dramatic in color and resemble the signs of a beating. Cullens and Turner signs are associated with a 37% mortality rate. N Engl J Med 353(13):1386 (2005); 254(9):979-80 (2006). In this case, no abdominal imaging was done, but the abdominal infection was evident from the lab reports (extremely high amylase and lipase) and the presence of the Bryant, Cullens and Gray Turner signs, as confirmed in hospital reports and photographs.

36. The autopsy showed that the abdominal infection was largely contained in the area outside the pancreas (peri-pancreatitis), with recent pancreatitis and a small bowel perforation. It is not possible to tell when the infection entered the pancreas or when the perforation in the bowel wall occurred, though one may reasonably assume that they occurred fairly late in the process, quite likely on February 7-8, when the child was refusing food.

Hospital records: Mt. Sinai

37. In the remainder of this affidavit, I comment on items of interest in the hospital records and trial testimony.

38. Steven arrived at Mt. Sinai hospital at approximately 6 p.m., approximately 3 hours after the choking incident. The initial history indicates that he was brought in because he was lethargic, that he had gone to sleep after choking and vomiting, and that his mother was unable to wake him. Med 5. The final E.R. report describes an irregular breathing pattern (grunting with periods of whining), posturing (a form of seizing), approximately 5 small 1 cm flattened red marks on the abdomen, erythema in the lower back (L2 or lumbar region), and redness on the buttocks. Diagnoses included pancreatic disease, hyperglycemia (an indicator of pancreatitis) and intracranial hemorrhage (which can be traumatic or hypoxic/ischemic). Med 6.

39. The attending doctor, Dr. Green, ordered CTs of the head, abdomen, pelvis and spine and a baby-o-gram (skeletal survey). Med 12. An initial spot check showed exceptionally high glucose (blood sugar), which is associated with diabetes or pancreatitis. Insulin was ordered but reportedly was not given. Med 13. The skeletal survey and CT of the head were taken, but the abdominal CT was cancelled.

40. The nursing notes indicate blood stains and bruising to the lips (not noted elsewhere), a reddened and bruised scrotum (the Bryant sign of pancreatitis), a contusion on the back of the head, multiple bruises to the back, and reddened buttocks. The notes indicate that a choking incident was described, that the child was posturing and nonreactive, and that there was bruising on the back, legs and scrotum "from obvious abuse". The posturing was continuing at the end of the stay at Mt. Sinai. Med 9. Medications were lidocaine, versed, ativan, mannitol, and succinylcholine (a paralytic drug). Med 10.

41. The CT scan of the head showed subdural or subarachnoid hemorrhage on the right with a right intracerebral hemorrhage. There was no midline shift, fracture, or soft tissue swelling. Med 19. These findings suggest hypoxia/ischemia, with no specific indicators of head trauma. The skeletal survey confirmed that there were no skeletal injuries. Med 21.

42. A trauma consultant found bruises on the abdomen and an abrasion on the scrotum, with a head collar in place. Med 14. Based solely on the CT scan, a trauma doctor accused Mr. Liebich of beating Steven and arranged for transfer to Rush for evacuation of a large subdural hemorrhage. As discussed below, this was a misread of the CT scan, which did not show a large subdural hemorrhage.

43. Blood tests taken at 6:22 p.m., shortly after hospital admission, confirmed very high glucose (517, normal range 60-100), amylase (3025, normal range 20-120) and lipase (2368, normal range 22-51). These tests indicate abdominal infection, most likely pancreatitis. The lab tests also confirmed high BUN, creatinine, bun/creatinine ratio, white blood cell count and neutrophils, with low lymphs, indicating infection and kidney involvement. Toxicology tests showed low levels of acetaminophen and salicylate. Urine and prothombin (coagulation) tests were abnormal, with urine showing high protein (over 300), high glucose (250), ketones (15) and blood (moderate blood, RBCs 16). Med 24-38.

44. Comments. Amylase and lipase more than three times normal are generally viewed as indicative of abdominal injury or infection, including pancreatitis. *See, e.g., Increased Lipase Plasma Levels in ICU Patients,* Chest 127(7):7-10 (2005) (amylase and lipase levels are markers of pancreatic inflammation and are standard tests to diagnose acute pancreatitis; GI disease may increase lipase by 2-3 times normal level). In this case, these test results were closer to ten times normal, indicating that Steven was probably septic (*i.e.,* the infection had spread into his bloodstream), placing him at risk of multi-organ failure. The extremely high glucose levels in the blood and urine indicate that the child was likely in hyperglycemic shock, which is associated with cerebral edema and multi-organ failure.

Transport

45. Steven was transported to Rush Presbyterian Hospital at approximately 8 p.m. A pediatric referral note indicates that he had several intracranial hemorrhages (epidural, right subdural and intraparenchymal); a bruise on the temporal region; and small red marks on his abdomen, back and buttocks. He was given mannitol (for brainswelling), phenobarbital (for seizure), ativan, lidocaine and another medication (illegible). The note recommended an abdominal CT scan as well as amylase, lipase and coagulation tests. Med 42.

46. An 8:35 p.m. transport note indicates that the parent(s) were being questioned by the police. The mother gave no history of trauma, said the child was minimally responsive with abnormal breathing when she returned from work, and indicated that the marks on the child's stomach were "old." The note states that the CT scan of the brain showed a large right subdural hemorrhage and intraparenchymal temporal hemorrhage. Other notes, including an 8:50 p.m. pediatric nursing note, indicate bruising on the right side of the head, red diagonal lines across the back, marks on the inner thigh, and red swollen scrotum. Med 43, 45-46.

47. The transport notes indicate that the child was in a c-spine and c-collar with posturing and poorly reactive pupils; bruising over the right temporal and occipital regions; 3 horizontal "rope marks" on the lower back (approx. 5-10 cm long); 2-3 cm red patches in epigastric region; and ecchymosis (redness) on the scrotum, head, back, abdomen and inner upper thighs. All bruises appeared to be of the same age, with signs of extensive trauma likely secondary to child abuse. The child was sedated and likely to go to the operating room for emergency evacuation of the subdural hemorrhage. A CT scan of the abdomen was recommended, as well as an ophthalmology exam for retinal hemorrhage. The family history was positive for asthma and diabetes. Med 47-48.

48. Comment. Once the CT scan was misread, all signs of pancreatitis and sepsis were misinterpreted as traumatic bruising. With pancreatitis, bloody exudates seeping from the pancreas cause apparent bruising, sometimes dramatic in nature, on various body parts. As indicated, in the absence of CT scan and/or lab results, both retroperitoneal infection and pancreatitis may be diagnosed from swollen discolored testicles (Bryant's sign), bruising on the abdomen in the umbilical area (Cullens sign), bruising of the flanks (Turner's sign), and/or bruising of the thighs (Fox's sign). In this case, the Bryant sign (discolored swollen testicles) was misinterpreted throughout the entire hospital stay, beginning when the trauma doctor at Mt. Sinai told Steven's mother that the child had been "kicked in the balls." The Cullens sign was correctly interpreted by one of the doctors at Rush but was repeatedly misinterpreted as trauma by other medical staff.

Hospital records: Rush Presbyterian Hospital

49. At Rush, the admitting diagnosis was head trauma, with no mention of abdominal injury or infection. Med 49. The grandmother consented to a craniotomy with evacuation of subdural hemorrhage, placement of ICP monitor, and possible craniectomy (removal of part of the skull). Med 56-7. The diagnoses on the billing form included subarachnoid and subdural hemorrhage, acidosis, convulsions, acute respiratory failure, retinal hemorrhage, multiple contusions, brain conditions, abnormal blood chemistry and child abuse, with no mention of abdominal injury or infection. Med 50.

50. The pediatric admission assessment states that Steven was admitted to Rush for subdural and intraparenchymal hemorrhage, again with no mention of abdominal injury or infection. His height was above the 95th percentile and his weight was in the 50th percentile, 3 kg less than his ideal weight and 4-5 pounds less than his recorded weight at a pediatric visit the previous November. Bruises were noted on his abdomen (likely Cullens sign), and his urine was orange colored. Med 64-66.

51. Progress notes by a doctor (signature illegible) at 9:30 pm state that Steven was severely beaten before hospital admission and was transferred to Rush for brain hemorrhage. The doctor was unable to examine the patient since the patient was taken to the OR by neurosurgery but recommended CTs of the chest, abdomen and pelvis, with labs (CBC, BMP, amylase and lipase). A note by another doctor (signature illegible) indicates that labs included amylase 1131, lipase 9598, SGOT 5429 and SGPT 3130; plan was to send patient for chest, abdominal and pelvic CTS after surgery and stabilization. Med 73. The blood draw was at 9 p.m. The complete lab report also indicates high glucose (207), with high segs and bands and low lymphs. Med 182-183. A urine test showed abnormalities in protein (high) and blood (large) but was negative for glucose. Med 184.

52. Pre-anesthesia assessment for the neurosurgery began at 9:20 p.m. Med 80. A 10 p.m. note by Dr. Severin indicated a large right subdural hematoma to be surgically evacuated, multiple bruises on the head, and lash marks on the back and buttocks, with no peritoneal signs. Med 75. A chest x-ray at 10:04 p.m. showed possible aspiration pneumonia and/or pulmonary hemorrhage. Med 197.

53. The neurosurgeon's notes indicate that the CT scan showed subdural hemorrhage on the right and subarachnoid hemorrhage throughout, with bilateral ecchymosis (redness). Steven was to be immediately taken to the operating room for evacuation of the subdural hemorrhage. Med 73-74. Steven was transferred to the O.R. at 10:06 and returned to the PICU at 11:20 p.m. Med 80.

54 Neurosurgery operative notes indicate a preoperative diagnosis of right subdural hematoma, with diffuse subarachnoid hemorrhage, severe head trauma and decerebrate posturing. However, the post-operative diagnosis indicates that no subdural hematoma was found. Other notes indicate a small thin subdural hemorrhage, significant subarachnoid hemorrhage, and a severely swollen brain. Med 77-78. Intracranial pressure was very high (over 90). Med 89.

55. Comment. The operative report confirms that the original diagnosis of a large subdural hemorrhage and traumatic head injury was incorrect. Small thin subdurals of the type identified at surgery are found in 48% of asymptomatic newborns and have many possible causes, including infection and/or hypoxia-ischemia. These hemorrhages are not suitable for evacuation. Subarachnoid hemorrhages are not generally associated with trauma and are not suitable for evacuation. Brain swelling is a nonspecific finding that has many possible causes, including infection and/or hypoxia/ischemia. The brain swelling described in the report

indicates that Steven's brain had swollen immensely between hospital admission and surgery, and that he was functionally brain dead by the time of surgery.

56. An 11:45 p.m. note by the neurosurgeon indicates that Steven's eyes were fixed and dilated and that it was his decision to address the brain issues first. Since Steven was very close to brain death, he did not feel that an abdominal evaluation would make any difference. Med 86. Later, a peritoneal (penrose) drain was inserted, and 500 cc of serosanguinous pink (bloody) fluid drained. Med 84A. A note indicates that Steven was a candidate for organ donation, with child abuse and questionable abdominal trauma. Med 87.

57. **February 9.** At about 3:30 a.m., a nurse noted "red lined" marks on top of the left foot and ankle. Med 92. Dr. Severin diagnosed right subdural hematoma, respiratory failure and non-accidental trauma, and a note by Dr. O'Brien indicates that the mother was to be taken into official custody. Med 94-95.

58. A child protective services note referenced retinal hemorrhages, red marks on the scalp, subdural hematoma, extensive linear red marks, erythema around the umbilicus, and signs of intra-abdominal trauma. The child's instability precluded imaging, but the linear marks on the back were photographed. Med 95A.

59. A chest x-ray confirmed a bent or kinked central line tip, with air space opacity possibly due to collapse, infiltrate pneumonia and/or pulmonary hemorrhage. Med 198.

60. Hospital photographs. Hospital photographs taken around 2 a.m. on February 9 show:

- Abdomen: reddened area around umbilicus (Cullens sign)
- Testicles: swollen discolored testicle (Bryant sign)
- Hip: deep red area on right hip and side (possibly also left hip) (Grey Turner sign)
- Back: series of discolorations down spine, with one to the side and larger mark in area of waist, connected by reddened area
- Inner thigh: discolored lines
- Under chin: reddened areas, possible line
- Left foot and toe: dark red discoloration on toe and sole of foot
- Buttocks: reddened.

Comment: The reddened areas on the abdomen, testicles and hip are well-known signs of pancreatitis. The other marks appear to be caused by hospital treatment or intervention. The marks on the inner thigh appear to be related to medical tubes or tape, which are visible in the photographs.

The area of discoloration on the left foot and toe is likely related to the pressure cuff. The redness in the buttocks is nonspecific. I do not see any whip, lash or rope marks, though there are linear lines from the IV, catheter and other tubes that surround the child in the photographs.

The marks of primary interest are the series of round marks down the spine, most of which appear to be the bony prominences. This suggests that they were caused by a fall or pressure on the back, possibly caused during transport to or from Mt. Sinai, during the CT scan (which would have required restraints), or during surgery. Bruising is common with sepsis. It is also possible that these marks represent retroperitoneal hemorrhage from the abdominal infection.

61. Hospital notes indicate that a craniectomy was performed for a right subdural hemorrhage and that both parents were in custody. The chart notes "periumbicial erythema" or "Cullen's sign", with no operative intervention due to brain death. Med 96. Other notes indicate multiple trauma, severe head injury and evidence of bowel injuries, with penrose drain (500 cc of fluid). Med 96A.

62. The medical records refer to "traumatic brain injury secondary to non-accidental trauma – shaken baby syndrome," with references to "bruises/whip marks" and elevated pancreatic and liver tests. A review by the house staff, CPS, neurosurgery, neurology and pediatric surgery notes the Cullens sign, abdominal drainage and "bruises and lash-like marks". Diagnoses are traumatic brain injury-child abuse (rule out shaken baby syndrome), and SIRS shock with multi-organ failure (CNS, hematological, pulmonary, bowel, CV) (rule out sepsis/septic shock). Med 97-99. Blood products and antibiotics were given, with continued abdominal drainage. Notes indicate that child was sent to the OR for drainage of a subdural bleed, and that brain death criteria were met. Med 101, 103-103A, 108.

63. Neurology consult. A neurology consult indicated that the CT scan showed right subdural and subarachnoid hemorrhage with edema. The history includes bruises all over the body with a large bruise on the right temporal region and whip marks on the lower back; retinal hemorrhages; ICP over 90; and fixed and dilated eyes post surgery. The diagnosis was severe blunt head trauma with right subdural and subarachnoid hemorrhage and increased intracranial pressure and probable countercoup injury on the left with massive left cerebral hemispheric edema and obliteration of cisterns. A desperate attempt at lowering ICP by evacuating the subdural hemorrhage failed to halt the process. There was no evidence of brainstem function, with irreversible neurological injury and no significant chance of recovery. Med 105.

64. Ophthalmology consult. An ophthalmology consult found bilateral retinal

hemorrhage, with diagram showing small blots. Med 106.

65. Labs. Sputum collected at 1:45 pm showed many white blood cells, moderate red blood cells and gram negative rods, with heavy growth of klebsiella pneumoniae and enterobacter cloacae. Med 192-4.

66. Social work consult. In a social work interview, the grandmother (Kenyatta's mother) said that Kenyatta told her that Steven started to vomit after she got home from work and that they took him to Mt. Sinai. She said she had never seen a mark on her grandson from Kenyatta or Randy, and that she was in shock that Randy might have done something to Steven. Kenyatta and Randy were being detained by the Dupage County Sheriffs Dept. Med 178-179.

67. **February 10.** On 2/10, Steven failed a second brain death test, and it was decided that that organ harvest would not be pursued. Med 107. Transfusions were continued, with penrose drainage of a large amount of sangous and serosangous fluid. Med 108.

68. The pediatric intensive care unit diagnosed brain death, respiratory failure, and SIRS shock consistent with multi-organ failure. Med 111. The progress notes diagnosed traumatic brain injury secondary to non-accidental trauma – shaken baby syndrome. Med. 109. The left foot continued to have red line marks on top. Med 114.

69. A social work note indicates that Kenyatta expressed a number of times that she would "never in a million years" have thought that this could happen to her and her family. Med 180.

70. **February 11.** On 2/11, the child had thick white/blood tinged secretions from the nose, with extreme swelling all over the body, head and extremities and a slightly swollen foot. The diagnosis was traumatic brain injury secondary to child abuse/shaken baby syndrome, with subdural and intraparenchymal bleed, SIRS and multi-organ failure. Med 116-117.

71. On 2/11, Dr. Scotellano, with CPS attending, repeats the information provided by the child's mother and Mr. Liebich and summarized the child's history, including "a large right subdural hematoma" and intraparenchymal bleed; bilateral retinal hemorrhages; extensive bruising of the scalp, trunk, extremities and scrotum; intra-abdominal trauma; diffuse facial edema; scattered bruising or hyperpigmentation on the back and buttocks; no chest lesions; red scrotum with healing abrasion; and swollen extremities with red lines on left foot. Dr. Scotellano suggested a second review of the Mt. Sinai skeletal survey for fractures and stated that extensive cutaneous and intra-abdominal injuries without a history of trauma are collectively diagnostic of child abuse. "There is no question that this child

was severely beaten and sustained a head injury of extreme force. These injuries cannot be explained by choking on a hot dog." Med 118-119.

72. Hospital notes indicated that the elevated lipase and amylase levels were likely secondary to trauma. Dr. Munoz told the family that the child was physically abused and brain dead. Steven was removed from life support at noon and pronounced dead at 12:24 pm. Med 120-122.

73. Doctors' orders and medications and labs (2/8-2/11). Doctor orders repeatedly state that Steven was admitted for a subdural bleed. During his stay, he was given multiple medications, with blood transfusions and antibiotics beginning 2/9. Med 127-142,145-159.

74. Discharge summary. The discharge summary repeats the history of abnormal breathing and choking on a hot dog and reports large erythematous (red) regions over the right temporal region and posterior occiput. Despite the surgical report, it continues to note "a large subdural hematoma" and brain edema secondary to severe head injury. The report indicates small somewhat ecchymotic (red) areas around the midgastric and periumbicial regions, a reddened and swollen scrotum, multiple whip marks on the mid to lower back and right posterior thigh, and redness on the buttocks. Lab tests showed extraordinarily high amylase, lipase, SGOT and SGPT, with high BUN, creatinine, glucose and other abnormalities. The urine contained high protein (more than 300) and moderate blood. Fluids and blood transfusions were given for organ donation, which was cancelled due to medical examiner priority. Med 60-62, 70-72.

75. Hospital diagnoses. Drs. Kramer and Scotellano agreed that the "large subdural" and intraparenchymal bleed with diffuse brain swelling and edema, bilateral retinal hemorrhage and abdominal injuries were diagnostic of child abuse, and the mother and her fiancé were detained by the DuPage County Sheriff's Department. Med 62. The hospital diagnosis was SIRS (systemic inflammatory response system) shock with multi-organ dysfunction involving the central nervous system, pulmonary, gastrointestinal and cardiovascular systems. Med 61.

76. Munoz letter. In a letter dated May 14, 2002, Dr. Munoz, the neurosurgeon, advised the DuPage County prosecuting attorney that the child's injuries occurred within 6 hours prior to admission to Mt. Sinai. He also said that if the child was talking and eating/drinking just prior to his mother's departure, the injuries could not have occurred prior to that time.

77. Comments. The medical records from Rush confirm severe abdominal infection and pancreatitis, which also explain the brain findings and many of the discolorations. The retinal hemorrhages are consistent with the

raised intracranial pressure and of no independent significance. The original misinterpretation of the CT scan persists throughout the medical records, with repeated references to a large subdural hemorrhage that did not exist. As a result, there are essentially two final diagnoses: a diagnosis of child abuse by a child abuse team, and a formal hospital diagnosis of septic shock followed by multi-organ failure.

Autopsy report

78. The death certificate and autopsy report give the cause of death as multiple injuries due to blunt force trauma due to child abuse, with the manner of death listed as homicide. Med 272, 282.

79. The autopsy report lists the height (39") and weight (44 lbs) as above the 97th percentile, with swelling due to fluid accumulation. Med 273.

80. There is no trauma to the lips or neck, and there are old scars or pigmentation changes to the neck, shoulder, left hand, lower back, left knee and right leg. Med 273-4. There were superficial abrasions and mild to moderate swelling on the right scrotum, consistent with postmortem artifact. Med 274.

81. External evidence of injury included purple/brown contusions on various parts of the body, with clusters on the inner thighs and lower back, and more recent bruises, including 7 purple/red linear lines ranging from 1.2 to 1.7 inches, on the left foot and toe. Med 274-276.

82. Internal abdominal findings were:
- Peritonitis and contusions in duodenum and small and large intestines
- Mesenteric, pericecal, peripancreatic and pancreatic hemorrhage, with superimposed acute traumatic pancreatitis
- Thick peripancreatic hemorrhage with clusters of calcium soaps, possibly consistent with early traumatic pancreatitis
- Focal hemorrhages in the pancreas
- Diffuse hemorrhage and perforation in small bowel, covered with fibrin, with contusion and necrosis
- Hemorrhage and necrosis on 7" of jejunum, with fibrin deposits over several inches of jejunum and mesentery
- Peritoneal hemorrhage in area of terminal ileum and cecum
- Healing subcapsular hematoma to right lobe of liver
- Blood-stained fecal material in large bowel
- Thin layer of retroperitoneal hemorrhage tracking down to scrotum

83. Internal findings on the head and related areas were:
- Healing left sided subgaleal hemorrhage (3" in largest dimension)
- Hematoma and right sided subgaleal from surgical procedure

- Bilateral subdural hemorrhage, with small amount of clotted blood on right and more on left (30 g), with diffuse subarachnoid hemorrhage
- Contusion hemorrhages and necrosis on right cerebral hemisphere
- Severely swollen brain
- Diffuse continuous subdural hemorrhage in spinal cord
- Bilateral optic nerve sheath hemorrhage.
- Intact skull with no scalp lacerations
- No hemorrhage or injury to neck or tongue;

84. Additional findings included:
- Heavy swollen organs, including tracheal and bronchial mucosa, lungs (with marked congestion), heart, liver, spleen and kidneys
- Healing subcutaneous hemorrhages in right arm and wrist and left hand, lower back (lumbar region), right and left buttock extending into right hip, right thigh and leg, and left ankle and foot
- Anasarca (generalized bloating and swelling)

85. There were many artifacts, *i.e.*, signs of medical treatment, including extensive subgaleal hemorrhage associated with the surgical site; an open dura with brain parenchyma oozing through the opening; and needle punctures, superficial impressions and abrasion on the flank from tape.

86. Neuropathology consult. The neuropathology consult found diffuse subdural blood throughout the spinal cord, with no other spinal cord abnormalities. There was residual clotted blood on the inner dura and in the dural sinuses. The brain was severely swollen and necrotic (dead cells), with diffuse subarachnoid hemorrhage and contusion necrosis on the left. There were striking and extensive changes throughout the brain due to hypoxic/ischemic damage (lack of oxygen). The anatomic diagnoses were severe brain edema (swelling); subdural hemorrhage; diffuse hypoxic/ischemic damage with superimposed respirator brain; and contusion necrosis of left cerebral hemisphere. Med 283-4.

87. Radiology. The pre- and mortem x-rays showed no fractures. Med 281. There was possible edema (swelling) of the feet and right leg. Air space disease in middle and left lower lobes of the lung may represent pneumonia or edema.

88. Surgical pathology (eyes). There were small retinal hemorrhages in both eyes, with a large amount of blood surrounding the optic nerves.

89. Autopsy photographs. The autopsy photographs show:
- Abdomen: small bruises of nondescript and non-patterned configuration; not diagnostic.
- Back: reddened marks and lines down lower spine, possible faded marks on upper spine

- Testes: swollen
- Thighs: red marks and indentations on inner thighs when diaper and tubing removed
- Hands: possible bruise at crook of finger
- Left ankle and lower leg: various lines (vertical, cross-crossed)
- Head: various discolorations, swollen lip consistent with edema
- Brain and spinal cord: no adequate gross photographs from neuropathologic examination. Insufficient for diagnosis.

Comments. The swollen testes and marks down the spine are generally consistent with the earlier hospital photographs, though the marks on the spine appear to be more linear, in different positions and lower on the spine at autopsy than in the hospital photos. Some marks identified at the hospital have disappeared while new marks have appeared. This suggests that many of the marks were minor, old or caused by hospital interventions. The linear lines are most likely attributable to tubes and tape shown in the photographs.

The internal abdominal photographs confirm that Steven had a severe abdominal infection, including pancreatitis. The photographs of the brain are of little or no value in determining causation or timing since they were taken after extensive neurosurgery.

90. The microscopic examinations (Med 289-96) showed:
- Central nervous system (CNS): intradural and subdural hemorrhage with admixed macrophages, approximately day 5
- CNS after fixation: edema & congestion; SAH; hypoxic-ischemic & vascular axonal injury; necrosis; early gliosis; recent SAH and SDH with signs of organization, approximately day 5; descending SDH
- Healing subgaleal hemorrhage, approximately day 5
- Intramuscular and subcutaneous hemorrhage with admixed macrophages, approximately day 5
- Heart: ischemic changes and secondary changes due to sepsis
- Lung: congestion and edema; bronchopneumonia
- Liver: subcapsular healing hematoma with surrounding necrosis.
- Pancreas: hemorrhage and numerous inflammatory cells consistent with subacute traumatic pancreatitis; focal necrosis & calcium salts in peripancreatic tissue
- Spleen: changes consistent with early sepsis
- Kidney: no major diagnosis
- Intact testicles.

Comments. In addition to the above findings, sections apparently from the vicinity of the porta hepatis (liver) show fibroblastic proliferation and prominent bile duct proliferation typical of an ascending cholangitis

(bacterial infection) of a minimum 7 to 10 days age.

Histopathological dating of disease processes and injuries must be interpreted with caution since wide variation in rates of healing may occur in the adult and pediatric population.

91. Autopsy sketches. The autopsy sketches show minor marks on the body, including the marks along the lower spine. Med 299. There is also a sketch of the internal findings, consistent with the report. Med 302.

92. Medical examiner case report. This report indicates that Steven was admitted to Rush with the diagnosis of head trauma and suspected child abuse, and was found to have a massive subdural hemorrhage and other marks on his body. The subdural hemorrhage was evacuated on 2/8, and the child was declared brain dead on 2/11. The report indicated that when the mother returned from work, she found the child unresponsive, and the male friend told her that he had choked earlier on a hot dog. Trauma was noted at Mt. Sinai, and the child was transferred to Rush. The mother said the child had a runny nose for the past few weeks but no other cold symptoms. Med 297-98.

93. Police and hospital report . This report indicates the child died from head trauma and that it is being handled as a child abuse case. Med 303-304.

94. Comments. Given the information provided to the medical examiner at autopsy, the conclusion of death from multiple blunt force injuries was reasonable, as are the timing estimates of 5 days prior to death, or 2 days prior to hospital admission. However, it appears that the medical examiner received an incomplete history and information. Specifically, it appears that the medical examiner was not told that no significant subdural hemorrhage was found at surgery, suggesting that the head injuries were secondary to the abdominal infection rather than traumatic. It also appears that the medical examiner was not given the hospital photographs, which showed different markings than seen at autopsy, suggesting that many marks reflected abdominal infection or hospital interventions, rather than inflicted trauma.

Trial testimony

95. At trial, the doctors gave widely disparate opinions on the cause and timing of the medical findings.

96. **Dr. Green.** Dr. Green, the attending doctor at Mt. Sinai, testified that Steven was assigned to the medical side of the E.R. rather than the trauma side. Dr. Green did not see signs of trauma on initial examination and thought this was probably a metabolic problem. The primary

symptom was abnormal breathing (whining and grunting), with posturing. Later, she noticed a flattened red mark on the scalp (sometimes described as a knot), erythematous macular marks (small flat discolorations) 1 cm in size on the abdomen, erythema (redness) in the lower back (L-2 region), and red buttocks. Dr. Green testified that the large round bruises or marks on the back, marks on the neck, and the linear marks on the back, legs and feet seen in the Rush photographs were not seen at Mt. Sinai. She confirmed that some marks seemed to be crease marks from bedding.

97. Dr. Green testified that the child's extremely high glucose level (over 500), could be metabolic or traumatic, and that she was concerned that the child might be in diabetic ketoacidosis or coma. She also ordered head and abdominal CT scans.

98. Dr. Green obtained the head CT results within 30 minutes of hospital admission. The radiology technician was alarmed that the CT scan showed hemorrhage, and Dr. Boykin, a trauma doctor, confirmed the bleed. After the CT scan, the trauma team took over, and Dr. Munoz, a neurosurgeon at Rush, was consulted. Dr. Munoz wanted them to stop what they were doing and send the child to Rush for neurosurgery. The child was intubated after returning from the CT scan since it was felt that he had a head injury. The abdominal CT was cancelled.

99. Dr. Green said that the history is critical for the diagnosis and that she wanted to know what happened just prior to the child's collapse. Mr. Liebich told her the "hot dog story," but she felt this was suspicious and went over the story repeatedly with him. She described Mr. Liebich as ~~restrained and respectful; she simply did not believe~~ "the hot dog story."

100. Dr. Green indicated that the child's brain injuries resulted from ischemia (lack of oxygen to the brain), which can cause bleeding. Dr. Green said that choking on a hot dog could not cause a head bleed.

101. Dr. Green speculated that the linear marks on the back, which she had not seen at Mt. Sinai, could have been caused by a plastic hanger and that the round bruises on the back could be caused by an adult male punching a child on the back, but noted that she is not a forensic pathologist. In medicine, she said, anything is possible.

102. Dr. Green remembered this case because any bleed in a child is trauma. She indicated that a child with a head injury this severe would not be able to eat, talk, walk or play after the traumatic event occurred.

103. Comments. Dr. Green was on the right track in her initial diagnoses and treatment plan. However, the misread of the head CT scan resulted in cancellation of the abdominal CT scan and postponement of the

evaluation or treatment of abdominal injuries or infection. Dr. Green is correct that the brain findings resulted from ischemia (lack of oxygen) and that choking on a hot dog did not explain the entire clinical picture. However, subsequent tests confirmed that the hot dog incident occurred on top of a pre-existing abdominal infection. It is not correct that any bleed in a child represents trauma. In fact, there are many alternative diagnoses, including infection and hypoxia/ischemia. Her comments on the severity of the head injury reflects the misread of the CT scan, which shows a small thin subdural of a type seen in 47% of healthy asymptomatic newborns, rather than the massive subdural described by the technician and trauma doctor.

104. **Dr. Boykin.** Dr. Boykin, a trauma doctor at Mr. Sinai, testified that when she saw Steven entering the hospital, she thought he might be suffering from a febrile seizure. He was not initially assigned to the trauma side of the emergency department, but shortly after arrival Dr. Boykin confirmed that he was posturing. Dr. Green initially got a high blood sugar and thought that that the child's problem might be metabolic. However, a radiology technician said that the child's head was "full of blood," and Dr. Boykin agreed. Dr. Boykin did not know the results of the neurosurgery.

105. Dr. Boykin immediately confronted Mr. Liebich and told him that Steven had suffered a severe brain injury secondary to trauma and that it was not caused by choking on a hot dog. She was very angry and told Mr. Liebich that it appeared he had been "sitting at home beating on [the child] all day." At one point, Mr. Liebich yawned, which confirmed Dr. Boykin's suspicions. Dr. Boykin did not ask about events prior to February 8 since she felt Steven would have been dead if the injuries had occurred earlier.

106. Dr. Boykin testified that if a child has severe abdominal injuries and blood is not getting to the brain, this would cause serious problems for the brain and other organs and that the child would likely suffer hypoxia (lack of oxygen) and hemorrhagic shock. She did not know whether much attention had been paid to the abdominal region.

107. Comments. The radiology technician and Dr. Boykin misread the CT scan, which did not show a massive subdural, as confirmed in surgery. The radiology report has no specific indicators of trauma, such as skull fracture or soft tissue swelling, and is consistent with hypoxia/ischemia. Dr. Boykin is correct that abdominal injuries would lead to shock and hypoxia, with secondary consequences for the brain and other organs.

108. **Letitia Beasley, R.N.** Ms. Beasley, an emergency nurse at Mt. Sinai, testified that Steven had abnormal breathing, but no airway obstruction was identified. She saw bruising in the scrotal area and a bruise on the right side of the head with a knot formation. Ms. Beasley testified that

many marks in the Rush photographs, including the linear red marks on the thigh, were not present at Mt. Sinai.

109. When Dr. Green questioned Mr. Liebich on the events of the day, he said that Steven had eaten a hot dog, choked, and slept. She did not recall any mention of vomiting.

110. Comments. The bruising in the scrotal area was a sign of pancreatitis. The reddened area and knot on the right side of the head are of undetermined significance since the mother reported that the child had pre-existing knots on both sides of the head. The absence of the linear red marks at Mt. Sinai indicates that these were probably from medical treatment, most likely tubing. The appearance and disappearance of red marks in multiple locations throughout child's hospital stay suggests that these were related to sepsis (infection), rather than abuse.

111. **Tammy Smith, R.N.** Ms. Smith, an agency nurse, helped transport Steven from Mt. Sinai to Rush and cared for him at Rush. Ms. Smith was surprised that they were transporting Steven to Rush since she felt he was already dead. Steven had a swollen scrotum, a reddened area on the side of his head, small red bruises on the abdomen, lines on his inner thighs, and two diagonal lines and some bruising on his back. At Rush, some marks (including "dots" on the abdomen and lines on the inner thigh and back) became larger or more defined during the night. Others (including a large mark on the abdomen, marks under the chin, and lines on the left foot) appeared for the first time at Rush. Ms. Smith testified that the transport team arrived at Mt. Sinai about 8 p.m. and left for Rush at 8:50, arriving around 9 p.m. Steven went into surgery around 10 p.m.

112. *Comments.* Ms. Smith confirms the swollen testicles, which are the Bryant sign of pancreatitis. Her descriptions of red marks and lines that appeared at various stages indicates that these were associated with pancreatitis, sepsis and medical interventions, rather than abuse.

113. **Dr. Severin.** Dr. Severin is a pediatric critical care doctor at Rush who had treated relatively few child abuse victims. He testified that Steven appeared to be critically ill and in shock on arrival at Rush, and that he observed posturing. There were long linear red marks along the left thigh, a very swollen scrotum (more on the right than the left), some bruises on the back, and a bruise on the right side of the head. There was some bruising on the abdomen but nothing out of the ordinary, and he did not think there were abdominal injuries. All bruises appeared to be about the same age. The major initial concern was with the head since the CT scan indicated that there was a lot of blood in the brain and that he would die unless the pressure was relieved.

114. Dr. Severin ordered lab tests, which showed very high pancreatic enzymes and poor liver function, raising concern with the abdomen. He also noted the Cullens sign (bruising around the umbilicus), which is a sign of pancreatitis. Dr. Severin testified that these findings were consistent with the abdominal infection identified at autopsy. He also testified that the abdominal injuries alone would have been sufficient to cause death.

115. Dr. Severin testified that he would not expect a child to be able to eat after receiving these injuries and that the onset of symptoms would be immediate. He testified that the amount of force required to cause these injuries would be force such as falling from a 20-30 foot height, a motor vehicle accident, or blunt trauma such as a fist or foot.

116. Dr. Severin testified that Steven's head injuries likely occurred within 4-6 hours of hospital admission since he did not believe that anyone could survive for a day with these injuries. As a pediatrician, he was rarely called upon to time injuries, and this was the first time he had done so. He testified that lack of oxygen in the brain could cause hemorrhaging, and that there was considerable pressure in the head. Dr. Severin testified that, according to Dr. Munoz, Steven's head injury was very severe, and that the subarachnoid bleed was so large that it looked like a subdural. He testified that his opinion on the timing of injuries would be unchanged even if the pathology showed the injuries were older.

117. Dr. Severin testified that the bruises were defused (i.e., none stood out more than others) and that they probably occurred within 1-2 days (24-48 hours) of hospital admission, possibly on the 6th or 7th. He testified that if the pathologist testified that the microscopic slides showed the injuries to be 5 days old, he would disagree since abdominal injuries do not take several days to become symptomatic. He agreed that the Rush radiologist felt that the chest x-rays were suspicious for pneumonia (possibly aspiration pneumonia), *i.e.,* aspiration of food into the lungs.

118. Dr. Severin testified that Steven's injuries were not consistent with choking on a hot dog. He also testified that the pain would have been excruciating if Steven had pancreatitis prior to eating on the 7th or 8th. He therefore felt that the pancreatic process had just started.

119. Comments. Dr. Severin recognized the signs of shock and abdominal infection (including the Cullens sign) but was misled by a misinterpretation of the CT scan, which was not in his area of expertise. His testimony on the head injuries is inconsistent with the pediatric head injury literature, which finds that symptoms do not necessarily begin when an impact occurs but when brain swelling begins, which can be as much as 48-72 hours later. Dr. Severin also assumed that the head injuries were caused

by impact. In this case, however, they are more likely secondary to the abdominal infection, possibly aggravated by the choking incident. Dr. Severin's testimony that abdominal infections or injuries are immediately symptomatic is not supported by the literature, which uniformly states that abdominal injuries are often slow to manifest themselves.

120. **Dr. Mileusnic.** Dr. Mileusnic, the medical examiner, testified that Steven was well developed, well nourished and in the 97th percentile for height and weight, though his weight was affected by anasarca (swelling and fluid accumulation from the hospital).

121. Dr. Mileusnic went through the bruises identified in the autopsy report. She testified that she had not seen the hospital photographs prior to the day of testimony and that the differences between the hospital photographs and autopsy findings were peculiar. The bruises on the upper back and the larger bruise slightly above the waist shown in the hospital photographs were not present at autopsy. In addition, the hospital photographs showed linear bruising that was not present at autopsy, and the autopsy showed linear bruising that was not present at Rush. She testified that the linear lines were consistent with being whipped with a hanger or narrow object. She suggested that perhaps the linear marks at Rush were superficial and had healed by autopsy, and that the marks at autopsy were possibly deeper and took longer to arise from within the deeper tissue. She noted that the lines on the scrotum were proximate to the IV and tape shown in the photographs and were probably artifacts.

122. Dr. Mileusnic testified that there was a thin layer of subarachnoid hemorrhage over the brain, with a larger subdural on the left that extended down the spinal cord. It was difficult to determine when these occurred given the neurosurgery. She testified that these findings were consistent with blunt force trauma to the head and severe brain injury.

123. Dr. Mileusnic described the abdominal findings, including inflammation, peritonitis, a perforation in the small bowel, and healing responses. She felt that the perforation caused peritonitis and hemorrhage in surrounding areas, and that it resulted from blunt force trauma. There was a healing subcapsular hematoma to the liver. There was also blood tracking down from the abdomen to the testicles or scrotum, with no injury to the testicles or scrotum.

124. Dr. Mileusnic testified that the cause of death was multiple blunt force trauma, but that the case was more complex because of the abdominal injuries. She testified that, with head trauma, there can be 24-48 hour delays before symptoms appear, and that brain swelling may be a late manifestation of a focal head injury. Dr. Mileusnic testified that it is easier to time head injuries, and that the pathology indicated that the

hemorrhages around the brain occurred approximately 5 days before death, plus or minus a day, maybe 2 days, but she was more comfortable with 24 hours before or after. She indicated that the injuries could have occurred on the 8th but could also have occurred as early as the 5th.

125. Dr. Mileusnic testified that it is harder to time abdominal injuries since the bowel deteriorates rapidly after death and abdominal injuries are notorious for developing slowly, with late manifestations. Eventually, such injuries lead to compromised circulation, sepsis and/or shock, and brain edema (swelling), even without head injury. She testified that the abdominal injuries probably occurred around the same time as the head injuries.

126. Dr. Mileusnic testified that some of the head injuries were in the third level of healing, with fibroblasts, which would place them 5-7 days before death (*i.e.,* on the 4th to 6th). The early pancreatitis could be from blunt trauma or part of a natural process involving breaking of the cell membranes. Symptoms might not occur until rupture.

127. Dr. Mileusnic was uncomfortable dating bruises based on appearance, with estimates generally given within a day or two.

128. Dr. Mileusnic testified that hypoxia could aggravate an original injury, and that even a seemingly mild injury could lead to later deterioration due to a cascade of reactions in the brain. She testified that it is well-known that hypoxia (lack of oxygen) can cause thin layer subdural or subarachnoid hemorrhage and brain damage. However, in this case, there was an area of impact, specifically, a subgaleal hemorrhage (hemorrhage directly under the scalp) on the left that was approximately 5 days old. In general, her best estimate was that the head injuries occurred 5 days prior to death, plus or minus a day or two.

129. Because there were so many interacting factors, she gave the cause of death as multiple blunt force trauma. If there was a head injury, she would not be able to tell if this was abuse or accident. She further testified that she would defer to the neurosurgeon on the timing of the injury and what he saw in the brain, and to the pediatric intensivist on what he saw in his examinations and learned in talking to the family.

130. *Comments.* I agree with Dr. Mileusnic that some of the injuries were 5-7 days prior to death, or older. Others may reflect the ongoing process of infection and hypoxia/ischemia.

I think that some of the confusion in this case arose because Dr. Mileusnic was not given the hospital photographs or told that the CT scan had been misread and no significant subdural hemorrhage was found during surgery. The appearance and disappearance of bruises and other marks

indicates that they resulted from medical interventions or the settling of blood, rather than abuse. The absence of a significant subdural hemorrhage indicates that the brain findings were secondary to the abdominal infection, possibly aggravated or triggered by the choking incident, rather than traumatic.

I agree that a head injury may not become apparent for 48 hours after injury (sometimes longer) and that abdominal injuries may take even longer to become symptomatic. This is all well established in the literature. I also agree that the abdominal infection could cause the brain damage, and that the early pancreatitis and bowel perforation could be traumatic or part of the natural process of infection, which can result in a breakdown of the cell walls. Without abdominal CT scans and with the major changes in the brain caused by neurosurgery, it is not possible to determine the precise progression of the injury and/or infection.

As Dr. Mileusnic's testimony suggests, there is no significant pathological dispute on the timing of the injuries. Some of the injuries are a minimum of 5-7 days old (closer to 7-10 days in area of the liver), with secondary findings in the 3-7 day range, consistent with an ongoing process of infection and hypoxia/ischemia.

131. **Dr. Munoz.** Dr. Munoz, the Rush neurosurgeon, testified that the child was posturing on arrival at Rush and that the subdural hemorrhage shown on the CT scan was highly suggestive of severe head trauma caused by a shearing of the bridging veins resulting from a mismatch between the movement of the brain and skull. In surgery, the brain was very swollen and started to come out of the skull opening, and the blood clot was "massive" and red. He testified that the brain swelling was comparable to that seen in children who fall out of 3-4 story buildings or who are in horrible car accidents.

132. He confirmed that in surgery, there was not as much subdural hemorrhage as expected but that the subarachnoid hemorrhage was massive and could not be evacuated. The amount of red blood meant it was recent. He testified that it was impossible for the injury to have occurred on the evening of the 7th as the child would have been unable to walk or talk. He also testified that it is impossible to have a head injury that would allow one to talk and later die.

133. Dr. Munoz testified that visualization of the blood clot is superior to pathology and is the "gold standard" for timing. He testified that he can time an injury by looking at the brain and blood clots at surgery, and that his best estimate was that the head injury occurred within 6 hours of arriving at Mt. Sinai. He also testified that all body parts are related, and that what happens to the liver also affects the lungs and brain.

134. Comments. Dr. Munoz confirmed that he did not find a significant subdural during surgery but instead found a vastly swollen brain and large subarachnoid hemorrhage that could not be evacuated. I agree with Dr. Munoz that a child with severe brain swelling would not be able to walk or talk. However, brain swelling may not begin for 48-72 hours after impact. It may also be the culmination of natural processes, including septic shock, hypoxia/ischemia and multiorgan failure. I disagree that the subarachnoid hemorrhage was massive. As shown in the autopsy photographs and as Dr. Mileusnic repeatedly testified, the subarachnoid hemorrhage was thin. Since a subarachnoid hemorrhage cannot be evacuated, the autopsy finding is dispositive.

In this case, the pathology establishes that the abdominal injury or infection began 5-7 days prior to death, or earlier. The hot dog incident may have been a symptom of this injury since the child would have found eating uncomfortable. It may also have triggered hypoxia/ischemia, as evidenced by abnormal breathing, with decreased oxygen to all parts of the body, including the brain. Lack of oxygen would have resulted in a hypoxic swollen brain and breakdown in the cellular walls of the bowels and pancreas, leading to sepsis and septic shock.

Dr. Munoz' testimony that the brain findings are comparable to what is seen in children who fall out of 3-4 story buildings or are struck by cars is misleading. Brain swelling is the response of a brain that is injured or deprived of oxygen. It can certainly occur in major accidents, but it can also occur from minor impact or natural causes.

Dr. Munoz is incorrect that it is possible to determine when an injury occurred simply by looking at the brain or blood clot with the naked eye. I know of no textbook or literature that allows neurosurgeons or forensic pathologists to date an injury within hours or even days based on the color of blood. Dr. Munoz is correct that, in a child, once brain swelling begins, the progress may be rapid and that the brain swelling seen at surgery likely began shortly before or after hospital admission. To determine when the original injury occurred, however, one must turn to MRIs and autopsy slides. In this case, no MRI was taken. However, the autopsy slides establish that the abdominal infection began at least 5-7 days prior to death, with secondary effects on the brain.

135. **Dr. Teas.** Dr. Teas, a forensic pathologist, testified for the defense. Dr. Teas did not challenge the findings of death from multiple blunt force impact, which had already been conceded by the defense. Instead, since the issue was when the injuries occurred, Dr. Teas focused on timing. From a pathology standpoint, timing is based on histology, *i.e.*, microscopic examination of tissues to determine the healing responses.

136. Dr. Teas concurred with Dr. Mileusnic that the injuries occurred approximately 5-7 days prior to death. Based on the presence of spindle cells, granulation tissue, layers of fibroblasts and beginning capillary formation, Dr. Teas testified that the abdominal injuries appeared to be 5-7 days old and could not have occurred less than 4 days prior to death.

137. Dr. Teas testified that there may be some period of time before an abdominal injury becomes symptomatic. Following an abdominal injury, the tissue in the area of injury may become necrotic, causing a perforation in the wall and leakage of bowel contents into the peritoneal cavity, causing localized infection, which may in turn lead to sepsis and multi-organ failure. In determining when an injury occurred, it is important to look at the oldest part of the injury. Dr. Teas testified that, in this case, the abdominal injury could have been caused by punching or hitting the abdomen, pushing from the back, or any type of crushing injury.

138. Dr. Teas testified that head injuries similarly may not become symptomatic until the brain begins to swell. In this case, Dr. Teas suggested that the brain findings might represent different stages of healing.

139. Dr. Teas noted that some bruises and marks were consistent with being struck by a belt or hanger but that some could be from hospital equipment, including the pressure cuff and tubes. Dr. Teas testified that choking on the hot dog was likely a symptom of the developing abdominal infection, which would have made it difficult to eat.

140. Comment. I agree with Dr. Teas that, at the time of death, the abdominal infection was at least 5-7 days old (with some findings in the 7-10 day range), and that it could not have been 4 days old. This is consistent with Dr. Mileusnic's testimony, which found the subgaleal hemorrhage to be 5-7 days old and the abdominal injuries to be likely in a comparable timeframe. It is not possible based on the medical evidence to determine whether the abdominal infection was caused by inflicted trauma, accidental trauma or even natural causes. Given the child's age and history, I would be inclined towards inflicted trauma; however, if the trial court's conclusion that the mother was non-abusive is correct, accidental trauma and/or natural causes are also real possibilities. The injury or infection may occurred earlier in the week when the child was with his mother, or prior to arrival on February 2.

General Comments

141. In many cases, the pathology simply confirms what is already known, *e.g.*, that the child died in a car accident half an hour before hospital admission. Other times, the pathology tells a different and sometimes surprising story.

In this case, several doctors were convinced that Mr. Liebich beat Steven on February 8, causing abdominal injuries, brain damage and death. However, the pathology establishes that the abdominal injuries and/or infection pre-dated February 8 by at least 2-4 days, and probably longer.

142. There are, moreover, no significant signs of a beating. Significantly, the first people to see the child after his collapse – his mother, Mr. Liebich, Mr. Liebich's employer and the emergency room doctor – did not see signs of trauma or abuse. Instead, they noticed abnormal breathing and thought he was ill. For the most part, the external marks were classic signs of pancreatitis or artifacts, combined with the normal accumulation of bruises and scars in active 3-year-olds, particularly those with dark skin, who have natural variations in pigmentation and scar easily.

143. Since an abdominal CT was not taken, it is not possible to accurately reconstruct the course of this child's injuries or illness, particularly given the impact of neurosurgery. However, it is possible to say with certainty that the abdominal infection started at least 5-7 days prior to death, and possibly earlier. Since there is no significant evidence of head injury, the brain findings were likely secondary to abdominal infection and may have been triggered by hypoxia/ischemia caused by choking on a hot dog.

Problem areas

144. The problem areas can be summarized as follows:
   a. major changes in the medical and scientific literature on pediatric head injuries over the past 10 years.
   b. the failure of the medical professionals to consider the child's clinical history, laboratory reports and autopsy findings.
   c. the lack of a considered differential diagnosis.

145. I will discuss these problems in sequence and will then summarize my conclusions. References are attached.

Changes in basic scientific understanding in the past 10 years

146. Ten years ago, it was widely believed that subdural hemorrhages, retinal hemorrhages and brain swelling ("the triad") were diagnostic of major impact or violent shaking ("shaken baby syndrome" or SBS). It was also believed that children who suffered head injury were immediately symptomatic, that short distance falls or relatively minor impacts could not cause head injury, and that natural disease processes rarely if ever produced the triad. Given these beliefs, if a caretaker could not describe a motor vehicle accident, fall from a multi-story building or comparable catastrophic event occurring immediately before hospital admission, it was assumed that the caretaker must have intentionally injured the child.

147. In the past decade, these beliefs have been challenged and in many instances disproven. Today, it is well-understood that the triad is also found in accidental injuries and a wide array of natural disease processes.

148. Shaking theory. The concept of shaking injury (was developed in the early 1970s to explain a small subset of children dying of apparent head injury in whom no impact site could be identified. This concept originated with Caffey and Guthkelch *as a hypothesis*, but was never proven. This hypothesis was attractive to physicians because it was a simple but plausible explanation for unexplained findings. However, it was never accepted by biomechanicians or many leading forensic pathologists.

149. For years, pediatric head injury theory was based on key SBS principles, namely, that: (1) subdural hemorrhages are caused by torn bridging veins, requiring extreme force comparable to fatal motor vehicle accidents or falls from multi-story buildings; (2) subdural hemorrhages indicate that the axons throughout the brain have been torn (diffuse axonal injury), requiring extreme force and causing severe brain damage; (3) the triad is caused by extreme impact or violent shaking, with few if any accidental or natural causes; and (4) all head injuries are immediately symptomatic. These beliefs were never evidence-based, and they have all been discredited or disproven.

150. Biomechanical studies. Biomechanical studies using computer modeling and anthropomorphic (CRABI) dummies have repeatedly confirmed that even the most violent shaking cannot create sufficient force to cross the injury threshold for subdural hemorrhage, while even minor impacts (including short falls) do cross this threshold. Much of this information was available by 1987, but its significance was not recognized. More recent work has established that the forces needed to cause a subdural hemorrhage through shaking would cause severe neck damage, which is rarely seen in allegedly shaken babies. This work is consistent with witnessed short falls causing fatal head injury.

151. Neuropathology. The first neuropathological studies of the brains of children who reportedly died from nonaccidental head trauma were published in 2001. The researchers expected to find diffuse axonal injury, but instead found hypoxia-ischemia (lack of oxygen). These studies also found that the thin subdural hemorrhages found in allegedly abused children were also present in children who died natural deaths.

152. Onset of symptoms. The belief that all children with fatal head injury collapsed immediately resulted in the theory of "no lucid interval" and the assumption that the adult who was present at the onset of symptoms must have caused the injury or death. Since 1994, numerous medical journal

articles have concluded that there is considerable variation in timing, with no scientific evidence to indicate whether the symptoms would occur immediately or be delayed following any given impact. In 1999, Gilliland found intervals of 72 hours or more between head injury and collapse, often with nonspecific symptoms, such as lethargy. Similarly, in 2003, Denton and Mileusnic reported a child who initially appeared alright after a short fall but became symptomatic and died three days later.

153. Radiology. When Caffey and Guthkelch developed their hypothesis in the 1970s, the only x-ray procedures available to determine whether a child had a subdural hemorrhage were plain x-rays of the head or procedures that were so invasive as to be dangerous to the patient. With the advent of CT scans and MRIs, however, it became apparent that there are many alternative (differential) diagnoses for subdural hemorrhages, particularly thin subdurals. Alternative diagnoses identified by Professor Patrick Barnes of Stanford University include accidental or birth trauma, hypoxia-ischemia (lack of oxygen), cardiopulmonary resuscitation, infection, vascular diseases, coagulopathies (bleeding disorders), venous thrombosis, metabolic disorders, neoplastic processes, certain therapies, and other conditions. These causes cannot be distinguished on CT scan.

154. Child abuse literature. By 2002, an article by leading child abuse pediatricians listed the alternative diagnoses for the symptoms previously identified with abusive head trauma as accidental trauma; medical or surgical interventions; prenatal, perinatal and pregnancy-related conditions; birth trauma; metabolic, genetic, oncologic or infectious diseases; congenital malformations; autoimmune disorders; clotting disorders; the effects of poison, toxins or drugs; and other miscellaneous conditions. Many of these entities can be excluded or confirmed by careful history, physical examinations, radiological studies and/or laboratory testing. A more recent book on child abuse devotes two chapters to accidental and natural causes.

155. Retinal hemorrhages. Until approximately 2004, it was widely believed that retinal hemorrhages were caused by abusive injury. This dogma still persists despite the fact that Terson identified increased intracranial pressure as a cause of retinal hemorrhages several decades ago. However, by examining the eyes in every autopsy using postmortem monocular indirect ophthalmoscopy, Dr. Patrick Lantz of Wake Forest Medical Center confirmed that retinal hemorrhages are found in a wide array of accidental injuries and natural disease processes.

156. Brain damage. Until recently, it was widely believed that subdural hemorrhages were markers of brain damage. However, recent studies have shown that 47% of healthy *asymptomatic* newborns have thin subdural hemorrhages. It is unknown whether these hemorrhages are

caused by birth trauma, hypoxia/ischemia (lack of oxygen) during birth, or some other cause entirely. These hemorrhages typically resolve within a month and are not associated with brain damage.

157. Bridging vein theory. Until recently, it was believed that subdural hemorrhages were caused by ruptured bridging veins. However, recent work suggests that thin subdural hemorrhages are not related to ruptured bridging veins at all, but consist of intradural leakage, possibly natural in origin. It is now believed that the paraphysiology involves some combination of increased intracranial pressure, increased venous pressure, hypotension or hypertension, vascular fragility, hematologic derangement and/or collagenopathy, superimposed on an immature central nervous system.

158. Brainswelling. Brainswelling is a nonspecific response of a brain that is injured by trauma or lack of oxygen from any cause, including those listed above. In 2001, a symposium convened by the National Institute of Health concluded that we do not yet understand this process. This is equally true today, though we do know that once the process begins, it is likely to proceed much more rapidly than in adults.

159. Lack of evidence base. Under evidence based medicine, the validity of theories and/or treatment plans is reviewed based on the quality of the supporting evidence. Reviews of the shaken baby literature in 2003 and later have established that existing theories of pediatric head injury, including shaken baby syndrome, are not supported by reliable evidence.

160. Legal implications. As a result of new research and the application of evidence based medicine to pediatric head injury, some Courts are beginning to reexamine earlier convictions. For example, in a recent Wisconsin shaken baby case in which I testified, the Wisconsin courts granted post-conviction relief to Audrey Edmunds, who had been convicted of shaking a child in her care, causing death, holding that she was entitled to a new trial based on advances in medical knowledge. After reviewing the evidence, the charges were dropped.

161. Convictions on pediatric head injury and deaths are also being revisited in the United Kingdom and Canada. In the Goudge Inquiry in Ontario, Canada, Justice Stephen Goudge issued a 1,000 page report on October 1, 2008 finding systemic flawed pathology and misdiagnoses of child deaths in pediatric head injury and shaken baby cases. In calling for a review of more than 200 convictions, Justice Goudge emphasized the advances in medical knowledge since the 1990s.

162. In this case, much of the trial testimony reflects the accepted dogma of the late 1990s, much of which is no longer accepted or has been disproven.

Specifically, it is now understood that Steven's brain findings, including a thin subdural hemorrhage, retinal hemorrhage and brain swelling, can also be accidental in nature or secondary to infection or hypoxia/ischemia. In addition, it is well understood that brain swelling – which is the real problem – may not occur for 48-72 hours after injury.

Premature diagnosis: failure to consider the clinical history, laboratory reports and autopsy findings.

163. In this case, a premature diagnosis of child abuse led to a failure to adequately consider the objective medical data or investigate the relevant time period. Any diagnosis of child abuse requires a complete review of the medical records, laboratory tests, and clinical history, with incorporation of new evidence as it becomes available. In this case, the medical staff diagnosed abusive head trauma and a fatal beating within approximately 30 minutes of hospital admission based on a misread CT scan. While understandable, this diagnosis was scientifically invalid and did not take into account the objective medical data, which confirmed severe abdominal infection.

164. The medical records further indicate that the preliminary diagnosis was never adjusted to reflect new evidence. For example, the initial claim that the CT scan showed a "massive" subdural hemorrhage continued throughout the hospital stay and into the trial testimony despite surgery notes showing that no subdural, or a very small subdural, was found during surgery. Similarly, despite pathology slides establishing that the abdominal infection was at least 5-7 days old, several doctors testified that the abdominal injuries had occurred within hours of hospital admission.

165. The premature diagnosis of head injury immediately preceding hospitalization further limited the scope of the medical and legal investigations. Because the focus was on head injury, very little attention was paid to the abdominal infection, and virtually no testing was done. Without contemporaneous CT scans and MRIs, it is very difficult if not impossible to determine the nature or progression of the abdominal injuries or infection. In addition, without testing, it is not possible to exclude underlying hematological, metabolic or vitamin deficiencies or other factors that may have contributed to the child's death. The law enforcement investigation was equally limited. Since the investigators were told that the injuries occurred on the day of hospital admission, the investigators limited their investigation to that day and did not investigate the preceding period even after the Medical Examiner concluded that the injuries likely occurred 2 days prior to hospital admission.

166. What we do have, however, is a well-documented investigation of the events of February 8, including a 17 hour interrogation of Mr. Liebich and

photographs of the Liebich apartment, with numerous descriptions of the hot dog incident and photographs of the cut up hot dog. The medical records also include descriptions and photographs of the child's bruises, which vary considerably over the hospital stay. While the significance of this information was not recognized at the time, these records have provided a record for a more complete medical review. '

Lack of considered differential diagnosis.

167. Good medical practice requires the consideration of all possible entities capable of explaining clinical symptoms.[39] This is known as a "differential diagnosis." A differential diagnosis should be careful, logical and include reasons for including or excluding diagnoses. In any situation involving critical decisions, such as the decision to indict for a felony, this should be formal and in writing. The importance of differential diagnosis is such that medical students spend a considerable part of their education learning how to do this. Interestingly, one decision from the 8th Circuit Court of Appeals required a differential diagnosis in a civil case involving a physician's opinion on causation. One would hope that this standard would apply to criminal cases in which the required standard of proof is much higher. However, I find no differential diagnosis in Steven's records, indicating that none was done.

168. Identifying innocent explanations and natural mimics of abusive injury in children requires a rigorous evaluation of all entities that may explain the child's findings. This type of evaluation is beyond the scope of most clinicians – and most clinical educational programs – since it requires careful analysis of witness statements, scene investigation, competent neuroradiology and neuropathology, biomechanical examinations of any reported short falls (including consideration of force direction, impact surfaces, strength of skull bones), and the like. E.R. pediatricians and neurosugeons are typically dealing with crises and rarely have the time or expertise to undertake this type of investigation. In practice, medical examiners also often lack the resources to address these issues.

169. In this case, the differential diagnosis presents special challenges. *First*, the child had an abdominal infection and/or injuries of unknown origin occurring at least 5-7 days prior to death or 2-4 days prior to hospital admission. These injuries are sufficient to explain the death without head trauma. However, the abdominal CT scan, which would have provided much better information on the abdominal injuries, was cancelled following the misread of the head CT scan. *Second*, by the time the child was transferred to Rush, the child's brain had swollen massively, resulting in a craniectomy (removal of part of the skull). As the Medical Examiner recognized, a craniectomy causes major anatomical distortions that make it impossible to identify or interpret pre-existing head trauma. *Third*, these

evaluations took place in a time period in which many doctors believed that subdural hemorrhage, retinal hemorrhage and brainswelling were primarily or exclusively caused by head trauma, leading to an insufficient evaluation of other causes, including sepsis and hypoxia/ischemia.

170. In this section, I briefly summarize the differential diagnosis for the medical findings in this case.

171. Abdominal injuries/infection. The abdominal infection was most likely caused by accidental or inflicted trauma. However, absent specific pattern bruising, it is not possible to distinguish between accidental, non-accidental or natural causes without a thorough investigation of the appropriate time period, which was at least 5-7 days prior to death. This investigation was not done. However, since Mr. Liebich was not alone with the child prior to February 8, Mr. Liebich can be excluded as a possible perpetrator of trauma.

172. Head injuries. There are no specific indicators of head trauma, such as fractures or soft tissue swelling. Instead, the brain findings are hypoxic/ischemic (*i.e.,* caused by lack of oxygen). These findings are likely secondary to the abdominal injuries but may have been triggered or aggravated by the choking incident described by Mr. Liebich.

173. Bruises. The swollen testes, abdominal bruising and redness on the hip and flank are typical signs of pancreatitis. Other marks are characteristic of hospital interventions, particularly in a septic child. With the possible exception of a larger bruise around the waistline on the back that was no longer visible at autopsy but that may be consistent with a belt buckle, none of the bruises suggest a specific agent causing the bruise (such as a belt, hairbrush, stick or other identifiable object), and they do not suggest a beating since they are small and lack any discernible pattern.

174. Natural causes. While the child's history suggests that the abdominal injury was likely caused by an abusive incident, the abdominal findings are also consistent with abdominal infection from any source, leading to sepsis and multi-organ failure.

Summary

175. In summary, my opinions are as follows:
   a. The abdominal infection and/or injury identified at autopsy were present at least 5-7 days prior to death (2-4 days prior to hospital admission), and likely longer.
   b. It is not possible to determine whether the infection was accidental, abusive or natural in origin.
   c. The brain findings are secondary to the abdominal infection and may

have been triggered or aggravated by the choking incident. There are no specific indicators of head trauma.

d. The bruises and linear marks that appeared at the hospital are related to abdominal infection, sepsis and hospital intervention rather than recent abuse.
e. There is no medical evidence that any injuries occurred on the day of hospital admission.

176. Should this case go to evidentiary hearing and/or retrial, my opinions will require more space and/or detail. I will also consider any additional information that becomes available, including scene photos. In the meantime, should the District Attorney, Court or reviewing doctors desire additional information or references, I am willing to provide this information.

I swear under penalty of perjury that the foregoing is true and correct.

Peter J. Stephens, M.D.

Subscribed and sworn to before me this 25 day of MARCH, 2009.

Notary Public in and for the
State of North Carolina

My commission expires: 8-28-2010

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Medico-legal report
15.03.2012

Steven Quinn

Dob: 4th August 1991
Dod: 28th November 1995

Prepared on behalf of:

Heather Kirkwood

By:

Dr Waney Squier, Consultant and Clinical Lecturer,
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I am Dr Waney Squier, Consultant Neuropathologist to the Oxford Radcliffe Hospitals and Honorary Clinical lecturer in the University of Oxford. I have been a consultant neuropathologist since 1984 having trained at the Institute of Psychiatry and Great Ormond Street Hospital for Sick Children. During my 26 years in Oxford I have specialised in the pathology of the developing brain in the fetus and neonate. My other areas of interest are developmental causes of epilepsy and muscle pathology. I have been involved in research into the nature and timing of brain damage due to intrauterine and perinatal insults, the effects of asphyxia on the immature brain, correlation of imaging and anatomic pathology in the pre-term human brain and the neuropathology of cerebral palsy in children. I have published widely on these subjects in peer reviewed journals and have edited a book "Acquired Damage to the Developing Brain: Timing and Causation". I am a member of the British Neuropathological Society and the British Paediatric Neurology Association. I am a fellow of the Royal College of Physicians,(by election following membership by examination in paediatrics) and a fellow of the Royal College of Pathologists.

In the last ten years my experience with infant brain pathology has extended to many forensic cases and I have written reports and given evidence in court for both the prosecution and the defence in many cases of sudden unexpected death in infants.

My expertise is based on my experience in examination of the brain, spinal cord and dura after death and as such assists in the interpretation of the mechanisms of injury and the imaging of the brain.

I understand that my overriding duty is to the court in preparing reports and in presenting evidence. In preparing this report I have attempted to provide an unbiased analysis of the facts of this case from the perspective of the neuropathology and based on the current state of scientific knowledge, the current peer reviewed literature and my personal professional experience.

I have attempted to be as accurate and as complete as possible and my opinions are restricted only to those subjects which are within my area of expertise. I believe that the facts I have stated in this report are true and that the opinions I have expressed are correct.

Dr Waney Squier MBCHB FRCP FRCPath

Consultant Neuropathologist

Honorary Clinical Lecturer

Review of Brain Histology

This report is based on limited information and material. I have seen 20 sections of brain tissue and dura which have been received from Heather Kirkwood. They are labelled 202-202 and stained with H&E. I have been asked to review these slides. I have seen no other materials. The history below was provided by Heather Kirkwood.

"Steven Quinn was almost 3 years old and had been mildly unwell since the evening of 2/7/02 and possibly earlier. He was given a sliced hot dog at approximately 3 p.m. on 2/8 and choked on it. He was admitted to hospital at approximately 6 p.m. with reports of abnormal breathing. He had pancreatitis on arrival (per labs) and probably DIC. The brain was immensely swollen at neurosurgery at approximately 10 p.m. He was ventilated for approximately 66 hours prior to removal of life support. Autopsy showed ischaemic bowel with small perforation, peritonitis and pancreatitis."

Comment on Neuropathological Review

(Detailed neuropathological findings are appended below.)
The most significant observation is of a swollen and congested brain that appears to be normally formed and is consistent with the age. All the sections examined show the tissues are fragmented and show loss of cellular integrity. The cells stain poorly. There are no reactive cellular changes such as glial swelling, macrophage infiltration or endothelial thickening or reduplication.
These characteristics are seen in babies who have been nursed on a ventilator with reduced blood and oxygen supply.

It should be noted that only one H&E stained section from each brain sample was received and that full Neuropathological assessment is limited without additional special stains.

Timing of the Findings:

The absence of cellular reactions indicates that the pathology is recent and timing is consistent with origin 66 hours before death. It should be noted that timing of brain findings by histology is imprecise and may not be accurate, particularly when a child is ventilated.

Many vessels contain small fibrin thrombi. There is a small thrombus in one dural sinus in section B. This appears old (2-5 days) as red cells have lost their integrity but timing cannot be assessed readily in free clot and certainly not without additional stains. Clot is best assessed when adherent as reactions of the vessel lining aid the timing of clot. When clots form and become adherent there may be a non-adherent tail or fragments may break off. It is therefore possible that more established clot exists in draining veins or sinuses elsewhere. The changes in the brain would be consistent with this. It is not possible to say whether clot preceded or followed collapse but the thrombus identified is all unattached and appears recent and consistent with origin at or shortly after collapse.

Cause of the Findings:

Several sections show patterns of fresh perivascular parenchymal bleeding. This is seen in association with subarachnoid and possibly subpial bleeding and surface vessel congestion. The parenchymal findings are consistent with contusion, but the surface congestion is not. All of these changes are consistent with venous outflow obstruction, including thrombosis.

The thrombus identified is consistent with altered coagulation secondary to hypoxia/ischaemia.

The bulk of the pathology is non-specific and consistent with interruption of blood and oxygen supply and also with "respirator brain". These findings may be seen in cardio-respiratory arrest of any kind, including choking.

There is no evidence of primary traumatic damage but this cannot be determined fully without special stains.

There is no evidence of malformation, infection or old acquired damage to predispose to collapse.

The findings should be interpreted in the light of detailed clinical and autopsy information which is not available to me at the time of writing this report. As noted, these findings are consistent with cardio-respiratory arrest of any kind. Based on the limited available information, the findings are consistent with the history of choking and subsequent resuscitation and ventilation. Any previous abdominal injury or infection is not capable of assessment from the Neuropathology.

Waney Squier
Consultant Neuropathologist
March 14th 2012

Detailed Neuropathological findings

1. Fragmented oedematous brain tissue. Many cells are pyknotic. There is a small amount of patchy perivascular bleeding. Red blood cells within vessels have lost their pigment. Nerve cells are pale. There is no vascular proliferation or other cellular reaction.

2. ?Pons. Neurones are pale. The ependymal lining of the fourth ventricle is normal. No cellular reaction.

3, 4. Fragmented brain - as 1.

5, 6. Cerebellum. Mature consistent with age over 8 months. Fragmented, pale, no cellular reaction.

7. Fragmented brain. Very congested surface vessels and fresh subarachnoid bleeding. Fresh thrombus is seen in a few surface vessels.

8. Fragmented brain. Congested surface vessels with fresh intravascular clot.
Pigmented leptomeninges. Fresh subarachnoid bleeding. No evidence of inflammation, infection or meningitis.

9. - as 8. Very congested surface vessels.

10. Fragmented brain. Focal perivascular bleeding into the parenchyma associated with intravascular fibrin.

11. Brain with focal parenchymal bleeding in the immediate subcortical white matter and on the overlying cortical surface. Here the bleeding is probably subpial (this requires further stains to confirm), also subarachnoid. Blood extends around small superficial vessels into the upper cortical

layers. The overlying vessels are very dilated and congested and are associated with thick subarachnoid bleeding.

12, 13. Fragmented brain.

14. Fragmented brain, congested surface vessels.

15. Fragmented brain, small amount of fresh parenchymal bleeding.

16. Swollen pons Neurones are eosinophilic with nuclei beginning to lyse. No other cellular reaction. The basal veins are extremely dilated and congested with associated subarachnoid and possibly subpial bleeding.

17. Several strips of congested dura. There is fresh blood beneath the ependyma of the sinuses and a small amount of fresh intradural bleeding. The dural sinuses are congested and there is a small amount of fresh clot.

18. Dura with large sinus (?superior sagittal). There is fresh clot in this sinus but in an adjacent smaller sinus there is a small fragment of clot which appears older. Accurate timing is not possible in most cases and any attempt at timing requires the use of special stains. There is no evidence of old subdural bleeding.

26.Recut dura and bone chips.

19. Four sections of spinal cord in dura. The cord is normally formed. It is swollen and congested. There is a small amount of fresh subarachnoid blood. There is no evidence of old subdural blood.

STATE OF ILLINOIS
IN THE CIRCUIT COURT OF THE EIGHTEENTH JUDICIAL CIRCUIT
DUPAGE COUNTY

Randy Liebich, Petitioner	)	Cir. Ct. No. 02-CF-654
v.	)	Post Conviction No.
People of the State of Illinois	)	

AFFIDAVIT OF RONALD H. USCINSKI, M.D.

I, Ronald H. Uscinski, under oath and penalty of perjury, state as follows:

1. My name is Ronald Uscinski. I received my M.D. from Georgetown University, Washington, D.C. in 1968. After internship and service in the United States Navy, I completed a residency in neurological surgery at Georgetown and affiliated hospitals. I have been board certified in neurological surgery since 1978.

2. During my career, I have taught neurosurgery at George Washington and Georgetown University Schools of Medicine. I am currently a Clinical Associate Professor in the Department of Neurological Surgery, George Washington University School of Medicine; a Clinical Associate Professor, Department of Neurosurgery, Georgetown University School of Medicine; and a Senior Adjunct Fellow at the Potomac Institute for Policy Studies, Arlington, Virginia. I maintain an active neurosurgical practice covering all age groups. Since the late 1990s, I have developed special expertise on shaken baby syndrome/abusive head trauma, and I have published, lectured and served as an expert witness on this subject on multiple occasions. My curriculum vita is attached as Exhibit 1.

3. I was asked to conduct a blind review (i.e., a review without access to any significant history) of a 2/8/02 CT scan for Steven Quinn (DOB 4/17/99) and to address the following questions:
   a. What does the CT scan show?
   b. Is surgery appropriate and, if so, what kind?
   c. What are the most likely causes for the CT findings?

4. CT findings. The CT shows findings indicating an anoxic insult to the brain (*i.e.*, a brain that has been deprived of oxygen, with a breakdown of grey white differentiation), more on the left. There is some subdural hemorrhage along the cerebellum and the occipital poles, very thin on the right side. There is subarachnoid hemorrhage and possible blood in the ventricles. These findings indicate that there has been an anoxic insult to the brain that is likely irreversible and nonrecoverable. There are no indicators of trauma (fractures, tissue swelling, etc.).

5. Surgery. Based on the CT scan alone, I would not expect this child to survive. The critical factor is the anoxic brain, not the thin hemorrhages, which are likely a side effect of anoxia. There is insufficient hemorrhage to drain surgically. If one wanted to try to attempt improving the situation, which is a natural but I would think fruitless endeavor, one might insert an intracranial pressure monitor, use diuretics, hyperventilate, etc. A craniotomy or craniectomy on the left might relieve the pressure but would be unlikely to stop impending brain death. Realistically, I would not expect any neurosurgical procedure to affect the outcome.

6. Causation. The CT findings are consistent with any process that deprives the brain of oxygen. Since the narrow airway in young children can be fully or partially obstructed by food or foreign body, one obvious possibility is upper airway obstruction. Other causes include heart dysfunction, respiratory distress (from any cause), and shock.

7. History. After providing this preliminary review, I was told that: (1) the history is of a child who refused food the night before hospital admission and choked when eating/drinking three hours before admission; (2) lab tests taken shortly after hospital admission confirmed pancreatitis; (3) the autopsy confirmed abdominal injuries and/or infection, including an ischemic bowel, peritonitis, pancreatitis and a liver hematoma; and (4) the pathology report indicated that the abdominal injuries and/or infection were approximately five days old (two days before hospital admission). The CT scan is consistent with the history of pre-existing abdominal injuries/infection, possibly aggravated by choking with critical oxygen deprivation to the brain.

I am giving this affidavit of my own free will. No promises or threats were made to me in exchange for making the statements contained herein. If called to testify, I would testify consistent with this affidavit.

Ronald H. Uscinski, M.D.

Date: 4 April 2012

HAZEL A. WATERS NOTARY PUBLIC MY COMMISSION EXPIRES 1/14/2017 DISTRICT OF COLUMBIA

District of Columbia : SS
Subscribed and Sworn to before me
this 4 day of April, 2012

Notary Public, D.C.
My commission expires 01-14-2017

Hazel A. Waters
Notary Public, District of Columbia
My Commission Expires 01-14-2017

RONALD H. USCINSKI, M.D.
NEUROLOGICAL SURGERY

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Chevy Chase, Maryland 20815
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CURRICULUM VITAE

Ronald H. Uscinski. M.D., FACS

Occupation: Physician

Education:

B.S., Fordham University, New York, NY, 1964

M.D., Georgetown University, Washington, D.C., 1968

Internship, Bronx Municipal Hospital Center, Albert Einstein University College of Medicine, New York, NY, 1968-9

Residency in Neurological Surgery, Georgetown University and affiliated Hospitals, 1971-1975

Military Experience:

Medical Officer, United States Navy; served with United States Marine Corps, Parris Island, South Carolina, and aboard The U. S. S. Thomas A Edison (SSBN 610-B) Atlantic Submarine Force, 1969-1971

Appointments & Positions:

Senior Surgeon, U.S. Public Health Service, Medical Officer, Surgical Neurology Branch, National Institute of Neurological and Communicative Disorders and Stroke, NIH, Bethesda, Maryland, 1975-1976

Instructor in Surgery (neurosurgery) Georgetown University School of Medicine, Washington D.C., 1975-1976

Consultant in Neurosurgery, NIH, Bethesda, Maryland, 1976-1977

Clinical Instructor in Neurosurgery, Medical University of South Carolina, Charleston, South Carolina, 1977-1980

Clinical Assistant Professor, Dept. of Surgery (Neurosurgery), Georgetown University School of Medicine, Washington D.C., 1980-2000

Clinical Associate Professor, 2000-present

Clinical Assistant Professor, Department of Pediatrics, Georgetown University School of Medicine, Washington D.C., 1980-present

Clinical Assistant Professor, Department of Neurological Surgery, the George Washington University School of Medicine, 1997-2008

Clinical Associate Professor, 2008-present

Adjunct Research Fellow: Potomac Institute for Policy Studies, Arlington, Va, 2004-2006.
Senior Adjunct Fellow, 2006-present

Certification

American Board of Neurological Surgery, 1978

Societies:

American College of Surgeons, 1980
District of Columbia Medical Society, 1981
Polish Society of Neurological Surgeons, corresponding member, 1983
Research Society of Neurological Surgeons, 1989

Publications:

1. Ventricular Septa in the Neonatal Age Group, Diagnostic Considerations of Etiology and Comparison of Sonography and Computed Tomography. Schellinger D, Grant E, Hanz H, Petranoa H, Uscinski R. AJHR: volume 7:1065-1071, 1987

2. Periventricular Leukomalacia in Combination with Intraventricular Hemorrhage, Sonographic Features and Sequelae. Grant E, Schellinger D, Smith Y, Uscinski R., AJHR: volume 7; 443-447, 1986

3. The Shaken Baby Syndrome. Uscinski R., Journal of American Physicians & Surgeons: Volume 9, #3; 76-77, 2004

4. The Shaken Baby Syndrome: An Odyssey. Uscinski R. H., Neurologia medico-chirurgica (Tokyo) 46, 57-61, 2006

5. The Washington Post, March 9, 2008: B08, Outlook; "The Larger Tragedy in an Unjust Accusation"

6. The Shaken Baby Syndrome: An Odyssey II. Origins and Hypotheses. Uscinski R. H., McBride D. K., Neurologia medico-chirurgica (Tokyo) 48 (3), 151-155, 2008

7. "I Stand with Humility" Uscinski R. H., Neurologia medico-chirurgica (Tokyo) 48 (9), 423-424, 2008

Presentations:

1. Research Society of Neurological Surgeons, "The Repaired Myelomeningocoele, and Its Relationship to Tethering of the Spinal Cord" June, 1989

2. National Child Abuse Defense Resource Center, Child Abuse, 2000 and Beyond "Rebleeding and Subdurals and Children," September, 2000

3. National Association of Counsel for Children, 23rd National Children's Law Conference-Improving the Professional Response of Children in the Legal System, Panel Discussant: "Shaken Baby Syndrome" November, 2000

4. Interdisciplinary Problem Solving in Cranial-Maxillofacial Surgery, Panel Participant, Washington D.C., February 2001

5. National Child Abuse Defense Resource Center, "The Shaken Baby Syndrome, an Odyssey" September, 2001

6. The Polish-American Health Association, Washington D.C., 2001"The Shaken Baby Syndrome, a Clinical Neurosurgical Perspective" March, 2001

7. Congress of the Polish Society of Neurosurgeons, Rzseszow, Republic of Poland "The Shaken Baby Syndrome, an Odyssey" September, 2001

8. Kings College Hospital, London, UK, "The Shaken Baby Syndrome, an Odyssey," February, 2002

9. Addenbrooke Hospital, Cambridge University, UK; "The Shaken Baby and Newtonian Physics," February, 2002

10. The Radcliffe Infirmary, Oxford University, UK; "The Shaken Baby Syndrome," February, 2002

11. The Neurosurgical Society of the Virginias, 37th Annual Meeting Hot Springs, Virginia, January, 2003; "The Shaken Baby Syndrome, History, Mechanism, and Paradox"

12. American Association of Physicians and Surgeons, Annual Meeting Portland, Oregon, October 2004; "The Shaken Baby Syndrome"

13. Japanese Society for Pediatric Neurosurgery Annual Meeting, Invited Guest Speaker, Nara, Japan, May 2005; "The Shaken Baby Syndrome," "Pediatric Neurotrauma" Ideas from the Arena"

14. National Child Abuse Defense and Resource Council, Annual Meeting, Las Vegas, Nevada, September 2006; "A Primer on Medical Recording"

15. United States Air Force Judge Advocate General School, Maxwell AFB, Montgomery, Alabama, Guest Lecturer, May, 2007: "The Shaken Baby Syndrome"

16. Administrative Office of the Courts, State of Kentucky, September, 2007: "The Shaken Baby Syndrome"

17. King Faisal Hospital, Kigali, Rwanda, Special Lecture, January, 2008: "Neurosurgery, Medicine, and Scientific Methodology"

18. The Neurosurgical society of the Virginias, 43rd Annual Meeting, Hot Springs, West Virginia, January, 2009, "Observations on Primate Birth"

19. Florida Public Defenders Association, 2009 meeting, Naples, Fla, "The Shaken Baby Syndrome: The Odyssey Through Time, Space, and Ontogeny"

20. Howard University, Department of Communication Science and Disorders, Annual Lecture, April 2010 "EBMS and the Quest for Scientific Integrity in Medicine"

21. 31st Annual McKendry Boyer Lecture, Montgomery General Hospital, June 2010, "The Shaken Baby Syndrome, From the Mists of Prehistory to Modern Times; an Odyssey Through a Small Passage"

22. New York City Abusive Head Trauma Shaken Baby Syndrome Conference, Sept. 23, 2011; "Anatomy of an AHT Diagnosis, Investigation, and Prosecution," panel discusser

STATE OF ILLINOIS
IN THE CIRCUIT COURT OF THE EIGHTEENTH JUDICIAL CIRCUIT
DUPAGE COUNTY

Randy Liebich, Petitioner	)	Cir. Ct. No. 02-CF-654
v.	)	Post Conviction No.
People of the State of Illinois	)	

AFFIDAVIT OF NATHAN FELIX

1. My name is Nathan Felix. My address is 10006 SW 44th Lane, Gainesville, FL 32608.

2. I have served with distinction as a Non-Commissioned Officer in the United States military (US Army and Air Force), where I received an extensive education in the medical field. During the first year of the NATO peace keeping mission, I volunteered for service in Bosnia-Herzegovina, where I served as a medic and linguist. I also served three tours of duty during Operations Enduring Freedom and Iraqi Freedom as a flight medical technician. I continue to serve my country in the Air Force Reserve as a member of the 927th Aeromedical Staging Squadron (ASTS) where I continue to serve as a medical technician. I currently hold the rank of Technical Sergeant.

3. As a line medic in the Army, I performed essentially the same functions as a physician's assistant, *i.e.,* I was responsible for diagnosis, treatment and minor surgical procedures, as authorized by the supervising physician. As an Aeromedical Evacuation Technician in the Air Force, I worked closely with doctors and nurses in cargo aircraft that had been converted into intensive care units for the transport of patients to appropriate facilities. Our job was to do whatever was necessary to maintain patient stability in flight. I also managed the unit's CPR and ACLS programs. In civilian life, I worked as a medical assistant at Country Doctor Community Clinic in Seattle, WA, a health clinic that provides comprehensive medical care. My responsibilities included most aspects of routine patient care and education. I currently work as a transfer center coordinator at Shands Hospital attached to the University of Florida in Gainesville, FL, where I facilitate incoming patient transfers from other facilities.

4. A few years ago, while visiting her home on another matter, Heather Kirkwood asked me to look at lab results and hospital photographs for a child, Steven Quinn.

5. The lab tests showed extremely high glucose (over 500), indicating that the child was likely in hyperglycemic shock. He was also throwing off excess glucose in the urine. The

amylase and lipase were extraordinarily high (amylase 3025 v. ref. range 20-120; lipase 2368 vs. ref. range 22-51), indicating that he had pancreatitis or a severe endocrine problem. These findings would require emergency treatment, usually with antibiotics, insulin and an abdominal CT scan. I would not expect the child to survive without prompt treatment.

6. The photographs showed oddly patterned circular and linear bruises or marks. Bruises can come from external impact or systemic abnormalities. In this case, the circular bruises looked too small for punches, and the lines were too thin for a belt. The lines could be from IV tubes or a hanger. However, the delayed timing – the linear marks seem to appear during the hospital stay – struck me as inconsistent with an earlier beating or beatings with a hanger. In addition, some of the lines seemed to be in different places in different pictures.

7. Since there was some suggestion that the child had been hit repeatedly with a hanger, I suggested an experiment. Since my skin color is about the same as Steven's, I suggested that Ms. Kirkwood hit me with a hanger as hard as she could. She was initially reluctant but did so. She broke the hanger on the first hit. I then took the straight part of the hanger and hit my arm and leg as hard as I could several times. Lines from each of the hanger hits appeared within seconds. Within minutes, there were raised welt-like red lines. Within 30-45 minutes, all marks had disappeared.

8. The later lab results indicated that all of the child's organs, including the kidney, liver and pancreas, appeared to be failing. This was not surprising since all organs, including the brain, will have problems with these sugar levels. I was surprised that there appeared to be no effort to address the abdominal and glucose issues in the first hours of hospitalization.

9. The records indicated that a Penrose drain was inserted to drain the abdomen the following morning. Ms. Kirkwood told me that a small bowel perforation was found at autopsy. This can occur with the insertion of a Penrose drain, particularly in the presence of abdominal infection.

I swear under penalty of perjury that the foregoing is true and correct.

Nathan Felix

Date: 04/02/2012

Sworn before me 2nd of April, 2012

Notary:

JENNIFER TALLENT
MY COMMISSION # DD 798486
EXPIRES: October 17, 2012
Bonded Thru Notary Public Underwriters

STATE OF ILLINOIS)
)
COUNTY OF DUPAGE)

AFFIDAVIT OF RANDY LIEBICH

I, Randy Liebich, being first duly sworn on oath, depose and say as follows:

1. My name is Randy Liebich. I was convicted of first degree murder in the death of my fiancee's son, Steven, based on medical testimony that Steven died from multiple blunt force injuries to his head and abdomen. Some of the State's witnesses said these injuries occurred when I was looking after Steven. Dr. Shaku Teas, who was Chair of the Aurora Child Fatality Review Team, testified that, according to the slides, the injuries were much older and could not have occurred during the time that I cared for Steven. Dr. Darinka Mileusnic, who performed the autopsy and testified for the State, mostly agreed with Dr. Teas.

2. I did not hurt Steven and repeatedly told the hospital and the police that the only unusual event that day was that Steven choked after eating part of a hot dog and bit my finger when I tried to see if his airway was clear. A few hours after that, Steven was breathing oddly, and Kenyatta (my fiancée) and I took him to the hospital.

3. Since I did not testify at trial, there were a lot of errors in the testimony that were not corrected, and a lot of evidence that didn't get into the trial, including medical evidence that Steven was sick before February 8. I wanted to testify but my attorney refused to put me on the stand. My attorney seemed to think he could get in some of this evidence in through other witnesses, but the judge said he did not ask the right questions or ask them of the right people, so the information didn't get in.

4. **Relationship with Kenyatta.** Kenyatta and I were going out for about two years before Steven died. Kenyatta told me she ran away from home when she was 15 or 16 because her stepfather beat her and her younger siblings, and that her mother wouldn't do anything about it. Steven was born when Kenyatta was 16. He mostly lived with Kenyatta's mother and aunts but Kenyatta picked him up for visits.

5. I did not like how Kenyatta treated Steven on his visits. She often hit him, sometimes cuffing him on the head, hitting him with broken plastic hangers, pushing or throwing him, or poking him in the head or stomach with her fingers. Kenyatta was very upset about how her stepfather treated her siblings but seemed to think that how she treated Steven was normal. She would not let me interfere.

6. When Steven stayed with us for a month around July 2001, I asked Kenyatta to take him back to his grandmother and aunts because I felt that he was better off with them.

7. When Kenyatta and I were staying with my mother, Kenyatta pushed my mother on her shoulder, causing her to fall backwards and hit her head on the corner of the door. My sister Denise and I were in the room. My mother was unconscious or groggy for a few minutes. She didn't want us to call 911, but she told me that I had to get Kenyatta out of the house, and we moved out.

8. I never hit Steven, not even for discipline. He would usually do what you wanted if you asked him. Steven and I got along well, and I looked after him if Kenyatta was getting her nails done or working. I often looked after my younger cousins, so I had a lot of experience with kids. Steven was easy to look after.

9. **February visit.** Our daughter Angelique was born on January 27, 2002. Kenyatta and I had moved into a new apartment, and I had just started work at the Patio restaurant. Kenyatta was working part-time at Carlene Research for my aunt. On February 2, we picked up Steven from Kenyatta's aunt.

10. That week, Steven was quieter than usual. I thought he was a little jealous or depressed because the baby was getting a lot of attention. He also didn't seem to feel well. He whined and cried more than usual that week, often for no reason. He was always a little slow moving, but this week he was slower than usual. The last couple days, he wouldn't eat unless Kenyatta almost made him eat.

11. During Steven's visit, I was working at the Patio on rotating shifts. I worked on the 4th and the 5th. On the 6th, Steven didn't want to eat, and Kenyatta almost forced him to eat, which was unusual. Kenyatta and I took Steven and Angelique to visit my cousin Frank that day. When we were leaving Frank's, Kenyatta shook or hit Steven because he was crying or whining.

12. When I went to work on the 7th, I found that I was supposed to have worked on the 6th (not the 7th), so I came home. Steven was crying in the bedroom but came running out when he heard me come in. I asked why he was in the bedroom, and Kenyatta told me she made Steven go in the bedroom because he wouldn't eat his dinner, which was pork chops and mashed potatoes. Kenyatta told Steven to go back in the bedroom, which he did.

13. Kenyatta and I went in the bedroom a little later and shared a joint of marijuana by the window. When Steven wouldn't stop crying, Kenyatta took my belt out of my pants, which were lying on the floor, and hit Steven with the belt over his diaper. I turned away because I didn't like to see this. When he didn't stop crying, Kenyatta pulled off his diaper and swatted him on the rear end several times with her hand, telling him to stop crying. She also slapped him on the side of his head. Again, I didn't like this.

14. Steven didn't come out of the bedroom the rest of the night. Kenyatta had put plastic wrap over his food and put it in the refrigerator. The plate would still have been in the refrigerator when the police searched the apartment.

15. When Kenyatta left for work around 10 a.m. the next morning, Steven was still sleeping. Kenyatta put some cereal in a bowl and told me to add milk and give it to him when he got up. I did that. Steven didn't want to eat but I kept encouraging him to eat, just as Kenyatta had been doing for days. He ate the cereal but left the milk. I don't remember how much cereal was in the bowl.

16. After that, Steven played with the dog and we watched TV. We all fell asleep at some point. When both kids were sleeping, I ran across the road to McDonald's and borrowed a cigarette from a girl who worked there. I was gone less than 5 minutes, and the kids were still sleeping when I got back.

17. Around 3 p.m., I fixed Steven a hot dog. I cut it up for him and put it on a plate with ketchup. I had to coax him to eat. He drank some orange juice and ate a little of the hot dog, but then wanted more to drink. I gave him water but he started choking. When I put my finger in his mouth to see if he had some hot dog caught in his throat, he bit down on my finger. I told him to let go. When he didn't let go, I slapped him on the cheek to get him to let go. I did not hit him hard, just light slaps. I also patted him on the back to dislodge any food that might be stuck.

18. When Steven let go of my finger, there was a little bite mark on my finger and some vomit in Steven's mouth. I cleaned him up and he seemed a bit dazed but more or less okay, so we watched a bit of Jurassic Park and he went to sleep.

19. When Kenyatta came home around 4, I had a shower and got dressed for work. When I came out, Kenyatta said that Steven was breathing funny and that she wanted to use my car to take him to a doctor. At first I didn't think she really wanted to go to the doctor since she sometimes went out partying. However, when I looked at Steven, he was breathing oddly, and I agreed we should take him to the doctor. Since we didn't think it was an emergency, I took the dog out first.

20. We left for the hospital about an hour and half after Kenyatta got home. We stopped at my work on the way, and I took Steven in so my manager could see he was sick. Since I had mixed up my workdays, I was afraid I would lose my job if I didn't show up or they didn't believe that Steven was sick. The manager told me to go ahead and take care of him.

21. <u>Mt. Sinai</u>. Kenyatta drove to Mt. Sinai Hospital since that's where Steven went for his clinic appointments. By the time we got to the hospital, Steven's breathing was really odd. The first doctor, Dr. Green, seemed to think he was sick and said she would run some tests. She asked me what had happened during

the day, and I told her about Steven choking on the hot dog. We were all at his bedside when she examined him, and no one saw any bruises.

22. After Steven had a CT scan, another doctor, Dr. Boykin, came in and said that Steven's head was filled with blood, and she accused me of beating on him all day. I hadn't touched him other than to try to make sure his airway was clear and to get my finger out of his mouth. Kenyatta hit him the night before, as I described. I didn't know if anything happened when I wasn't home on that day or the preceding days.

23. **Rush Hospital.** At Rush, I wasn't allowed to see Steven. None of the doctors at Rush asked me what had happened.

24. **Interrogations.** The police began to interrogate me at the hospital and they continued for more than 17 hours. I was not allowed to leave during this period. I think they interrogated Kenyatta on and off,but she was free to see Steven and move around the hospital. I answered their questions and told them about the events of the day over and over, including choking on the hot dog. They kept telling me that Steven had been beaten and that I had to tell them what happened. I finally told them I wasn't going to answer any more questions without a lawyer. They ignored me and kept questioning me. The police reports on these interrogations contain many errors but also make clear that I never described anything unusual other than choking on the hot dog. I am providing a supplemental affidavit on the interrogations.

25. On February 14, a television station ran a story saying in effect that I had murdered Steven and that I was on the run. I was not on the run; I was staying with my mother and stepfather, which the police knew. My cousin Dion heard the story and came to my mother's house. He was angry and upset, and we went to talk to our cousin, Robert, who is a police officer in Rosselle, Illinois. I barely knew Robert but agreed to talk to him to clear up that I was not on the run and to see if he could help figure things out.

26. Robert came to the Rosselle police station and put Dion and me in an interrogation room. He questioned me for about an hour, and I told him everything I told the police earlier. Robert was angry that I couldn't explain why Steven died, and it quickly turned into an interrogation. Robert asked more detailed questions than the police asked earlier about the hot dog incident, and he had me demonstrate what I did when Steven bit down on my finger. I showed him what I had done, which he didn't seem to think would have caused any harm. These were light slaps on the cheek, not hard, just to get Steven's attention and try to get him to open his mouth. I also patted him on the back to see if that would dislodge anything that was stuck. I showed Robert my finger, which had a small cut at the bottom of the fingernail where Steven clamped down.

27. I didn't know much about DNA but I heard about it on TV shows, so I asked Robert if my DNA would show up on Steven's teeth (not stomach). Robert also asked me if I had told the police about slapping Steven to get him to let go of my finger. I didn't think I had because they weren't interested in the incident with the hot dog. Shortly after that, I was arrested.

28. **Trial preparation.** After I was arrested, I was assigned a public defender, Ricky Holman. At that time, we knew that the pathologist and some of the hospital doctors were saying that Steven died from a beating, but no one other than Dr. Boykin said when the beating was supposed to have occurred. I think that Mr. Holman told the judge pretty early that no one was arguing about the cause of death, only about the timing of the beating.

29. I didn't now what caused Steven's death other than, if it was a beating, it was not by me.

30. Later, Mr. Holman hired a forensic pathologist, Dr. Shaku Teas, to look at the medical information and determine when the injuries occurred. It took a long time for the State to give Dr. Teas the information she needed. When Dr. Teas got the slides, she said that based on the amount of healing the injuries occurred at least five days before the end of life support. This meant that the injuries occurred before noon on February 6th or earlier. Since I wasn't alone with Steven and Angelique before the 8th, this meant that I could not have caused the injuries.

31. I understood that the pathologist who did the autopsy agreed with Dr. Teas and that the 5 day figure was in her reports. After that, my attorneys were very confident since it seemed clear that I couldn't have caused the injuries.

32. Based on this, I assumed that Kenyatta must have injured Steven when I was working earlier in the week. He could also have been injured before he came to our house. There were lots of kids at Kenyatta's aunts, who did daycare and foster care, and I wondered if one of them had fallen on Steven or something like that. I also wondered if Kenyatta had pushed Steven and caused him to hit his head or stomach, much like what happened with my mother, and did more damage than she intended.

33. Since my attorneys said the only issue was whether I had beaten Steven or whether Kenyatta had beaten Steven, I gave my attorneys a lot of information about Kenyatta before the trial. I told them that Kenyatta had hit my mother and knocked her out, and that many people had seen Kenyatta hit or throw Steven. In the beginning, Mr. Holman had an investigator interview a couple possible witnesses. After that, he didn't follow up.

34. **Trial.** At trial, my attorneys assumed that the State didn't have a case since the pathology showed that Steven's injuries were at least five days old when life support was removed and that I therefore could not have inflicted them. When

the State's doctors testified that the slides did not matter and that the injuries occurred within hours of arriving at the hospital, my attorneys were not prepared to cross-examine them. I don't think they had spoken to any of these witnesses before trial.

35. My attorneys also didn't meet with the witnesses who had seen Kenyatta hit or throw Steven before trial or go over their original statements with them. These statements were videotaped and taken by an investigator. Small parts of the statements were played at trial to impeach them. There were little inconsistencies in their statements, and the Court decided not to believe any of them.

36. I also told my attorneys that Kenyatta had told me for years that her stepfather abused her and her younger siblings, and that her mother refused to protect them. This was also in her diary, and there were department of family services reports. My attorneys didn't use any of this at trial, and the judge decided that Kenyatta's mother would have reported any abuse by Kenyatta.

37. I also told my attorneys that Steven wasn't feeling well the week he was with us and that he hadn't been eating much for a couple days before the 8th. I later learned that Steven had Tylenol or aspirin in his system, which showed up in the lab tests. I hadn't given him anything, and I didn't know that Kenyatta had given him anything. Since we didn't have any children's medicines in the house, this meant that Kenyatta must have been giving Steven some of her own Tylenol or aspirin. My attorneys didn't bring this up at trial.

38. The medical records also showed that Steven had lost 5 pounds between his last doctor visit in November 2001 and when he collapsed on February 8. My attorneys didn't introduce this evidence, either. As a result, the Court said that Steven was in good health on the morning of the 8th.

39. Lots of the information that the Court was given was wrong. The State said that I had never been alone with Steven before the 8th. However, Kenyatta and I had been together for two years, and I had often been alone with Steven since I looked after him when Kenyatta got her nails done or worked. Even Kenyatta's aunt told the police that I picked up Steven on my own. My attorneys never introduced this evidence and left with the judge with the impression that this was the first time I had been alone with Steven.

40. At trial, my attorneys were not prepared to cross-examine the State's witnesses. I think that because they thought that the evidence on timing was conclusive, they did not prepare for this. Mr. Holman often seemed lost, and the Court often told him that he was asking questions wrong or not using the right witness. After a bit of this, it seemed that he gave up.

41. I wanted to testify on my own behalf since much of the information that the Court was given was incorrect or incomplete. For example, Robert Liebich testified that

I said that I hit Steven when trying to get my finger out of his mouth and that I said I didn't hit him that hard, but he didn't say that I showed him what I did, and that he said this couldn't have hurt him. My attorneys never explored this with him. My cousin Dion, who was also present, would also have confirmed this. The Judge said that Robert's testimony that I had hit Steven to get my finger out of his mouth was a key piece of evidence against me.

42. Kenyatta also gave a lot of testimony that wasn't right. For example, she said that she and I stopped to smoke PCP in a park when driving Steven and Angelique home from her aunt's house. However, Kenyatta used PCP, not me. I have smoked pot since I was a teenager. I used heroin in my late teens, but stopped by going to a methadone clinic. I used heroin again as a pain reliever after I was in a car accident and hurt my back but went back on methadone before Angelique was born. I was not secretive about this, and I told this to the police when they interrogated me. The judge used my drug use, including use of PCP, to increase my sentence.

43. At my trial, the police misstated what I had said or attributed things that Kenyatta said to me. For example, they seemed to be saying that I said that Steven couldn't feel it through his diaper when Kenyatta hit him with a belt. I never said that; that was what Kenyatta said. None of this was ever corrected.

44. I asked Mr. Holman to have my statements to the police suppressed since most of them were obtained after I asked to talk to a lawyer and the police refused to let me do so but instead kept questioning me. Mr. Holman wouldn't do this and said it wasn't in my interest to do this. Since Mr. Holman wouldn't put me on the stand, this meant that I couldn't correct anything they said in their reports.

45. Mr. Holman told me that he wasn't putting me on the stand since he said that the State could introduce my criminal record, which included a few misdemeanors but no felony convictions. However, the judge already knew this since it was discussed at the bond hearing. Mr. Holman also said that if I testified State could impeach me with the police statements, but these were the statements I wanted to clear up.

46. If I had known that I had the constitutional right to testify on my own behalf, I would have testified. As it was, I was being convicted on testimony that was incorrect, and I wasn't given the opportunity to correct it.

47. **Post-trial proceedings.** Mr. Holman and Mr. Casey were very upset by the Court's decision. They made clear that they believed I was innocent and that this was proven by the pathology. Dr. Teas also wrote a letter to the Court. I was disappointed when the judge refused to read Dr. Teas' letter and surprised when Mr. Holman told her that he agreed that she shouldn't read it. Mr. Casey was very supportive since he believed that the medical evidence proved that I couldn't have caused the injuries.

48. I filed my own post-trial motion on ineffective assistance of counsel. At first the judge denied the motions without hearing the evidence. Later, she had a hearing. One of the issues was my right to testify on my own behalf. Mr. Holman told the court that he told me of this right, and the court said she usually advised defendants of it, too. Mr. Holman may have believed that he told me this but he did not do so, and the transcript shows that the Court didn't either. I would have remembered if anyone told me of this right since I wanted to testify.

49. **Appeal.** After the trial, my mother cleaned houses to pay for a private appellate lawyer. The lawyer first said she would charge $10,000 for the appeal. Later, she said she wouldn't complete the appeal unless she was given more money. I believe that my mother raised another $5,000 or so. My mother was not well, and I believe this contributed to her early death.

50. Even so, the appellate lawyer refused to argue that the pathological evidence proved that I was innocent. One of the appellate judges understood this from Dr. Teas' letter. The other two judges said, essentially, that the judge was free to ignore the pathological evidence in favor of doctors who were not trained in pathology.

I swear under penalty of perjury that the foregoing is true and correct to the best of my knowledge and belief.

Randy Liebich

Subscribed to and sworn before me this 23rd day of February 2008

Notary Public in and for the State of Illinois

My commission expires 5.20.2012

OFFICIAL SEAL
SHERWIN K. MILES
NOTARY PUBLIC, STATE OF ILLINOIS
MY COMMISSION EXPIRES 5-20-2012

STATE OF ILLINOIS	)	**SUPPLEMENTAL AFFIDAVIT**
	)	**OF RANDY LIEBICH**
COUNTY OF DUPAGE	)	**RE: INTERROGATIONS**

I, Randy Liebich, being first duly sworn on oath, depose and say as follows:

1. My name is Randy Liebich. This affidavit sets forth the circumstances of the police interrogations that were used as evidence against me at my trial.

2. **February 8-9 interrogations (17 ½ hours).** On February 8, 2002 at about 11:20 p.m., I was in the waiting room at Rush Hospital with my fiancée, Kenyatta Brown, our daughter Angelique, and several of her immediate family members when DuPage County Sheriff Detective Greg Figiel, Investigator Boris Vrbos from the DuPage County Children's Center, and nurse Tammy Smith told me to follow them to a conference room to be interviewed. When we entered the room, the detectives told nurse Smith to take my daughter, Angelique, from my arms.

3. During this interrogation, I was in a small room with only one door and a long rectangular table. I initially sat down in the chair closest to the door, but Investigator Vrbos told me to move to the end of the table in the back of the room. Det. Figiel and Inspector Vbros sat on each side of me, which prevented me leaving. Several times during the interrogation, I attempted to stand to leave, telling them that I needed to get back to the family, but each time I tried to stand up, Investigator Vrbos physically pushed me back into the chair and told me that I had no family here and that he would tell me when I could leave.

4. After about an hour of interrogation, Det. Figiel and Investigator Vrbos told me that I was not to leave the conference room, and they placed another police officer outside the door to ensure that I could not leave. They then left the room.

5. About 45 minutes later, at about 1:05 a.m. on February 9, Det. Figiel and Investigator Vrbos returned with a piece of paper that listed my rights. Det. Figiel told me to sign the paper, and I said that I didn't believe I should sign anything. Det. Figiel told me that signing the paper just meant that I had read it. Investigator Vrbos interjected and in an angry voice said, "Sign the damn paper and tell us what happened to Steven Quinn or I'll see to it that Child Services takes your daughter and you'll never see her again." I was concerned for Angelique and felt compelled to do whatever they told me to do, and I signed the form.

6. Det. Figiel and Investigator Vrbos continued to question me. Several times, I said I no longer wanted to answer questions and I tried to leave to see how Steven was doing. They stopped me, and Det. Figiel said, "No, you're not going anywhere near Steven Quinn." Det. Figiel and Investigator Vrbos finally left the room.

7. As they left, Lt. Szalinski and Sgt. Kunz opened the door to the conference room and took me to another room in the hospital. That room had a telephone, and I asked to use the phone to call my family to tell them what was happening and to ask them to contact an attorney for me. I made this request several times. Each time the response was "no."

8. At approximately 2:15 a.m., Lt. Szalinski and Sgt. Kunz took me downstairs to the emergency room where the hospital staff was watching Angelique. The detectives told a nurse to bring Angelique to me and I was allowed to hold her for approximately three minutes before Lt. Szalinski told me to take a good look at her because it would be the last time I would see her or hold her unless I cooperated and gave a better explanation of what happened to Steven. Lt. Szalinski then directed the nurse to take Angelique from me. Sgt. Kunz then said, "Why don't we step outside and smoke a cigarette while you think about that."

9. Sgt. Kunz and hospital security escorted me outside to where Kenyatta was smoking a cigarette. Kenyatta gave me a cigarette and I tried to speak to her. However, Sgt. Kunz intervened and instructed Kenyatta to go back inside the hospital. Sgt. Kunz then asked me more questions about Steven.

10. After finishing the cigarette, Sgt. Kunz and hospital security escorted me back to the fifth floor, put me in an isolated room and prevented me from leaving by placing an officer outside the door.

11. At about 3:15 a.m., I was taken from the isolated room and escorted to a room with Det. Figiel, Investigator Vrbos and Steven. Investigator Vrbos placed his hand on the back of my neck, squeezed hard, and asked me repeatedly, in an accusing manner, "What did you do?" "Why did you do this?" and "You had better give us some answers." I told Investigator Vrbos and Det. Figiel that I was tired of being intimidated, and I demanded a phone call to contact an attorney.

12. I was then escorted to the waiting area and the officers were directed not to allow me to call or talk to anyone. At about 4:10 a.m., after I signed waivers for Steven's medical records, I was told that Kenyatta and I were to accompany detectives to the sheriff's office for further investigation. Officers remained with me until approximately 7 a.m., when I was placed in handcuffs and taken by Det. Figiel and Investigator Vrbos to the DuPage County Sheriff's office. On the way, they stopped to get something to eat.

13. When we arrived at the Sheriff's office at about 8 a.m., I was taken to an interrogation room where I remained alone until approximately 8:40 a.m., when I began throwing up in a wastebasket. Officers entered the room, and I explained that I had been deprived of sleep all night and was extremely sick and was taking methadone. I told them that I had not had the medicine all night, and needed to get it. I was told that I could not leave.

14. For about three hours, different detectives entered and exited the interrogation room asking questions. I told them that I didn't do anything, that I needed to leave to get my medication, and that I didn't want to answer any more questions. I was told that I could not leave.

15. At approximately 12:15 p.m., I was questioned by Officer Richard O'Brien, who identified himself as a polygraph examiner. I was told that I would be taking a polygraph that day. I told Officer O'Brien and the detective who was with him that I was extremely sick, that I didn't want to answer any more questions, and that I did not want to take any test under these circumstances. At that point, the officers asked me to sign a Miranda form. I told them that I would not answer any more questions and I refused to respond to anything further. Officer. O'Brien and the detective finally left the room.

16. About a minute later, Lt. Szalinski entered the room and began asking questions about what happened to Steven. I told him that I didn't have anything else to say, and that I had already asked for a lawyer. He was persistent and told me that I wasn't going anywhere and that he knew I was suffering from heroin withdrawal and was in a lot of pain. He said that the longer it took for me to tell them what they wanted to know, the longer I would sit there and suffer. I felt compelled to continue to answer questions. I asked several times for an attorney, and Lt. Szalinski said no.

17. At approximately 2 p.m., Investigator Ray Bradford entered the interrogation room and continued the questioning. I told him that I was tired of being accused of hurting Steven and tired of being lied to and held hostage. I again demanded an attorney and the right to use a phone, and I refused to answer any further questions, and I gave them the same information, over and over. At that point, I had been detained and questioned for over 15 hours.

18. Investigator Bradford brought me a phone, and I called my mother, Linda Liebich and stepfather, Walter Sikocinski, and asked them to try to contact an attorney and to come down to the DuPage County Sheriff's office. After the phone call, I sat in the interrogation room in silence and refused to respond to Lt. Szalinski's and Investigator Bradford's continued questions. Eventually they left the room.

19. After I again asked for an attorney, Mr. O'Brien and my fiancée, Kenyatta, came in the interrogation room, and Kenyatta accused me of hurting Steven. I had no choice but to respond since I hadn't harmed Steven in any way. After a few heated exchanges, the officer removed Kenyatta from the room. Lt. Szalinski and Investigator Bradford continued to try and elicit additional statements from me.

20. I was finally released at approximately 4:40 p.m. after 17 ½ hours of involuntary detention and repeated assertions of my constitutional rights.

21. After I was released, I called my mother to ask her come to the jail to pick me up. My mother and stepfather told me they had gone to the sheriff's office when I first called them but were told by detectives that I had been charged with first degree murder and would not be going anywhere. The detectives sent them away.

22. **February 13 interrogation.** On February 13, Det. Figiel, Investigator Bradford and Assistant State's Attorney Dave Imielski came to my parent's home and insisted that I accompany them to the police department. I told them that I did not have anything further to say to them and did not want to go with them. They told me that I had no choice and that they had an interview room set up at the Hanover Park Police Department.

23. I was placed in the officer's car, and my parents followed behind us. I was taken to an interrogation room and questioned for 2 ½ hours. I continued to refuse to give statements, and Inspector Bradford threatened that if I did not cooperate and admit that I was the person responsible for the injuries to Steven, he would be forced to tell the Judge and the State's Attorney that I refused to cooperate with them and that I should think about how they would view that. I clearly stated that I did not harm Steven in any way and that I had nothing further to say. Investigator Bradford then asked if he could bring in a tape recorder and go over the events of February 8. I said no, that I wanted an attorney. At that point, I was released to my parents.

24. **February 14 interrogation (Officer Robert Liebich).** On February 14, after hearing that a t.v. news channel was reporting that I was "on the run" from a murder investigation, my cousin, Dion Liebich, came to my parent's home in Hanover Park and told me that one of our cousins, Officer Robert Liebich, of the Roselle Police Department was trying to locate me. Dion convinced me to accompany him to the Roselle police department to clear up the insinuation that I was "on the run."

25. After arriving at the Roselle police department, I told an officer that Officer Robert Liebich was looking for me but it wasn't clear why. The officer told me to wait and he would have dispatch call Robert in. Dion and I stepped outside to smoke a cigarette and Robert approached us, telling me to follow him inside so he could ask some questions. I said I'd prefer to stay outside but he said that they had a room inside.

26. Robert put us in an interrogation room and closed the door, and began asking questions about what happened to Steven. No Miranda warnings or waivers were read or signed. I answered his questions, as set forth in my affidavit. There was about an hour of heated interrogation. I was uncomfortable talking to Robert since, even though we were cousins, I had only met him a couple times previously. I told Robert I was not on the run and didn't know why he wanted to see me. I said that I wasn't comfortable being questioned by a relative, that I

hadn't signed any waiver of my rights, and that I wanted to know why I was in an interrogation room. I was then released.

27. My sister Denise told me that at about 10 a.m. on February 14, Det. Figiel asked her to locate me and to determine why I hadn't called about the polygraph examination. I contacted Det. Figiel and told him that I had contacted an attorney, Dennis Born, and he could call him at 847-501-3388. Mr. Born had told me that polygraph tests are voluntary, unreliable and not admissible, and that any attorney would advise me not to take one. I told the detective that I was not going to take a polygraph test based on Mr. Born's advice.

28. **Arrest.** At about 11:55 p.m. on February 28, I was arrested, and sheriff's deputies continued to elicit statements from me. I repeatedly told them that I wasn't waiving any rights and that I wanted an attorney present for any questioning.

29. On March 1, at approximately 1 a.m., I was brought by sheriff's deputies to an interrogation room. Sheriff's deputies, detectives and an assistant state attorney were present, and I was verbally read my Miranda rights. I said that I understood my rights and refused to sign the waiver. They told me to think it over, and left the room. A few minutes later, I knocked on the door. When a detective opened the door, I told everyone again that I wanted an attorney. The sheriff's deputies then transported me to the jail facility.

30. **Reports.** I have attached three police reports by Det. Figiel (7 pp, 3 pp and 1 page); a handwritten report by Officer Robert Liebich (3 pp); a report by Richard T. O'Brien (3 pp); a waiver form dated 1:05 a.m. on Feb. 9 (1 page); and a waiver form dated 11:20 a.m. on February 13 (1 page).

31. **Requests to suppress.** I repeatedly told my trial attorney about requesting an attorney and how they ignored my requests to use the phone, and asked him to suppress the reports. He said that the statements didn't hurt his strategy, so he wasn't going to challenge them. However, he never took the time to get into any detail about the case or to investigate anything until the weekend before trial started. At that time, he told me there were no legal grounds to challenge the statements and that he wasn't going to waste time doing it.

I swear under penalty of perjury that the foregoing is true and correct to the best of my knowledge and belief.

Randy Liebich

Randy Liebich
Stateville Correctional Center
P.O. Box 112
Joliet, ILL 60434-0112

Subscribed to and sworn before me this 25th day of February 2008.

Notary Public in and for the State of Illinois

OFFICIAL SEAL
SHERWIN K. MILES
NOTARY PUBLIC, STATE OF ILLINOIS
MY COMMISSION EXPIRES 5-20-2012

My commission expires 5.20.2012.

DuPage County Sheriff's Police
Follow up Report

ORIGINAL

Incident # 02-4531 Page 1

	TYPE OF INCIDENT	DATE OF FOLLOW-UP	TIME	Complainant's name
0110	Homicide	2-8-02	1950	Chicago PD

Code	NAME: LAST, FIRST, MIDDLE	ADDRESS	CODE	D.O.B.	S	R	TX HOME	TX BUS
C	Officer Sullivan John #8479	10th District Chicago PD 2259 S Damen	165	Adult	M	W	Cell 773-203-1779	773-747-5028
W	Officer Filipiak Thomas #19723	Chicago PD 10th District 2259 S Damen	165	Adult	M	W		773-747-5028
V	Quinn Steven	16 W 505 Mockingbird Apt 204	793	041799	M	B	No Phone	
S	Liebich Randy R	16 W 505 Mockingbird Apt 204	793	071279	M	W	330-1432	
W	Brown Kenyatta M	16 W 505 Mockingbird Apt 204	793	100683	F	B	773-722-7976	Cell 708-646-5801
W	Liebich Angelique Marie	16 W 505 Mockingbird Apt 204	793	012702	F		No Phone	
W	Lt. Szalinski	D.P.S.O.	512	Adult	M	W		682-7279
W	Det Delgiudice	D.P.S.O.	512	Adult	M	W		682-7865
	Det Sgt Kunz	D.P.S.O.	512	Adult	M	W		682-7278
	Det Sgt Price	D.P.S.O.	512	Adult				682-7802
W	Investigator Vrbos. Boris	DuPage County Children's Center	512	Adult	M	W		681-2432
W	Smith Tammy Registered Nurse	Rush Presbyterian St. Lukes	165	021964	F	W	708-366-3635	312-942-6191
W	Dr. Severin Paul N	Rush Presbyterian St. Lukes	165	Adult	M	W	Pager 312-333-4251	312-942-6194
W	Dr. Munoz Lorenzo Pediatric Neural Surgeon	Rush Presbyterian St. Lukes	165	Adult	M	W	Pager 877-665-4050	
W	Assist S.A. Guerin Dan	DuPage County States Attorney Office	512	Adult	M	W		682-7760
W	Assist S.A. Brennan Liam	DuPage County States Attorneys Office	512	Adult	M	W		682-7669
W	Assist S.A. Reidy Michael	DuPage County States Attorney Office	512	Adult	M	W		682-7669
W	Deputy Chief Bradford Ray	DuPage County Children's Center	512	Adult	M	W	Pager 722-8582	681-2426
W	O'Brien Richard T	15 Spinning Wheel Road	796	Adult	M	W		325-4404
	Clark Karen L	714 S Independence Blvd	165	101564	F	B	773-722-7976	773-736-9636
	Herron Dorothy J	4937 W Augusta Blvd	165	111049	F	B	773-379-9440	773-419-8317

#502

MR 10

OFFICER #1 CLEARED

DuPage County Sheriff's Police
Follow up Report

ORIGINAL

Incident # 02-4531

	TYPE OF INCIDENT	DATE OF FOLLOW-UP	TIME	Complainant's name
0110	Homicide	2-8-02	1950	Chicago PD

W	Liebich Denise M	1231 Prairie Brook Drive Palatine	999	040670	F	W	847-991-0731	847-991-464(
W	Patio Restaurant	7440 Rt 83	722					920-0211
W	Mileusnic Darinka	Cook County Medical Examiner 2121 W Harrison	165	Adult	F	W		312-666-050(

POLICE ACTION: REFER TO STATES ATTORNEY [ ] COMP. SIGNED YES [ ] NO [ ] ARRESTS: TYPE____ # OF PERSONS_____ OTHER ____

NAME OF ARRESTEE	CHARGE	STATE #	I. R. #	C.C OR TICKET #

NARRATIVE

Lead# 1

2-8-02 1950hrs. This detective received a telephone call from Lt. Szalinski at the office and indicated that there was a report of an injured infant, possibly a shaken baby syndrome case from Willowbrook. The child was currently at Mount Sinai Hospital in Chicago. The Chicago Police Department c"ed our office and two Chicago police officers were at the hospital. It was requested that this detective one of the officers on his cell phone, an Officer Sullivan.

2-8-01 2005hrs. This detective called Officer Sullivan who related what information he had at this point. The victim child, Steven Quinn, a two-year-old male black was in critical condition. This child had trauma to the head, scrotum and bruises throughout the body. It was indicated that the doctors could not tell if the injuries were recent. The mother of the child, eighteen-year-old Kenyatta Brown and her twenty two year old boyfriend, Randy Liebich transported the child to the hospital in their car. Steven Quinn is a child from a previous relationship of Kenyatta Brown not between her and Randy Liebich. Also in the company of this couple was another 11-day-old female infant, Angelique Liebich who is their natural child. The explanation Randy Liebich gave to the Chicago Police Officers in relation to what occurred was that he was at home in Willowbrook sleeping this afternoon. When he got up he fed the victim child a hot dog and Steven started to choke. After Steven stopped choking he laid Steven down and the child became unresponsive. The reason given for their drive from Willowbrook to Mount Sinai hospital in Chicago was that Kenyatta Brown felt more comfortable at this particular hospital. Randy Liebich was very vague with his explanation. Officer Sullivan related that he notified D.C.F.S. in this incident. It was also indicated that the Chicago Officers would stay at the hospital until personnel from this office arrived. It was further indicated that Steven may be transferred to Rush Presbyterian St. Lukes due to his serious condition.

2-8-02 2020hrs. This detective called Lt. Szalinski back and informed him of the situation. He indicated he would be coming to the office.

2-8-02 2100hrs. At the office Lt. Szalinski paged A.S.A. Dennis Harrison who was on call.

2-8-02 2115hrs. This detective phoned Officer Sullivan who related that Steven Quinn had already been transported to Rush Presbyterian Hospital and was on floor 5 (Kellogg) in pediatrics.

2-8-02 2120hrs. Lt. Szalinski contacted the DuPage County Children's Center and was advised an agent would respond to our office.

MR11

OFFICER #1 | CLEARED

DuPage County Sheriff's Police
Follow up Report

ORIGINAL

	TYPE OF INCIDENT	DATE OF FOLLOW-UP	TIME	Complainant's name
0110	Homicide	2-8-02	1950	Chicago PD

2-8-02 2145hrs. Criminal Investigator Boris Vrbos from the DuPage County Children's Center arrived at the office. He was advised of the incident and he called Assistant States Attorney Dan Guerin of the domestic violence section to relate what had transpired and information known at the time.

2-8-02 2150hrs. This detective and Investigator Vrbos left our office and rode together to Rush Presbyterian St. Lukes Hospital. Lt. Szalinski, Det Sgt Kunz and Det Delgiudice also drove out to the hospital.

2-8-02 2240hrs. These investigators arrived at the John L. & Hellen Kellogg Pavilion pediatric critical care unit floor of the hospital and met with the Chicago Police Officers, registered nurse Tammy Smith and attending physician Paul Severin in a conference room marked 537. The Chicago Police Officers related further that the mother, Kenyatta Brown was at work all day and her boyfriend Randy Liebich was watching the kids at their apartment. Around 1500hrs the victim child Steven Quinn had choked for a short period on his hot dog and at 1700hrs the mother came home from work. It was at this time that they decided to take Steven to a hospital. The mother and boyfriend had Steven for the last 3 to 4 days, prior to this the child was being taken care of by his grandmother.

Attending physician Paul Severin spoke of the injuries the child sustained. It was indicated that there was a bleed in the brain on the right side. There was internal abdominal injury. The child was showing signs of severe brain injury. Steven had bruising about the head, he had marks on his back and on the area of his inner legs. There was a little blood in his urine. He was brought into the hospital at hrs and went into surgery at 2158hrs., he was still currently in surgery. Randy Liebich and Kenyatta Br n originally brought Steven into Mount Sinai Hospital at 1800hrs. Nurse Tammy Smith related that Steven weighed almost thirty pounds and when brought into the hospital the child had a low body temperature. The surgery was in an effort to reduce blood swell to the brain. According to Doctor Severin, the child was grunting and not crying, he was also posturing, meaning he had abnormal movement. Doctor Severin demonstrated this type of movement by holding his arms at the sides of his body then he pushed his arms forward up around his chest. He said that this type of movement was consistent with severe brain injury. It appeared the child had injury of a vein that is below the outer portion of the skull and brain. There were no broken bones observed by a visual check. The bruising on his body looked relatively recent and all bruising appeared to be of the same age. An opinion by Doctor Severin which he indicted would be a guess on his part is that the bruising occurred sometime between 24 to 48 hours. None of the bruising stood out more than the other. Pediatric Neural Surgeon Doctor Lorenzo Munoz was performing the surgery.

2-8-02 2320hrs. Nurse Tammy Smith was requested to bring the boyfriend Randy Liebich to the conference room for the purpose of conducting an interview with him. She said at least she could get him away from Kenyatta Brown's family members who were making comments that he was probably involved in this. Tammy also noted that Randy Liebich clenched the infant Anglique close to him in his arms while at the hospital.

2-8-02 2325hs. Tammy Smith brought Randy Liebich into the conference room. This detective and investigator Boris Vrbos then conducted an interview with him. Randy Liebich related that he was watching Steven this morning. The child's mother left for work at 1030hrs., she works at Car-Lene Market Research inside the York Town Mall in Lombard. Randy Liebich said that Steven sat down at the kitchen and ate a bowl of Apple Jacks cereal but didn't drink the milk. Randy said that Steven didn't want to ything yesterday. After eating his cereal, Steven then played around with the small dog they have.

MR12

OFFICER #1	CLEARED BY	DATE
G. Figiel #502	TJS #176	3/5/02

	TYPE OF INCIDENT	DATE OF FOLLOW-UP	TIME	Complainant's name
0110	Homicide	2-8-02	1950	Chicago PD

Randy related he (Randy) was on the mattress in the living room and fell asleep around 1430hrs. and woke up at 1500hrs. When he got up he made Steven a hot dog. Randy said that Steven only ate half of his hot dog. Steven had a glass of water and when he began drinking the water he started to choke and then threw up. Randy related that he patted Steven on the back because he was breathing funny, he was wheezing. Randy asked Steven if he was OK, Steven verbally said, "Yea" and shook his head up and down. Randy laid Steven down by him on the bed which is on the floor. This bed is a mattress on the floo in the living room. Randy said that Steven moaned a little then he fell asleep. Kenyatta came home from work at 1600 or 1630hrs and woke him up. Randy informed Kenyatta of what happened with Steven. Because of the difficulty Steven experienced in his breathing they decided to take him to the hospital. Steven at this time was not talking, he just looked around. Randy carried Steven to the car, Kenyatta drove to the hospital. Randy related that Steven hadn't been eating, he wasn't saying much and he was not as active as he usually was. It was indicated that he still moved around and played but not as much. Steven was at his grandmother's residence for two weeks, he's been back with Randy and Kenyatta for the last three days. The last time Steven was at the doctor was two weeks ago, this was a general examine and he was fine.

Randy and Kenyatta have been living at the apartment on Mockingbird in Willowbrook for the last three months. Kenyatta's name is on the lease and they both contribute to the rent. They moved there because the rent is cheap, they only pay ninety-nine dollars a month. Before this, Kenyatta lived with ıdy's parents in Hanover Park and then they moved to Palatine. Randy's mother didn't get along with r ʻatta. Randy has known Kenyatta for the last two years. They have plans on being married within the next couple of months. They met at a friend's house of Randy's in the city of Chicago, the friends name is Charles.

Randy works at the Patio Restaurant in Darien, he has been employed there for the last two and a half weeks. Prior to this job he worked at Foot Locker in the York Town Mall in Lombard as a manager. He lost that job after Christmas.

Randy indicated that Steven is an active kid and that he does cry a lot. His little girl Anglique cries too and sometimes Randy is unable to get any sleep. Randy said that Steven is a bed wetter. They place a diaper on him at night because he sometimes has accidents, usually they need to remind him to go. They have to ask Steven about going poop to, sometimes he goes in his diaper. Sometimes he goes on himself and other times not.

Randy was asked what kind of clothing Steven had been wearing yesterday. It was indicated he wore black sweatpants and a blue Rock a Wear sweatshirt.

The following were some points that were gone over. Steven was still sleeping when Kenyatta left for work. When Kenyatta left, Randy called out for Steven to get up, he did and came over to the kitchen table to eat his cereal. Going back to the time Randy said that Steven threw up, Randy indicated he picked Steven up and Steven's body went limp into Randy. Randy did ask Steven if he was OK, Steven nodded his head that he was. Randy was asked if Steven fell or bumped his head at all in the apartment. Randy related that Steven did not fall in the apartment at anytime that morning. Randy fed infant Angelique baby formula around 1030hrs and again fed her formula when Steven was eating his hot dog. It was indicated that Anglique sometimes spits up her formula. Randy cleaned up Steven's throw up with kins and the napkins were thrown in the garbage can at the apartment. After eating his hot dog, ɜn laid down on the mattress in the living room and watched television.

MR 13

OFFICER #1	CLEARED BY	DATE
G. Figiel #502	775#176	3/5/02

	TYPE OF INCIDENT	DATE OF FOLLOW-UP	TIME	Complainant's name
0110	Homicide	2-8-02	1950	Chicago PD

Going over the story again, Randy related that everyone woke up at 1000hrs. Kenyatta got dressed and walked out the door after filling a bowl of cereal for Steven. Steven did eat all of his cereal in about five to ten minutes. Randy knew about the times he has indicated because there is a clock in the apartment. Steven then sat down on the mattress in the living room and watched television and patted the dog. Around 1400-1430hrs. Randy fell asleep on the living room mattress with Anglique who was in his arms. Steven was still roaming around. Sometime around 1430hrs or just after, Randy got up from sleeping on the mattress and made Steven a hot dog. Randy cut the hot dog up and poured ketchup on the plate too because that is what Steven liked. Steven ate half the hot dog, he had orange juice in a can also. Randy held his daughter during this period. Steven finished the can of orange juice and Randy poured him a glass of water. Steven started to drink the glass of water and started to choke. Randy indicated that Steven was gulping the water down fast. Because of the choking, Randy patted Steven on his back. When this was under control, they all laid down on the mattress in the living room and watched Jurassic Park 2 that was on tape in the recorder. The end of the movie was never seen, they fell asleep. Kenyatta came home between 1600-1630hrs and woke them all up. Kenyatta discovered that something was wrong with Steven and they all left the apartment at 1630hrs or shortly thereafter to go to the hospital. The ride to Mount Sinai was about thirty minutes long. Randy related that Steven never stopped breathing nor was he ever unconscious. Randy was asked if he ever left the apartment at all between the time Kenyatta left for work and the time she returned back. Just after the time Steven threw up and was lown on the mattress in the living room, Randy said he left the apartment to go to the McDonald's to a girl there for a cigarette. He said he didn't know this girl personally but has seen her on occasion having a cigarette outside. He described this girl as being on the chunky side who has blonde hair and was about 18 years old. This girl gave him a cigarette and he returned back to the apartment, he was gone for less than 5 minutes. Randy left the door unlocked to the apartment and both kids were asleep during the time he was gone. When Randy returned back to the apartment everything was as he left it. Randy was asked about his dog. His dog is the type that is friendly and doesn't bark at all. Randy related that he and Kenyatta had not been involved in any arguing recently, that they have been getting along and were pretty much in love. The only two children at the apartment were Steven and Anglique. No one else stopped by the residence when Randy was there with the children, he was home with the kids by himself. Randy said he basically laid around the residence most of the day. He said he changed Steven's diaper that morning sometime after Kenyatta left for work because it was wet. Throughout the interview, Randy was asked if he wanted anything to drink and or eat.

2-9-02 0015hrs. This detective and Investigator Vrbos left the conference room and were told by a nurse to view Steven who had recently come out of surgery. Steven was seen in a critical unit room, nurse Tammy Smith who was inside the room said that Steven was declared brain dead, he was on life support.

2-9-02 0025hrs. This detective went to his unit and retrieved a tape recorder and cassette tapes.

2-9-02 0105hrs. This detective and Investigator Vrbos went back inside the conference room where Randy Liebich was seated and admonished him Miranda. Randy related that he understood his rights and signed the waiver form indicating so. He agreed to speak with us about what occurred with Steven again. These investigators stepped out of the room again. A taco salad was supplied to Randy.

2-9-02 0115hrs. These investigators went back inside the conference room and again spoke with y, his story was gone over again. He had denied harming Steven. His story was the same except for

MR14

OFFICER #1	CLEARED	
G. Figiel #502	BY 775#176	DATE 3/5/02

	TYPE OF INCIDENT	DATE OF FOLLOW-UP	TIME	Complainant's name
0110	Homicide	2-8-02	1950	Chicago PD

the following. Randy was told that the bruises on Steven's head were inconsistent with what he was telling us. Randy was asked how these bruises got there and if he ever fell. Randy said that Steven fell c his right side when he patted him on the back during the time he choked on the water. Steven fell to his right side and hit his head on the floor but Randy indicated it wasn't hard. Randy was asked if Steven appeared OK prior to him eating his hot dog and having his orange juice. Randy indicated that he was.

2-9-02 0210hrs. The interview with Randy ended. Randy wanted to go to the room where Steven was, he was instructed that he couldn't at his time. Randy then went with Lt Szalinski and Sgt Kunz to th family area room.

2-9-02 0230hrs. This detective, Investigator Vrbos, Det Delgiudice and Assistant S.A. Liam Brennan discussed this incident in the conference room.

2-9-02 0315hrs. Randy was approached again by this detective and Investigator Vrbos in the family area waiting area. Randy was brought over to see Steven and to view the injuries on Steven's body. Nurse Tammy Smith was also in the room. A conversation with Randy concerning the condition of Steven was conducted in this room. Randy was advised that Steven was clinically brain dead. These investigators questioned him as to why this happened and that his story on the events that occurred Friday were inconsistent with the severe injuries sustained by Steven.

2-9-02 0325hrs. Randy returned back to the family waiting room.

2-9-02 0340hrs. This detective, Investigator Vrbos, Lt Szalinski and A.S.A. Liam Brennan spoke in in the conference room discussing this incident. Lt. Szalinski was on the telephone with Det Sgt who was at the Willowbrook apartment.

2-9-02 0405hrs. Hospital waivers were filled out for a full set of medical records on Steven Quinn, these were signed by Randy and Kenyatta. Based on the information received, A.S.A. Brennan indicated that Randy Liebich and Kenyatta Brown were to accompany us to the Sheriff's Office for further investigation into this matter.

2-9-02 0515hrs. This detective spoke with Price on the phone, he read small portions of Kenyatta's diary. Det Sgt Kunz had supplied information on this. He had spoken with Kenyatta who told him that the diary was in her purse inside the apartment and gave permission to retrieve it.

2-9-02 0550hrs. This detective and Det Degiudice checked Randy Liebich's car, a red 1989 Grand AM, Illinois plate of 218 5639, vin 1G2NE54D81C257240 parked outside in front of the emergency room. The keys were obtained from Kenyatta. Nothing of evidentiary value was located within. The vehicles trunk would not open. Kenyatta had told us that the trunk would not open even with the key.

2-9-02 0700hrs. This detective left Rush hospital with Investigator Vrbos and Randy Liebich.

2-9-02 0750hrs. This detective initiated a stop for food at McDonald's on the corner of Rt 38 and County Farm Road in Wheaton. An orange juice and potato cake was purchased for Randy. After the order was made, Randy related that he didn't like McDonald's so we drove across the street to Burger King where Randy was ordered a chicken sandwich by his request.

2-9-02 0800hrs. Arrived at the Sheriff's Office, Randy was provided with a seat in the interview room where he ate.

2-9-02 0830hrs. A.S.A. Michael Reidy arrived at our office. Deputy Chief Ray Bradford from the DuPage County Children's Center was notified and was en route to our office.

MR15

OFFICER #1	CLEARED BY	DATE
G. Figiel #502	TTS #176	3/5/02

	TYPE OF INCIDENT	DATE OF FOLLOW-UP	TIME	Complainant's name
0110	Homicide	2-8-02	1950	Chicago PD

2-9-02 0840hrs. Randy threw up in a waste paper basket inside the interview room. Randy indicated that he was addicted to a twenty dollar a day heroin habit. Randy said that he has been on the synthetic narcotic Methadone for the last three weeks and his supply was out.

2-9-02 0900hrs. Lt Szalinski paged polygraph examiner Richard O'Brien.

2-9-02 1025hrs Richard O'Brien answered the page and indicated that he would be on his way to our office.

2-9-02 1045hrs. This detective paged Det Ledogar and spoke with him by phone. Det Ledogar was requested to stop by the McDonald's across the street from Randy and Kenyatta's apartment in Willowbrook and locate the chunky eighteen-year-old blonde girl who Randy said gave him a cigarette yesterday afternoon.

2-9-02 1150hrs. Richard O'Brien arrived at our office. O'Brien was briefed by Lt. Szalinski on what had transpired in this incident.

2-9-02 1215hrs. Richard O'Brien made his original contact with Randy Liebich in the interview room. Randy indicated to him that he didn't want to take the test today.

2-9-02 1330hrs. Lt. Szalinski conducted an interview with Randy Liebich in the interview room.

2-9-02 1355hrs. Richard O'Brien made original contact with Kenyatta Brown in the second interview room. It was decided by O'Brien that the polygraph test would not be given to Kenyatta today. Kenyatta did agree to take the test tomorrow.

2-9-02 1400hrs. Investigator Ray Bradford from the DuPage County Children's Center joined Lt nski and Randy Liebich in the interview room.

2-9-02 1455hrs. Kenyatta Brown asked Richard O'Brien if she could speak with Randy Liebich about what happened to her son. Richard O'Brien brought Kenyatta into the interview room where Randy was seated.

2-9-02 1510hrs. Both were separated, Kenyatta returned back to the interview room she was originally in.

2-9-02 1640hrs. It was decided by the State's Attorney Office that both Kenyatta Brown and Randy Liebich would not have any criminal charges filed against them at this time. They both left our office several minutes apart from one another.

MR16

OFFICER #1	CLEARED BY	DATE
G. Figiel #502	TJS #176	3/5/02

DuPage County Sheriff's Police
Follow up Report

COPY

Incident # 02-4531

	TYPE OF INCIDENT	DATE OF FOLLOW-UP	TIME	Complainant's name
0110	Homicide	02-12-02	0815	Chicago PD

code	NAME: LAST, FIRST, MIDDLE	ADDRESS	CODE	D.O.B.	S	R	TX HOME	TX BUS
W	Liebich Denise M	1231 Prairie Brook Drive Palatine	999	040670	F	W	847-991-0731	847-991-4646
W	Deputy Chief Bradford Ray	DuPage County Children's Center	512	Adult	M	W		681-2426
W	A.S.A. Imielski Dave	S.A.'s Office	512	Adult	M	W		681-2691

POLICE ACTION: REFER TO STATES ATTORNEY [ ] COMP. SIGNED YES [ ] NO [ ] ARRESTS: TYPE ____ # OF PERSONS ____ OTHER ____

NAME OF ARRESTEE	CHARGE	STATE #	I. R. #	C.C OR TICKET #

NARRATIVE:

Lead# 3

02-12-02 0815hrs. While this detective was on the telephone with another party, Randy Liebich called and left a voice message. He said that he heard this detective wanted to speak with him and that he would call back in a little while. No phone number or other information was provided.

02-12-02 1500hrs. After no further attempt was made by Randy Liebich to call this detective, a call was made to his sister Denise Liebich. She was found to be home and was asked if Randy was with her. Denise said he wasn't and she was asked if she knew where he was. Denise related she didn't know where he was. It was asked if he was in Chicago and Denise replied that she didn't think so. Denise was asked if she could get word to him that this detective wished to speak with him. She said she would. Denise was advised that this detective would be in the office until 1600hrs and that if she couldn't make contact with him today, to please have him call tomorrow during the day. Denise related she would have Randy call. The office number was supplied.

02-13-02 0930hrs. Randy Liebich called this detective. He was asked if he would mind being spoken with in person. He indicated he didn't mind and related that he was staying with his stepfather Wally Sikocinski in Hanover Park at 1941 Hollywood. Randy related that his mother was also staying more. This detective advised on being at his residence within an hour or so of which he was in agreement

OFFICER #1 #80 MR 71 | CLEARED

DuPage County Sheriff's Police
Follow up Report

COPY

Incident # 02-4531

	TYPE OF INCIDENT	DATE OF FOLLOW-UP	TIME	Complainant's name
0110	Homicide	02-12-02	0815	Chicago PD

02-13-02 1105hrs. This detective, Ray Bradford from the DuPage County Children's Center and A.S.A. Dave Imielski went over to the Hollywood address and met with Randy. He was asked if we could talk over at Hanover Park PD. He agreed and the four of us drove over to Hanover Park PD.

02-13-02 1120hrs. In one of the departments interview rooms, this detective and Ray Bradford spoke with Randy. He was admonished Miranda, he indicated he understood his rights and agreed to speak with us after signing the waiver form.

Randy was advised of the medical evidence against him. That we knew he was responsible for the tragedy to Steven. That he was at the apartment by himself with the children. That he probably didn't think the injuries Steven sustained were as serious as they were. How this could have been an accident on his part and that he didn't intend for this to happen. That he was the only one who could help himself. Randy would sit and listen for long periods of time and on occasion when the subject of harming Steven was brought up he would say that he'd never hurt Steven like that. Randy would say that he loved Steven ouldn't do that to him. Various other subjects were covered which included how a judge, a jury and th tates Attorney would view him. How his family would view him. Randy was told that nothing could be promised to him and that we could not make any deals but that his cooperation and any regret on his part could be passed on to the prosecutor's office. This was the general conversation with Randy. It was discussed in a very repetitive manner, which lasted two and a half hours. In addition to denying he hurt Steven, Randy indicated that just before he and Kenyatta left for Mount Sinai hospital with Steven, he took his dog out to relieve itself. When he returned back to the apartment they left.

02-13-02 1350hrs. Ray Bradford left the interview room, this detective stayed with Randy. The conversation continued on about how his family would view him. Randy was asked and indicated that he had only eaten twice since last Saturday and that he wasn't getting the proper rest he should. This detective expressed the fact that he needed to release this from within because it was built up inside of him, that he should to do this for his daughter Angelique and especially for Steven. Randy started to cry but continued to deny hurting Steven.

02-13-02 1410hrs. Ray Bradford returned back inside the interview room, he asked Randy if he was willing to be wired when he spoke with Kenyatta, he related he would. (This was only asked to find hat Randy's reaction would be, there was no intention on going through with this). Randy was also

MR 72

OFFICER #1	CLEARED BY	DATE
G. Figiel #502	TA #770	3/11/02

	TYPE OF INCIDENT	DATE OF FOLLOW-UP	TIME	Complainant's name
0110	Homicide	02-12-02	0815	Chicago PD

asked if he would take the polygraph today, Randy said he wouldn't take it today because he was still sick due to his heroin habit. Randy was asked if we could audiotape his account of events that occurred on Friday the 8th, he agreed to do this. This detective left the room to get the tape recorder, Ray Bradford remained with Randy. This detective returned to the interview room after a few minutes to obtain the car keys from Ray Bradford. The door to the interview room was opened and Ray Bradford indicated that Randy had asked for an attorney.

Randy was brought out to the lobby, his mother Linda Liebich was there waiting for him. Before leaving with his mother, Randy was asked if he would take the polygraph tomorrow. He related that if he could obtain his methadone supply he would definitely do so. Randy was advised to call this detective (supplied him a business card) tomorrow at 0900hrs so the examine could be set up. Randy said he would call.

02-14-02 0955hrs. Randy Liebich had not called. This detective called the cell phone number of y Sicosinski, the stepfather of Randy Liebich. No one answered the Nextel phone.

02-14-02 1000hrs. Denise Liebich was called, it was indicated that her brother Randy didn't call this detective at the previously arranged time of 0900hrs. Denise related that she would call a neighbor by Wally Sicosinski's residence and have them go to their door to deliver the message to Randy.

02-14-02 1010hrs. Randy Liebich called this detective on Wally's cell phone. He was advised that the polygraph examiner was scheduled to be in our office between 1400-1430hrs today. Randy was asked if he needed a ride to our office, he related he didn't, that he had a ride. Randy related that he would be in at 1400-1415hrs.

02-14-02 1419hrs. Randy Liebich called and related that he called an attorney, a Dennis Born (847-501-3388) who told him not to come in and take the test. Randy was advised that the test was voluntary, that the decision to take one was entirely up to him and any attorney would tell him not to take one. An offer was again made to supply him with a ride. It was indicated that even if he wasn't feeling that well he should take it. Randy indicated that he wasn't going to come in today and that he hasn't been able to get his methadone. Randy was further advised that Kenyatta took the polygraph test when asked and that this detective wished he would reconsider. Randy related he wasn't coming in today.

MC73

OFFICER #1	CLEARED BY	DATE
G. Figiel #502	#176	3/11/02

ON 02-14-02 AT 1630 HRS, I WAS ADVISED BY DISPATCH THAT I HAD A FAMILY MEMBER IN THE LOBBY.

MY COUSINS, DION AND RANDY LIEBICH WERE WAITING FOR ME. I BROUGHT THEM INTO THE ROOM NEXT TO DISPATCH, AND RANDY WAS ASKING IF IT WAS SAFE TO TALK IN THIS ROOM. I GAVE HIM A QUIZZICAL LOOK, AND HE ASKED IF IT WAS "BUGGED". I SAID "NO".

RANDY SAID HE WANTED TO TALK TO ME AND LET ME KNOW HE WASN'T RUNNING, AND THAT HE HAS BEEN TALKING WITH THE DETECTIVES HANDLING THE CASE. RANDY ALSO WAS CONCERNED ABOUT THE CUT ON HIS FINGER (RIGHT INDEX), SAYING THE KID BIT HIM AND DREW BLOOD. I SAID, "SO WHAT". RANDY WAS WORRIED ABOUT HIS BLOOD BEING INSIDE THE KID'S BODY. I ASKED RANDY WHAT HAPPENED, AND HE STARTED EXPLAINING EVENTS ON 02-07-02 AND 02-08-02.

RANDY SAID HIS GIRLFRIEND "KIKI", WAS HITTING HER CHILD IN THE EVENING OF 02-07-02 BECAUSE HE KEPT COMING OUT OF THE BEDROOM. RANDY SAID HE LIT A CIGARETTE AND WENT INTO THE ROOM. RANDY SAW KIKI HIT STEVEN IN THE HEAD FOUR(4) TIMES. RANDY TOLD HER THAT SPANKINGS SHOULD BE ON THE BUTT. KIKI TOOK A BELT AND STRUCK STEVEN ON THE BUTT. MR10

IN THE MORNING, RANDY SAID STEVEN WAS LYING IN BED WITH HIS EYES OPEN. RANDY SAID STEVEN NORMALLY GETS OUT OF BED RIGHT AWAY AND IS ALWAYS TALKING.

KIKI TOLD STEVEN TO GET OUT OF BED. STEVEN DID GET OUT OF BED AND WALKED OUT ON HIS OWN. KIKI WENT TO WORK, AND LATER RANDY WAS FEEDING STEVEN A HOT DOG. RANDY SAID STEVEN WAS CHOKING AND HE HIT STEVEN ON THE BACK TO DISCODGE THE FOOD. RANDY PUT HIS FINGERS IN STEVEN'S MOUTH TO PULL OUT THE FOOD AND STEVEN BIT DOWN ON RANDY'S FINGER (RIGHT INDEX). RANDY COULD NOT GET STEVEN TO LET GO, SO HE HIT STEVEN IN THE HEAD WITH AN OPEN HAND. RANDY SAID HE HIT HIM ON THE OTHER SIDE THAN WHERE KIKI HAD HIT HIM THE NIGHT BEFORE. RANDY SAID HE HIT STEVEN AGAIN AND THAT'S WHEN STEVEN LET GO. STEVEN THREW UP AFTER THIS AND JUST WASN'T ACTING RIGHT. KIKI CAME HOME A SHORT TIME LATER, AND THEY BOTH TOOK STEVEN TO THE HOSPITAL.

RANDY TOLD ME THAT HE DIDN'T TELL THE DETECTIVES ABOUT THE BITE OR HITTING STEVEN. RANDY ASKED WHAT HE SHOULD DO, AND I TOLD HIM TO TELL THE TRUTH. I ASKED RANDY SEVERAL TIMES IF HE HIT STEVEN HARD ENOUGH TO DO THIS. THE LAST TIME HIS EYES LOOKED AT THE FLOOR, AND I WAS UNCOMFORTABLE BELIEVING HIS ANSWER. I ASKED HIM TO SWEAR ON HIS FATHER'S GRAVE, RANDY LOOKED ME RIGHT IN THE EYE AND THEN SAID HE DIDN'T HIT THE KID THAT HARD.

RANDY ASKED IF I HEARD ANYTHING TO LET HIM KNOW. I SAID NO, IF HE WAS LYING TO ME, THEY WILL COME FOR YOU.

MR 103

DION DROVE RANDY HOME AND CALLED ME AFTERWARD.

DION SAID AN UNMARKED CAR WAS WAITING WHEN THEY ARRIVED. WE BOTH ASSUMED RANDY HAD BEEN ARRESTED. IT WASN'T UNTIL A WEEK LATER THAT I FOUND OUT RANDY HAD NOT BEEN ARRESTED, AND I CALLED DETECTIVE FIGIEL TO INFORM HIM OF MY CONVERSATION WITH RANDY.

Robt Lutz 02270

NOTE

COMPLETED AT ROSELLE P.D.
2-27-02 11:00 AM

MR104

Richard T. O'Brien & Associates, Inc.

15 SPINNING WHEEL ROAD
SUITE 14-B
HINSDALE, ILLINOIS 60521

CHICAGO (312) 236-4242
SUBURBAN (630) 325-4404
FAX (630) 325-4734

February 12, 2002

Lt. Thomas Szalinski
DuPage County Sheriff's Police
Detective Division
501 N. County Farm Road
Wheaton, IL 60187

RE: Battery of a Child

On February 9, 2002, Randy R. Liebich, was scheduled for a polygraph examination, but on this date the subject refused to submit to the testing procedure claiming that he was sick and not in the mood to be taking the polygraph test at this time. The subject was given his Miranda Rights, which he read and signed, copy of which is incorporated as part of our laboratory file in this case.

The subject stated in essence, but not verbatim that the victim's mother, Kenyatta M. Brown, got up on Friday morning to go to work. She had prepared some cereal for the victim, and the subject claims that he did attempt to feed the child however, the child was not very hungry and did not eat much of the cereal. Later in the day at approximately 3 PM, he gave the victim a hot dog to eat. He claims that the victim was choking on some water that he had given him and he patted the victim on his back, and the victim threw up at this point. The subject claims that the victim fell over, but did not injure himself. The subject stated that he and the victim laid down for a while. The subject woke up wanting a cigarette and he left the apartment to run over to the McDonald's restaurant across the parking lot from his residence, leaving the victim and his stepsister alone in the apartment. The subject got a cigarette from a female worker at the restaurant, and smoked it in the hallway of his apartment. He went back into the apartment and laid down until approximately 4 PM when the victim's mother returned home, at which time it was determined that the victim required immediate medical attention and they drove to Mr. Siani Hospital in Chicago.

The subject claims that he witnessed Steven Quinn being beaten with a belt by his mother on Thursday night, and that the child's mother also "popped the victim in the head a few times". The subject stated that he was present in the room but looked away because he did not like to watch the victim being spanked.

JR 2/12

Richard T. O'Brien & Associates, Inc.

15 SPINNING WHEEL ROAD
SUITE 14-B
HINSDALE, ILLINOIS 60521

CHICAGO (312) 236-4242
SUBURBAN (630) 325-4404
FAX (630) 325-4734

RE: Battery of a Child

INTERVIEW OF: Randy R. Liebich

At this point, the subject declined to make any other statements concerning this investigation, and the interview was terminated.

Respectfully submitted,

Richard T. O'Brien
Polygraph Examiner

SS

NR213

Miranda Rights

Date 2-9-02

I have been informed by Richard D. O'Brien, polygraph examiner, that he wants to question me about the inquiries to Steve Quast on 8 Feb 02

He has told me that I have the right to remain silent and that I do not have to agree to be questioned at this time unless I wish to do so. He has also told me that even though I agree to be questioned I have the right to change my mind at any time during the questioning and can refuse to answer. I may also request that the questioning be stopped and he will abide by any such decision. He has further advised me that anything I say may be used against me in court at some future time.

He has further advised me that I have a right to consult with an attorney or in the event that I can not afford to retain an attorney one will be appointed to represent me. I have a right to have said attorney present if I wish during the time I am being questioned.

I fully understand what I have been told and I hearby agree that I am willing to discuss the ABOVE;
I agree to be questioned at this time and I do not wish to consult with, retain, or have an attorney appointed to represent me.

I have been requested to read the above statement, which I have done, and hearby state that there has been no interrogation about the ABOVE prior to my having read and signed this statement.

MR
217

X Signature Randy Cook

DU PAGE COUNTY SHERIFF
FIELD INTERROGATION WAIVER

A. You have a right to remain silent and do not have to say anything at all.

B. Anything you say can and will be used against you in Court.

C. You have a right to talk to a lawyer of your own choice before we ask you any questions, and also to have a lawyer here with you while we ask questions.

D. If you cannot afford to hire a lawyer, and you want one, we will see that you have a lawyer provided to you, before we ask you any questions.

E. If you are willing to give us a statement, you have a right to stop any time you wish.

F. Do you agree to answer our questions here and now?

YES ✓ NO .....

G. [signature] .....
Signature

Witness [signature] .....

Witness [signature] .....

Time and Date 1:05 A.M. 2-9-02

SO-00251

MR 223

DU PAGE COUNTY SHERIFF
FIELD INTERROGATION WAIVER

A. You have a right to remain silent and do not have to say anything at all.

B. Anything you say can and will be used against you in Court.

C. You have a right to talk to a lawyer of your own choice before we ask you any questions, and also to have a lawyer here with you while we ask questions.

D. If you cannot afford to hire a lawyer, and you want one, we will see that you have a lawyer provided to you, before we ask you any questions.

E. If you are willing to give us a statement, you have a right to stop any time you wish.

F. Do you agree to answer our questions here and now?

YES ...X... NO .....

G. ...Randy Lieb...
Signature

Witness ...B Tigl...

Witness ...Raymond J. Bradford...

Time and Date ...11:20A — 2-13-02...

SO-00251

MR 824

JOHN E. ZARUBA
SHERIFF

501 N. COUNTY FARM ROAD
WHEATON, ILLINOIS 60187
ADMINISTRATION (630) 682-7269
CIVIL DIVISION (630) 682-7250

OFFICE OF THE SHERIFF
COUNTY OF DUPAGE

DUPAGE COUNTY SHERIFF'S OFFICE
RIGHTS ADVISEMENT WAIVER FORM

RL 1. You have the right to remain silent.

RL 2. Anything you say can be used against you in a court of law.

RL 3. You have the right to talk to a lawyer and have them present with you during questioning.

RL 4. If you cannot afford to hire a lawyer, one will be appointed to represent you without cost before any questioning.

RL 5. You can exercise these rights at any time.

RL 6. A videotape providing a video and audio record of this interview is being made and I voluntarily consent to this recording.

I understand all of the above rights and voluntarily waive those rights.

Signature: _____

Name: _____
(print)

Witness: _____ **Date:** 3-14-02

Witness: _____ **Time:** 1:00 A.M.

Case Number: _____

MR 775

SO-00267-B

DuPage County Sheriff's Police
Follow up Report

COPY

Incident # 02-4531

CODE	TYPE OF INCIDENT	DATE OF FOLLOW-UP	TIME	Complainant's name
10	Homicide	2-28-02	2350	Chicago PD

code	NAME: LAST, FIRST, MIDDLE	ADDRESS	CODE	D.O.B.	S	R	TX HOME	TX BUS
W	Det Delgiudice	D.P.S.O.	512	Adult	M	W		682-7865
W	A.S.A. Guerin	S.A.'s Office	512	Adult	M	W		682-7760

POLICE ACTION: REFER TO STATES ATTORNEY [ ] COMP. SIGNED YES [x] NO [ ] ARRESTS: TYPE 1 # OF PERSONS 1 OTHER

NAME OF ARRESTEE	CHARGE	STATE #	I. R. #	C.C OR TICKET #
Liebich Randy R	First Degree Murder	L29676919	154095	02-CF 654

NARRATIVE:

02-28-02 2355hrs. This detective was contacted by Det Delgiudice at home who indicated that Randy Liebich had been picked up on the first degree murder warrant by Deputies Dubeck and Josic.

03-01-02 0015hrs. This detective arrived at the Sheriff's Office and met with Det Delgiudice and A.S.A. Dan Guerin.

03-01-02 0045hrs. Randy Liebich was brought into the interview room by Deputies Dubeck and Josic.

03-01-02 0100hrs. Liebich was spoken with after his Miranda rights were read to him. Liebich said he understood his rights but refused to sign the waiver form. In summary, it was explained that the evidence gathered in this case showed he was involved in the death of Steven. It was indicated that he could only help himself and that the time to do this was now. Liebich was given an opportunity to be alone and think about what he was told. He wanted something to drink and also to smoke a cigarette, a Coke and cigarette were provided. He was advised to knock on the door when he was ready to speak with us again.

03-01-02 0210hrs. Liebich knocked on the door, he indicated that he thought about it and wanted an attorney.

03-01-02 0220hrs. Deputies Dubeck and Josic brought Liebich to the jail facility.

This case is cleared with arrest. 3

m823

OFFICER #1	CLEARED BY	DATE
G. Figiel #502	TTS#7C	5/1/02

STATE OF ILLINOIS)
) **AFFIDAVIT OF DION LIEBICH**
COUNTY OF DUPAGE)

I, Dion Liebich, being first duly sworn on oath, depose and say as follows:

1. My name is Dion Liebich. I work for 6-D Aluminum, which is located in Crest Hill, Illinois (phone 708-487-1855). I am Randy Liebich's cousin.

2. In February 2002, I saw a television report saying or implying that my cousin Randy had murdered his girlfriend's son, Steven. I immediately called Randy's sister, Denise, who told me that she heard that Steven had been beaten when Kenyatta was at work and Randy was looking after him.

3. When I heard this, I was extremely angry. This was completely unlike anything I knew of Randy, and I could not believe he had done this. However, since he was the only one home, he had to know what happened.

4. Denise, Randy's sister, told me that Randy was at his mother's, and I drove there immediately and grabbed Randy by the back of his neck. Before we talked about what happened, we decided to go to see our cousin, Robert Liebich, who is a police officer in Rosselle, Illinois. I don't remember whose idea this was but I thought this would let us figure out what was going on. I gave Randy a ride to the Roselle police station. It was a very quiet ride. Since Randy generally did not trust the police and wasn't close to Robert, I thought that Randy was going to confess or at least tell Robert what really happened.

5. Robert took us in an interrogation room. Robert was very angry, and there were a lot of raised voices. There were no Miranda warnings or anything like that. This was a full interrogation, with a lot of anger directed at Randy. Robert and I wanted answers on how Steven died, and Randy wasn't giving us answers. Robert and I had kids, and we were angry with Randy for letting this happen to a child. We thought he had to know what happened since he was there.

6. I let Robert do the questioning since he was a police officer and experienced at doing this. Randy said that Steven choked on a hot dog and that Randy put his finger in Steven's mouth to see if something was stuck in his throat. When he did this, Steven clamped down on the index finger of his right hand. Randy said that when Steven wouldn't let go, he hit Steven on the side of the head to get him to let go. Randy showed us a small cut by his fingernail where he said Steven had bitten him.

7. Robert questioned Randy on how he struck Steven, and Randy illustrated. He showed a tapping from a few inches away that should not have hurt anyone. It

was almost like a push, more pressing than hitting. Robert went over this with Randy several times. Randy's description seemed like a natural instinctive reaction, exactly what I think most people would have done if someone was biting their finger. It did not seem like anything that would have hurt Steven.

8. The atmosphere in the room was very tense and filled with anger, almost hate, since nothing Randy described explained the serious injuries that we heard about. Robert kept telling Randy this but Randy kept telling the same story, over and over again. Robert and I were very angry that Randy wouldn't explain how Steven ended up dead.

9. Finally, Robert asked Randy if he would swear on their fathers' graves that he was telling the truth and the whole truth. Randy was very close to his father, who died when he was a teenager. Robert's father had also died fairly recently. In our family, this type of oath means a lot. Randy said that he swore on their fathers' graves that he was telling the truth.

10. At first, Randy looked at the floor when he said this. I didn't put a lot of importance on this since Randy always looked at the floor when he was upset. When his father died unexpectedly, almost everyone was crying. However, Randy, who was very close to his father and the most upset, sat by himself with his head down. That is how he looked at the police station. Robert, who doesn't know Randy as well as I did, asked the same question again, maybe in different words, and Randy looked Robert straight in the eye and repeated that he swore on their father's graves that he was telling the truth and that he had not hurt Steven.

11. Randy talked about Steven not feeling well the week before he collapsed. He also said that Steven didn't seem that sick after he choked on the hot dog but that after Kenyatta came home they noticed that he was making odd moaning noises and they took him to the hospital. Randy said he took the dog out first. Robert and I didn't pay much attention to any of this since it didn't explain how Steven died.

12. Robert was angry and frustrated because Randy kept saying the same things over and over and wasn't giving any explanation for Steven's condition. Since Robert's father died of a cocaine overdose, Robert despises drugs and looks down on people who use them, including Randy. He was very harsh, therefore, in interrogating Randy. He wanted a confession, and he wasn't getting it.

13. I did not know what to believe. I knew Randy much better than Robert did since I spent a lot of time with him after his father died. Randy could be easily persuaded to do things, but he was never violent and he was really good with kids.

14. Robert kept asking me things like, "what do you think?" I didn't know what to think. Robert said he was trained to know when people were lying and that he knew for a fact that Randy was lying. He seemed to think Randy was lying because he hung his head, but I knew that Randy did this when he was depressed.

15. Robert asked Randy over and over if he could have hit Steven hard enough to hurt him or kill him. Randy always said no. I asked the same questions, and Randy gave the same answers. When Robert asked important questions like this, Randy would look him straight in the eye and say that he didn't do anything that could have hurt Steven and that the only unusual thing that day was when Steven choked on the hotdog. He kept saying that he didn't know what happened to Steven. Robert and I were angry that a three year old had been killed and no one could give an explanation.

16. When Randy was going with Kenyatta, I helped him get an apartment and a job. When I picked him up for work, he talked about problems with Kenyatta. I didn't like the situation with Kenyatta, and I didn't want to hear about the problems.

17. When I visited Randy after ~~Angelique was born~~ DSL, it was over ninety degrees in their apartment, and Kenyatta was frying chicken in the kitchen. It was much too hot for the children, so I went out and bought an air conditioning unit and put it in the window. When Steven went over and started to play with the knobs, Kenyatta grabbed him by the left arm, opened the door to the bedroom and chucked him in, like she was throwing a baseball.

18. I saw this from the kitchen table, and I jumped up and started going after Kenyatta, saying, "how could you do this?" Randy got in the middle, and I yelled at him too, saying, "how can you let this happen?" I told both of them I would call the department of child services if I ever saw or heard about anything like this again. I told my wife about this after I got home.

19. Earlier, Randy told me that Kenyatta would use broken coat hangers to hit Steven. I didn't know why Randy didn't stop it but I don't think he thought he could. Kenyatta was a strong personality, and I don't think she would have listened to him. I was mad with Randy because I felt he was condoning it by staying with her. However, it may have been worse if he left.

20. In all the years I have known Randy, I have never seen him harm or hurt a child. He has been around many children, including my own son, and he never showed a mean streak and rarely showed impatience. When he and Kenyatta lived with us, Randy and Steven got along well, and Randy seemed to like to have him around.

21. When we heard that Steven had been beaten, it occurred to me that if Steven had been beaten, it was more likely to be Kenyatta than Randy. We all knew that Kenyatta hit Steven, but no one had ever seen Randy hit Steven or any other child. I don't think that Robert knew any of this when he interrogated Randy.

22. Months after Steven died, detectives came to my home. They told me that Randy had hit Steven on the head so hard that his brain swelled and there was bleeding on his brain, and that this was the only explanation for his injuries. Since I was

present when Robert interrogated Randy and heard what Randy had to say, I thought that Randy's attorneys would also want to talk to me, but they never did.

23. Randy and Kenyatta lived with my wife and me for awhile, and they had some fights. I never knew who started them or what happened. One time, when my wife and I were home, Randy and Kenyatta had a fight in the bedroom. Randy left the apartment, and Kenyatta came out crying and saying that he hit her. I gave her a hug because she was crying. She immediately came on to me sexually, and I walked out. She also had men come by in Cadillacs. I told Randy over and over that he needed to break off the relationship, but he wouldn't. This created a bit of problem in our relationship.

24. In my gut, I always believed, and will always believe, that Randy is innocent. I couldn't see Randy hurting a child or not telling what he had done, even when interrogated. The only problem was that he couldn't explain why the child died. Since none of this made sense, I always wanted to know what really happened, and I never felt that anyone had the right answers. .

I swear under penalty of perjury that the foregoing is true and correct to the best of my knowledge and belief.

Dion Liebich

Subscribed to and sworn before me this 19 day of Feb, 2008. 9 (MS)

Notary Public in and for the State of Illinois

My commission expires April 03/2011

OFFICIAL SEAL
MARLENE FRANCES SZAFRANSKI
NOTARY PUBLIC - STATE OF ILLINOIS
MY COMMISSION EXPIRES:04/03/11

IN THE CIRCUIT COURT OF THE EIGHTEENTH JUDICIAL CIRCUIT, DU PAGE COUNTY, ILLINOIS

PEOPLE OF THE STATE OF ILLINOIS, Plaintiff	)	
v.	)	No. 02 CF 654
RANDY LIEBICH, Defendant	)	

AFFIDAVIT OF ROGER LILLY

I, Roger Lilly, under oath and penalty of perjury state as follows:

1. I am an investigator with the DuPage County Public Defender's Office.

2. On February 9, 2012, at the request of attorney Jaime Escuder, I, along with Chief Investigator Jason Harvey, went to 508 W. Columbia Ave., in Champaign, Illinois, to speak with Kenyatta Brown. Kenyatta had changed her last name and was going by the name of Kenyatta Harris.

3. Upon arrival, we handed Ms. Brown our business cards, informed her that we were investigators for Randy Liebich's attorney, that we were not police officers, and that we did not work for the prosecution.

4. Ms. Brown agreed to speak with us.

5. Ms. Brown stated that she woke up late for work on the morning of February 8, 2002. She checked in on Steven who sat up from his sleeping place on the bedroom floor, said hello, and then laid back down. Ms. Brown got dressed, gave Randy instructions regarding feeding Steven cereal and letting him watch television, and then she left for work.

6. Ms. Brown said that Randy had never babysat for Steven, or for their newborn daughter, Angelique, before, but that she trusted Randy to watch both of the children.

7. Ms. Brown said that when she returned from work that afternoon, she found Steven lying on his side, and he appeared to be regurgitating and making a "gurgling sound." Ms. Brown picked Steven up and tried to clear his airway of a "greenish/blackish vomit." She asked Randy what was wrong with Steven, and Randy said that he did not know.

8. Ms. Brown said that she decided to take Steven to Mount Sinai hospital, and Randy said he would come along, but he wanted to stop by his job first, in order to explain why he would not be at work. After stopping at the restaurant where Randy worked, they took Steven to the hospital. Ms. Brown said she chose to go to Mount Sinai because she had recently delivered Angelique there and she was familiar with it. Ms. Brown said that she did not think Steven's condition was not serious enough to call an ambulance, although she also admitted that she was "young and didn't know anything."

9. At Mount Sinai, Ms. Brown said that she was asked about "knots" on Steven's head by one of the nurses. She explained that Steven's skull naturally had lumps. Ms. Brown then said that she felt two bumps, one on each side, of Steven's head. When she asked Randy about these, he stated that Steven had been playing with the dog under the table and may have bumped his head while doing so.

10. Ms. Brown then stated that Steven was transferred to Rush Medical Center by ambulance, where she was informed that Steven's brain was too damaged to treat. After this she was questioned by the police at the DuPage County Sheriff's Office.

11. The next day, Ms. Brown stated that she returned to Rush to pick up her newborn daughter, Angelique. While there, she viewed Steven's body and observed injuries that she had not noticed the day before. These injuries included dark bruises on Steven's ankles, legs, and arms, nicks on the skin of his fingers and his left big toe, and and a scrape and pinkness on his scrotum.

12. When I asked Ms. Brown about Steven's health prior to February 8, she stated that, two or three days prior, Steven had been complaining about stomach pain.

13. Ms. Brown denied knowing why some of the injuries in Steven's abdomen were in a state of healing.

14. When asked, Ms. Brown stated that she never saw Randy hit or otherwise discipline Steven. She said that neither she nor Randy were in charge of disciplining him because Steven was not the type of child to get in trouble.

15. When I asked Ms. Brown about her statements at trial that Steven was crying the night of February 7, 2002, she stated that Steven was continuously crying for no apparent reason, and that she tried to get him to stop. She admitted that she spanked him in an effort to get him to stop crying. She stated that she "slapped him on the butt," but he continued crying. According to Ms. Brown, in response to this, Randy stated "he's laughing at you, he didn't feel that." Randy then handed Ms. Brown his belt and she administered a couple of "licks" to Steven's backside and

told him to lay down. After this, Ms. Brown stated that she went to cook and she left Randy with Steven.

16. While cooking, Ms. Brown said she thought she heard a slap and Steven make an "I'm hurt" kind of cry. She did not see if Randy slapped Steven, and she did not ask him. The family then had some dinner, after which time, according to Ms. Brown, she and Randy smoked marijuana laced with PCP, had sex, and went to sleep.

17. Ms. Brown said that Steven was being watched by her mother during the time that she was giving birth to Angelique, and that she had not heard any report about Steven receiving an injury while staying there. Ms. Brown stated that Steven seemed fine when she picked him up on February 2.

18. Ms. Brown stated that she had received some notes from Randy after his conviction, but that, aside from that, she has had no contact with him.

19. On March 29, 2012, again at Attorney Escuder's request, I called Ms. Brown at (217) 402-0066 and asked her if she would sign an affidavit relating our February 9 conversation.

20. I read the affidavit to Ms. Brown and she said she did not agree with it. I asked her what changes she would make, and she said that she would not sign any affidavit that would assist Randy. Ms. Brown stated that that part of her life is over and she does not want to revisit it again.

Subscribed and sworn to before me
this 4 day of April 2012
Marysol Diaz
Notary

"OFFICIAL SEAL"
Marysol Diaz
Notary Public, State of Illinois
My Commission Expires 11/08/14

Roger Lilly

Date: 4-4-12

IN THE CIRCUIT COURT OF THE EIGHTEENTH JUDICIAL CIRCUIT, DU PAGE COUNTY, ILLINOIS

PEOPLE OF THE STATE OF ILLINOIS, Plaintiff	)	
v.	)	No. 02 CF 654
RANDY LIEBICH, Defendant	)	

AFFIDAVIT OF DENISE FOSTER

I, DENISE FOSTER, under oath and penalty of perjury, state as follows:

1. My name is Denise Foster, and I am Randy Liebich's sister. My date of birth is April 6, 1970, and my address is 1231 Prairie Dr., Apt. 3A, Palatine, IL.

2. Sometime after January 27, 2002, my aunt, Debra Minucciani, and I visited Randy, Kenyatta, Steven, and newborn Angelique in Willowbrook.

3. My aunt and I brought food to the apartment, but Steven refused to eat.

4. I went into the bedroom and tried to talk Steven into eating, but he still refused.

5. Steven was wearing a diaper and a T-shirt, and I saw no marks on his body.

6. I never saw Randy strike or slap Steven at any time.

7. Kenyatta and Steven stayed at my home on occasion, during which time I saw Kenyatta slap Steven on the leg and back of the head in anger.

8. I am giving this affidavit of my own free will. No promises or threats were made to me in exchange for making the statements contained herein. If called to testify, I would testify consistent with this affidavit.

Signature: Denise Foster
Denise Foster

Subscribed and sworn to before me
this 30th day of March 2012

Notary

"OFFICIAL SEAL"
Roger B. Lilly
Notary Public, State of Illinois
My Commission Expires 9/09/12

Date: 3-30-2012

~~DRAFT~~

STATE OF ILLINOIS)
)
COUNTY OF DUPAGE)

AFFIDAVIT OF DENISE FOSTER

I, Denise Foster, being first duly sworn on oath, depose and say as follows:

1. My name is Denise Foster. My address is ~~1231 Prairiebrook Drive, Apt. A3, Palatine, IL 60074~~ 1941 Hollywood Harwood Pk, IL. I have worked at a daycare center at a health club for twenty years. I am Randy Liebich's sister.

2. I testified at Randy's trial about Kenyatta's treatment of Steven. Kenyatta did not take care of Steven. She paid no attention to him, she hit him regularly, and she did not do the things that a mother would normally do. Since I work in daycare, I know how mothers and children behave, and Kenyatta did not have a mothering or nurturing instinct towards Steven. She expected behavior that was not age appropriate, and she would hit him to make him stop crying. This was all well known at the time of Randy's trial, and I testified to some of it.

3. Before Randy and Kenyatta got an apartment, Randy lived with my mother and Kenyatta would stay with them, sometimes bringing Steven. When I went over to visit one day, Kenyatta and my mother were arguing. I don't remember what they were arguing about, but when I turned my back, I heard a thud. When I turned around, my mother was lying on the floor by the door. When I ran over, she was unconscious. Randy also ran over.

4. I was going to call 911 but my mother came to quickly. My mother said that Kenyatta hit or pushed her, which was obvious, and she asked me not to call 911. I didn't call. From the way my mother had fallen, it looked like she had hit the door.

5. After seeing this, I was very concerned about Randy's relationship with Kenyatta. Randy always wants to see the best in people, but sometimes he is wrong. He does not have a temper, I have never known him to hit anyone, and he generally backs away and stays out of potentially violent situations, as he did with Kenyatta. We have many cousins (11 aunts and uncles on my dad's side, with more on my mother's side), so we had many family gatherings. At family gatherings, children always swarmed around Randy. Steven did the same. Randy is very good with children, but I did not think he would be able to stand up to Kenyatta. Instead, he made excuses for her.

6. The incident with my mother caused me real concern for Steven. We already knew that Kenyatta regularly hit and shoved Steven. My concern was that she would do with Steven as she had done with my mother, that is, push him, or shove him, or slam him into something, with serious consequences. The incident with

my mother, who was not a fighter, showed me that Kenyatta did not have much self-control and that she did not think of the consequences of her actions.

I swear under penalty of perjury that the foregoing is true and correct to the best of my knowledge and belief.

Denise Foster

Subscribed to and sworn before me this 3rd day of April, ~~2008~~ 2012

Notary Public in and for the State of Illinois

My commission expires 09-09-12.

"OFFICIAL SEAL"
Roger B. Lilly
Notary Public, State of Illinois
My Commission Expires 9/09/12

STATE OF ILLINOIS)
) **AFFIDAVIT OF MARLENE SZAFRANSKI**
COUNTY OF ~~DUPAGE~~)

I, Marlene Szafranski, being first duly sworn on oath, depose and say as follows:

1. My name is Marlene Szafranski. I am Randy Liebich's aunt. My address is [redacted] Illinois (tel. 815-693-8943). I work for M.J.S. Transport Inc.

2. In the fall of 2001 and 2002, I was the office manager at Carlene Research, a consumer survey company located in the Yorktown Mall in Lombard, Illinois.

3. Randy worked at the Foot Locker in the same mall, and he stopped by the office almost every day to eat lunch or visit. Sometimes he brought his girlfriend, Kenyatta, with him, and they sometimes brought Steven, Kenyatta's son.

4. Randy asked me to help find work for Kenyatta, and I hired her to work for me part-time. She worked approximately 10-12 times in approximately December 2001 to February 2002, usually for about four hours but sometimes longer.

5. If Kenyatta and Randy were working at the same time and Kenyatta was only working a few hours, Kenyatta brought Steven with her as we had a room with a television and he was well-behaved. Since they only had one car, Randy and Steven often came to the office with Kenyatta.

6. If Randy had Steven for the day, he would sometimes call me the night before and ask me to bring children's videos to the office. He and Steven would watch videos together. Randy would pick up food for Steven and eat with him, then he would take Steven out in the mall to walk around and ride the train. Randy was good with children, and he and Steven got along very well.

7. Randy had always been good with children. At family gatherings, the kids would pile on Randy and get him to play with them or read books to them. He enjoyed kids, and they enjoyed him. Randy spent a lot of time with my son, Vince, and with his sister's son, Jeffrey. Vince and Jeffrey adored him, and he never lost patience with them.

8. I only had one significant problem with Kenyatta at work. Since I took weekends off, I assigned Kenyatta to work with Erin on a couple weekends. After one of the weekends, Erin told me that she wouldn't work with Kenyatta when I wasn't there because she didn't like Kenyatta hitting Steven. Around the time of Randy's trial (?), I tried to find Erin but she had moved and I couldn't find a new address for her.

9. When Angelique was born, Randy was excited and happy, and he and Kenyatta brought Angelique to my office. They didn't have many baby supplies, so we went shopping and I bought some baby clothes and other supplies. Kenyatta and I laughed at Randy because he fussed so much over the baby, changing her clothes and making sure he knew what to feed her.

10. I was interviewed by the police shortly after Steven died, and I am told that a police report says that I didn't feel comfortable around Randy and didn't want too much to do with him. This is incorrect. I saw Randy regularly at family gatherings and at my sister's (his mother's). When he worked at the Foot Locker, I saw him virtually every day. Sometimes he came to eat lunch or visit, other times he brought Kenyatta to work and either stopped in or brought Steven in to watch videos and play at the mall.

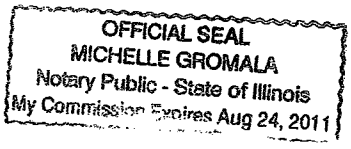
11. I was concerned about Randy's relationship with Kenyatta because she wasn't mature and didn't seem ready to settle down. Much later, my sister Linda (Randy's mother) told me that when Randy and Kenyatta lived with her, Kenyatta had pushed her and caused to hit her head and lose consciousness. Linda told me that she didn't want to make a report but insisted that Kenyatta move out. I believe that Linda told Randy's attorneys about this incident.

12. Initially, I didn't think that Randy's relationship with Kenyatta would last, but they were together about two years. In the last months, they had a place of their own, Kenyatta was trying to work, and Randy was very excited about the baby and being a parent.

13. When Randy was in jail I visited him and told him that I needed to know what happened to Steven. I told him words to the effect of, "you're not saying that you did it, you're not saying that Kenyatta did it, and this doesn't make any sense. Since you were there, you have to know what happened, and you need to tell us." He just kept saying that he didn't know. I told him that the family didn't see how he couldn't know since he was there.

14. At the same time, what the police were saying didn't make sense either. Quite apart from the fact that no one could see Randy beating a child, I wouldn't expect a person who had beaten a child to death to take the child in to work to show his or her boss that the child was sick or to drive the child across town to see the child's own doctors. Since none of this made sense, I felt that something was missing. I also thought that if our family had more money, perhaps we would have been able to find the answers.

I swear under penalty of perjury that the foregoing is true and correct.



Marlene Szafranski

Subscribed to and sworn before me this 20 day of Feb, 2008.

Notary Public in and for the State of Illinois

My commission expires Aug. 24, 2011

IN THE CIRCUIT COURT OF THE EIGHTEENTH JUDICIAL CIRCUIT, DU PAGE COUNTY, ILLINOIS

PEOPLE OF THE STATE OF ILLINOIS, Plaintiff	)	
v.	)	No. 02 CF 654
RANDY LIEBICH, Defendant	)	

AFFIDAVIT OF DEBRA MINUCCIANI

1. My name is Debra Minucciani, and I am Randy Liebich's aunt. My date of birth is October 24, 1953. My address is 8606 Alden Rd., Wonder Lake, IL.

2. Approximately three days before Steven went to the hospital, my niece, Denise Foster, and I visited Randy, Kenyatta, Steven, and newborn Angelique at their Willowbrook apartment.

3. When we arrived, Steven appeared to be whining and crying for no reason.

4. He was wearing a diaper and T-shirt, and I saw no marks on his body.

5. We offered Steven some of the food from McDonald's, but he refused to eat it.

6. I never saw Randy strike Steven in any way.

7. I do recall an occasion in which I was riding in the car with Kenyatta and Steven and Kenyatta slapped Steven on the leg because he was crying.

8. I am giving this affidavit of my own free will. No promises or threats were made to me in exchange for making the statements contained herein. If called to testify, I would testify consistent with this affidavit.

Date: 4/3/12 Signature: _____
Debra Minucciani

Subscribed and sworn to before me this 3rd day of April 2012

_____
Notary

"OFFICIAL SEAL"
Roger B. Lilly
Notary Public, State of Illinois
My Commission Expires 9/09/12

IN THE CIRCUIT COURT OF THE EIGHTEENTH JUDICIAL CIRCUIT, DU PAGE COUNTY, ILLINOIS

PEOPLE OF THE STATE OF ILLINOIS, Plaintiff	)	
v.	)	No. 02 CF 654
RANDY LIEBICH, Defendant	)	

AFFIDAVIT OF RICKY HOLMAN

I, RICKY HOLMAN, under oath and penalty of perjury, state as follows:

1. My name is Ricky Holman. I am an attorney licensed to practice law in the State of Illinois. My ARDC number is 6205161. My business address is DuPage County Public Defender's Office, 503 N. County Farm Rd., Wheaton, IL 60187. My business phone is (630) 407-8300.

2. In 2004, I was an Assistant Public Defender in DuPage County assigned to the defense team of Randy Liebich. My co-counsel was John Casey.

3. At no time during my representation do I remember advising, nor do I remember Mr. Casey advising, Mr. Liebich of his right to testify at trial.

4. I have reviewed my notes concerning the representation of Mr. Liebich and nowhere do they reflect that he was advised of his right to testify.

5. I am giving this affidavit of my own free will. No promises or threats were made to me in exchange for making the statements contained herein. If called to testify, I would testify consistent with this affidavit.

Date: 3-26-12

Signature: _____
Ricky Holman

Subscribed and sworn to before me this 26th day of March 2012

"OFFICIAL SEAL"
Roger B. Lilly
Notary Public, State of Illinois
My Commission Expires 9/09/12

_____
Notary

IN THE CIRCUIT COURT OF THE EIGHTEENTH JUDICIAL CIRCUIT, DU PAGE COUNTY, ILLINOIS

PEOPLE OF THE STATE OF ILLINOIS, Plaintiff	)	
v.	)	No. 02 CF 654
RANDY LIEBICH, Defendant	)	

AFFIDAVIT OF JOHN CASEY

I, JOHN CASEY, under oath and penalty of perjury, state as follows:

1. My name is John Casey. I am an attorney licensed to practice law in the State of Illinois. My ARDC number is 6229112. My business address is 1725 S. Naperville Rd., Suite 200, Wheaton, IL 60189. My business phone is (630) 290-4252.

2. In 2004, I was an Assistant Public Defender in DuPage County assigned to the defense team of Randy Liebich. My co-counsel was Ricky Holman.

3. At no time during my representation did I advise, nor do I remember Mr. Holman advising, Mr. Liebich of his right to testify at trial.

4. I am giving this affidavit of my own free will. No promises or threats were made to me in exchange for making the statements contained herein. If called to testify, I would testify consistent with this affidavit.

Date: 3/28/12 Signature: _____
John Casey

Subscribed and sworn to before me
this 28th day of March 2012

"OFFICIAL SEAL"
Roger B. Lilly
Notary Public, State of Illinois
My Commission Expires 9/09/12

_____
Notary

No. 2--04--1238

IN THE

APPELLATE COURT OF ILLINOIS

SECOND DISTRICT

THE PEOPLE OF THE STATE OF ILLINOIS, Plaintiff-Appellee, v. RANDY R. LIEBICH, Defendant-Appellant.	)	Appeal from the Circuit Court of Du Page County. 4000 S.A.D. No. 02--CF--654 Honorable Ann B. Jorgensen, Judge, Presiding.

Affirmed

RULE 23 ORDER

Defendant, Randy R. Liebich was indicted on four counts of first-degree murder (see 720 ILCS 5/9--1 (West 2002)), one of which was later nolle prossed, arising out of the death of Steven Quinn, who was two years old at the time. Following a bench trial, defendant was convicted and sentenced to 65 years' imprisonment. He now appeals, alleging the following errors: First, he contends that the trial court failed to conduct an adequate inquiry into his pro se claims of ineffective assistance of counsel. Second, he argues that he was not proven guilty beyond a reasonable doubt because the trial court relied on medical evidence that was incompetent, the trial court also made inconsistent findings, and the State failed to satisfy the mental-state element of the offense. Third, he alleges error in the admission of certain opinion testimony of two physicians who treated Steven. Fourth, he asserts that he was denied the effective assistance of counsel. For the reasons that follow, we affirm.

I. BACKGROUND

At the time of the incident that led to the death of Steven Quinn, defendant resided in an apartment in Willowbrook. Also residing in the apartment were Kenyatta Brown, defendant's girlfriend, and Angelique Liebich, the eleven-day-old child of defendant and Kenyatta. From time to time, Steven would also stay at the apartment. Steven was Kenyatta's child from a previous relationship. On February 8, 2004, Steven was left in the care of defendant while his mother was at work. Defendant and Kenyatta took Steven to Mount Sinai Hospital in Chicago later that day because Steven was exhibiting signs of certain medical problems, which we will discuss later. Subsequently, he was transferred to Rush Presbyterian Hospital due to the severity of a head injury he had suffered. Steven was eventually taken off a ventilator, and he died on February 11, 2002. The balance of what follows is taken from the evidence adduced at trial.

The first witness to testify for the State was Karen Clark, Kenyatta's mother. When Kenyatta was 15 years old, Kenyatta gave birth to Steven. He was born at Mount Sinai. Following Steven's birth, he and Kenyatta resided with Karen for about two or three months. Karen never observed Kenyatta exhibit any violence toward Steven. When Steven was about four months old, Karen took over his primary care, as Kenyatta was too young and immature to raise a child. Dorothy Herron, Steven's great aunt, also provided care. Steven would, however, stay with Kenyatta during weekends and sometimes for two weeks at a time. Karen never observed any injuries to Steven when he was returned to her care. On February 8, 2002, Karen was called to Mount Sinai Hospital, where she observed Steven. The child was lying in bed with his clothes off. Karen noted that his right testicle was swollen and red, but did not note any unusual marks on his thighs. Later, after Steven had been moved to Rush Presbyterian Hospital, Karen again observed Steven. She noted marks on his thighs

that were not visible at Mount Sinai and explained "They just started appearing." The marks were "like whip marks, red lines on his thighs, his foot, his ankle, neck, across his stomach and *** on his back and like pressure marks on his neck." During cross examination, Karen acknowledged that she had told a police officer that Kenyatta was an "excellent liar." Further, Karen had never seen defendant hit Steven.

The State next called Sadie Brown. Sadie ran a day-care business out of her home. The center had been in operation since 1996 and is licensed by the Department of Children and Family Services (DCFS). Sadie is Kenyatta's aunt. Sadie provided day care for Steven when he was in Karen Clark's care, as Karen had a job. During the time Sadie watched Steven, he appeared well nourished and she never had any reason to believe he was abused or neglected.

Kenyatta Brown testified that she was Steven's mother. She was 20 years old at the time of the trial. Steven was her child from a prior relationship. She met defendant when she was 16 years old and moved in with him sometime thereafter. Steven would either live with Kenyatta and defendant or stay with her mother or aunt. At the time of Steven's death, they were living in an apartment in Willowbrook. On January 27, 2002, Kenyatta gave birth to a daughter, Angelique. Defendant was the father. Steven stayed with Dorothy Herron while Kenyatta was in the hospital giving birth. Steven returned to the Willowbrook apartment on February 2, 2002. He appeared to be in fine condition. On the way back from picking up Steven, defendant and Kenyatta bought some PCP and stopped at a park to smoke it. The children were present. They then drove to Willowbrook.

At the time of Steven's death, Kenyatta worked for Car-Lene Research. As part of her job, she took surveys at the Yorktown Mall in Lombard. Defendant got Kenyatta the job through his aunt. Sometimes, Kenyatta would bring Steven to work with her, sometimes her aunt or mother

would watch Steven. Steven was never left home with defendant. Kenyatta testified that during the period from February 2, 2002, to February 6, 2002, Steven had a runny nose but did not require medical attention. Further, during that period, she did not discipline or hit Steven.

When at home, Steven would typically follow Kenyatta around the house. Kenyatta referred to him as her shadow. This behavior annoyed defendant. Sometimes, when defendant and Steven were alone in a room together, Kenyatta would hear a "hollow" sound and Steven would cry.

On February 7, 2002, Kenyatta was home with Steven and Angelique. She fed Angelique and prepared pork chops, mashed potatoes, and corn for dinner. Steven ate a few bites of pork and refused to eat anymore, so Kenyatta sent him to his room. Defendant came home at about 8:30 p.m., and Steven came out of the bedroom. Kenyatta testified that she had not struck Steven in any way prior to this time. Defendant had purchased some marijuana before coming home. Defendant sat at the table, rolling the marijuana into a "blunt." Kenyatta noted that defendant's eyes were glazed and his face was "droopy." She believed defendant was under the influence of heroin.

Kenyatta asked Steven if he was ready to finish his food. Steven replied "no," so she sent him back to his room. Steven was crying. Defendant went into Steven's room, and Kenyatta heard a "hollow" sound. When defendant emerged from the bedroom, Kenyatta asked him if he had hit Steven. Defendant stated that he had not. Kenyatta went into the bedroom to talk with and comfort Steven. Shortly thereafter, defendant returned to the bedroom, and he and Kenyatta smoked the "blunt." Steven was crying the whole time. After finishing the "blunt," the two sat on the floor to smoke a cigarette. Defendant said, "Shut the damn kid up." Kenyatta tried to speak to Steven, but he was not paying attention. She "muffed" him to get his attention. Kenyatta explained that to "muff" someone was to shove them with one's fingertips on the side of the head. She told Steven that

she was going to spank him if he did not stop crying. She testified that she was trying to scare him into stopping crying on his own. She asked defendant for his belt. She struck Steven three times on the diaper with the belt, which was folded over at the time. Steven was still crying, so Kenyatta removed Steven's diaper and told him she was going to spank him. She then slapped him on the butt with an open hand. She put the diaper back on and suggested that she and defendant just leave the room, which they did. Steven stopped crying.

Sometime later, Steven came out of the room and stated that he was ready to eat his food. Steven ate about half of his dinner. She let him watch television for a short time and then put him to bed. Steven was able to walk normally at this time, and he slept through the night.

The next morning, Kenyatta woke up at about 9:45 a.m. because she had to go to work. She changed and fed Angelique. At 10 a.m., she entered Steven's room, and he said, "Mom." He appeared fine. Kenyatta got dressed. Before leaving for work, she made a bowl of cereal for Steven. Kenyatta worked until 3:30 p.m, and then she ran some errands.

Kenyatta returned to the apartment at about 4:30. She set down some bags that she was carrying and asked defendant where Steven was. Defendant indicated that Steven was lying on the floor in the living room. Steven was, in fact, covered with a blanket and facing the wall. Kenyatta picked up Angelique, who had wet herself. Defendant started to get ready to go to work. Kenyatta approached Steven and noted that his breathing was not normal. When she rolled him over, she observed that his eyes were "just like a cold stare" and that he had vomit in his mouth. She asked Steven what was wrong, but he did not answer. She asked defendant how long Steven had been in this condition. Defendant said, "About an hour." Kenyatta saw four small red marks, "[a]bout the

size of fingers," on Steven's neck. Kenyatta asked defendant what had happened to Steven, and defendant said that he did not know.

Kenyatta stated that she needed to use the car to take Steven to the hospital and that she might be late picking defendant up from work. Defendant stated he would accompany her to the hospital. Kenyatta told defendant that he needed to go to work, but defendant was insistent. When Kenyatta picked up Steven, she was surprised that defendant had changed his diaper. Kenyatta, defendant, Steven, and Angelique embarked for the hospital. On the way to the hospital, they stopped by defendant's place of employment so defendant could tell his boss that he was not coming in to work. When defendant went inside to do so, he brought Steven with him, so his boss could see how sick Steven was. They then proceeded to Mount Sinai Hospital in Chicago. Mount Sinai was the hospital where Steven was born, and Kenyatta felt familiar with it. During the trip, Kenyatta again asked defendant what happened. Defendant stated that he had fed Steven and that Steven had choked on a hot dog. Defendant stuck his fingers into Steven's mouth. Steven bit defendant's finger. Defendant stated that he was going to hit Steven at this point, but Steven let go of his finger.

When they arrived at the emergency room, medical personnel immediately started to treat Steven. Subsequently, a doctor asked about a lump on Steven's head. There were also bruises on Steven's back. She noted the marks that she had observed earlier on Steven's neck had gotten bigger. Kenyatta asked defendant about the lump on Steven's head, and defendant stated he did not know how Steven got it. When a nurse took off Steven's diaper, Kenyatta noticed that one of his testicles was swollen. Steven was transferred to Rush Presbyterian Hospital for surgery. Additional bruise marks continued to appear after the transfer. Kenyatta spoke with law enforcement personnel from Du Page county during this time. She consented to a search of the apartment.

Kenyatta acknowledged that she would sometimes physically discipline Steven. This would involve striking him on the hand or butt. Steven never sustained an injury or required medical attention as a result of such discipline.

During cross-examination, Kenyatta agreed that Steven spent most of his life staying with relatives. She reiterated that February 8, 2002, was the only time Steven had been left alone with defendant. She acknowledged that there were times in the past when she had hit Steven with her hand on the butt, but denied ever hitting him on the head, striking him in the face, throwing him, picking him up by one arm or shaking him. Kenyatta also clarified that a "muff" was really just a touch with no real force behind it. Kenyatta acknowledged having pleaded guilty to a charge of prostitution in Winnebago County. She agreed that she had never seen defendant strike Steven.

John Georgopolous, a manager at the Patio Restaurant in Darien, also testified for the State. Georgopolous was working during the evening of February 8, 2002. Defendant had been working at the Patio for about two weeks at the time. Shortly after 5 p.m., defendant entered the restaurant holding a child. Defendant explained that his girlfriend's child was sick, and he had to take it to the hospital. Georgopolous stated that that was fine. The child appeared sick to Georgopolous, but he did not observe any marks on the child. Nicolas Brinias, another manager, later testified that on February 5, 2002, defendant had to leave work early because of a sick child. Also, on February 6, 2002, defendant missed work without explanation.

The State next called Dr. Paula Green. Green testified that she is a physician who specializes in emergency medicine. She was working in the emergency room at Mount Sinai on the day that Steven was brought there. A nurse asked Green to examine Steven. Green initially thought Steven was experiencing a febrile seizure, which can result from a high fever. Defendant and Kenyatta were

present. Green had a conversation with defendant regarding what had happened to Steven. Defendant stated that Steven had choked on a hot dog and had been in the state in which they brought him to the emergency room ever since. Defendant said that he did not pat Steven on the back when he choked and that Steven had not fallen or bumped his head. Green stated that during her first examination of Steven, she did not note any signs of trauma. A short time later, Green performed what she termed a "secondary survey." At this point, she noticed some marks. This indicated that Steven's condition was acute, that is, "something [that] has just occurred." Green called Kenyatta over and asked her if she had ever seen a mark that was on Steven's head before. Kenyatta turned to defendant and said, "[W]hat did you do to my baby." Steven was gazing to the left, which is indicative of a head injury, and he had a bruise on his lip. There was a red bruise on Steven's abdomen. The color signified an injury occurring within hours. There were also red marks on Steven's buttocks and back. Green examined a photograph of Steven that was taken after Steven left Mount Sinai. It showed bruising, particularly lateral marks on Steven's legs and feet, that had appeared subsequent to the time Green examined him. Green also noted "posturing" in Steven. "Posturing" is an involuntary flexion of the extremities and is a sign of severe brain injury. Dr. Boykin began assisting Green. A CAT scan revealed a "bleed," so a neurosurgeon, Dr. Munoz, was called. Munoz was working at Rush Presbyterian Hospital that night, so Steven was transported there.

During cross-examination, Green agreed that a mark on Steven's back could have been caused by a belt buckle. Similarly, the marks on Steven's buttocks could have been caused by someone striking him forcefully in that area. Also, the trauma team, which was called in after the results of the CAT scan were known, did note abdominal bruising while Steven was still at Mount Sinai. Green further agreed that lethargy, slurred speech, irritability, nausea, lack of appetite, and finicky eating

could be symptoms of a head injury (though during redirect, she stated that she had never seen a person with a head injury of the severity of Steven's walking, talking, eating, or playing). Further, a person who has suffered a head injury can remain conscious. Green found defendant's hot-dog story incredulous. She did, however, acknowledge that a layperson could confuse a choking incident with a seizure. Green testified that at the time Kenyatta asked defendant what he had done to Steven, Green had not yet indicated to Kenyatta that she suspected child abuse was involved. Green described defendant's demeanor as calm and respectful during the time he was at the emergency room, despite his being interrogated by doctors and nurses.

Dr. Tracy Boykin, the other emergency room physician who treated Steven at Mount Sinai, testified next for the State. Boykin stated that Mount Sinai was a level one trauma center, the "highest definition of a trauma center." She is board certified in emergency medicine. On the evening of February 8, 2002, Boykin was working at the Mount Sinai emergency room. She observed defendant carrying Steven into the hospital. Steven was limp. Boykin assumed Steven had had a febrile seizure. About three to five minutes later, someone came and told Boykin that Green needed her assistance. She went to Steven's bedside and immediately observed that he was posturing. Boykin was called to the CAT scan machine in the radiology department. The person that summoned her stated that the results of the CAT scan were really bad and that Steven's head was full of blood. Boykin then ran back to the emergency department and informed Green of Steven's condition.

Boykin then went to find Kenyatta and defendant. Boykin stated that she was angry at this time. She explained that she was angry because "Stevie had obviously suffered a severe brain injury secondary to trauma," and that "[i]t wasn't secondary to the hot dog that they said he choked on earlier." Defendant was "very calm, nonchalant," which further angered Boykin. Boykin told

defendant, "[I]t doesn't look like Stevie choked on a hot dog at all, it looks like [you] had been sitting at home beating him all day." Defendant did not say anything and just shrugged. Boykin told defendant that she was going to contact the police and DCFS. At this point, Steven was brought back to the emergency department, where he was intubated. The Chicago police arrived at about this time.

During cross-examination, Boykin testified that Steven could not have possibly eaten a hot dog after sustaining the injuries that she witnessed. When asked by defense counsel whether she would have wanted to know of events transpiring the night before Steven came into the emergency room, Boykin replied no because, had he sustained the injuries at that time, Steven would have been dead by the evening that she treated him.

The next witness called by the State was Marlene Szafranski. Szafranski was an office manager at Car-Lene Research, where Kenyatta worked. She is defendant's aunt. Kenyatta also worked for Car-Lene research. Szafranski testified that Kenyatta would sometimes bring Steven to work. On February 8, 2002, Kenyatta arrived to work at 10:40 a.m and worked until 3:40 p.m. She did not bring Steven with her on that day.

Letitia Beasley, was working as a nurse in the emergency room at the time Steven was brought in. She testified that she evaluated Steven when he first arrived there. Beasley noted that Steven was not breathing normally and that he was posturing. His eyes deviated to the left, and there was a bruise on the right side of his head. She rated Steven a four on the Glasgow Coma Scale. The Glasgow Coma Scale is a scale for recording a persons conscious state, which ranges from 3 (most likely dead) to 15 (normal and healthy). Steven's temperature was 94.7 degrees. There were bruises to Steven's scrotum. However, linear marks that were photographed after Steven had been moved

to Rush Presbyterian Hospital were not visible at the time Beasley performed her examination. Beasley observed Kenyatta speaking to defendant. Kenyatta was tearful and wanted to call her mother. Defendant told her not to do so. Defendant had a "very flat affect" and was not tearful.

On cross-examination, Beasley testified that defendant's demeanor was not paranoid, nervous, or fidgety. She also testified that she administered Ativan to Steven. Ativan is a medication used to treat seizures and to "break out *** [of] a dystonic reaction, a tightened, rigid reaction."

The next witness called by the State was Sergeant Michael Price. Price was involved in the investigation into the death of Steven beginning in the early morning of February 9, 2002. Price was dispatched to Rush Presbyterian to photograph Steven. He arrived about 2 a.m. After photographing Steven, Price went to defendant's apartment and took several photographs of it as well.

Lieutenant Edward Kunz of the Du Page County sheriff's office then testified that he arrived at Rush Presbyterian Hospital between 11:30 p.m. and midnight on February 8, 2002. Defendant was pacing a lot. At one point, defendant stated that he wanted a cigarette and to see his daughter. Kenyatta gave defendant a cigarette, and Kunz accompanied defendant outside. There, the two had a conversation. Kunz told defendant that he had a son about the same age as Steven. Kunz asked defendant if Steven's crying was aggravating to him. Defendant replied, "[Y]es, very." Kunz asked how defendant dealt with Steven's crying. Defendant stated that he normally does not have to deal with it, since Kenyatta does.

Defendant was granted permission to call Ruben Martinez out of order. Martinez testified that he had known defendant for about seven years. Martinez had witnessed Kenyatta strike Steven on one occasion. According to Martinez, Kenyatta struck Steven one time, back-handed, in the head. Martinez thought that this incident occurred at Kenyatta's aunt's house, but Martinez could not say

when. Cross-examination revealed that Martinez and defendant were good friends and that Martinez had visited defendant several times while defendant was in jail.

The State next called Tammy Smith. Smith is a pediatric and neonatal intensive care nurse who works at Rush Presbyterian. She was working on the evening of February 8, 2002, and was involved in Steven's treatment. She traveled to Mount Sinai by ambulance to transport Steven to Rush Presbyterian. A physician also accompanied her. Smith performed an initial evaluation of Steven. He was unresponsive, his hands and feet were cold, and his body temperature was low. There was a fresh bruise on the side of Steven's head, and there were also red bruises on his abdomen. Smith identified a bruise in a photograph of Steven that was not visible when she first examined him at Mount Sinai. They left Mount Sinai at 8:50 p.m and arrived at Rush Presbyterian at approximately 9 p.m. Steven went into surgery at about 10 p.m. and was in surgery for one and one-half hours.

Smith remained with Steven until 7:30 a.m. She was present when a police officer came and photographed Steven, which was approximately two hours after surgery. Smith noted additional marks that were present in the photographs that were not present when she examined Steven at Mount Sinai, and she told a physician of their existence because they were not there earlier. Also, bruises she had seen at Mount Sinai on Steven's thigh had become more defined in the interim. Similarly, marks on Steven's back had become more defined, and bruises on his abdomen were more pronounced. A mark under Steven's jaw, which Smith did not notice at Mount Sinai, had also appeared by this time. His abdomen became distended. Throughout the night, Steven's bruises darkened in color and were generally more pronounced. On cross-examination, Smith acknowledged that, other than two marks appearing on Steven's foot, she did not make any entries in Steven's chart

regarding bruises changing throughout the night. She explained that she was extremely busy during the latter portion of her shift.

Greg Figiel, who was previously employed with the Du Page County sheriff's office, testified next. Figiel was involved in the investigation of Steven's death. Figiel and Boris Vrbos of the Du Page County Children's Advocacy Center traveled to Rush Presbyterian at about 10:40 p.m. Figiel and Vrbos had a conversation with defendant at 11:20 p.m. in a conference room at the hospital. Tammy Smith brought defendant to the room. Defendant stated that he was watching Steven while Kenyatta was at work. Steven came to the kitchen table that morning and ate his cereal but did not drink his milk. Steven then played with the dog. Defendant took a nap in the afternoon. At about 3 p.m. he got up and made a hot dog for Steven. Defendant stated that Steven ate one-half of the hot dog. Steven also had a glass of water and, as he was drinking from the glass, he started to choke. He then vomited. Steven was breathing funny and wheezing, so, defendant said, he patted Steven on the back. Defendant asked Steven if he was all right. Steven nodded and said, "[Y]eah." Steven then laid down in the living room, moaned a few times, and fell asleep. According to defendant, Kenyatta arrived home at 4:30. She checked Steven and determined that he was not breathing properly, so they decided to take Steven to the hospital. Defendant also told Figiel that Steven was playing that day, but not as hard as he usually did.

Figiel and Vrbos went over defendant's story with defendant. Figiel asked what happened after Steven vomited. Defendant stated that he picked Steven up, and Steven went limp in his arms. Figiel asked what caused Steven to go limp and whether Steven had fallen or hit his head. Defendant answered that Steven had not fallen that day. They went over the story a third time. This time, defendant stated that everyone woke up at 10 a.m. He also stated that he had cut up the hot dog that

he later fed to Steven, that Steven had a glass of orange juice in addition to water, and he did not mention anything about Steven vomiting. This interview terminated at about 12:15 a.m. Figiel then spoke with Smith and was updated about Steven's condition.

Figiel met with defendant a second time at 1:05 a.m. This meeting took place in the same conference room as the previous interview, and defendant, Figiel and Vrbos were present. Figiel testified that he gave defendant Miranda warnings. At this point, someone knocked on the door and said that they had food for defendant. Defendant was given a taco salad, and the officers left him alone for about 10 minutes to eat it. They went over defendant's story again. Figiel told defendant that he did not believe that nothing happened in the apartment that day, given the nature of Steven's injuries. Defendant then told Figiel that after he had patted Steven on the back, Steven had fallen head-first to the floor. Defendant said that it was not a hard fall, however. Figiel stated that this is the first time defendant mentioned a fall.

At about 7 a.m., according to Figiel, he, defendant and Vrbos left the hospital in Figiel's unmarked car. Defendant voluntarily accompanied them. They stopped at McDonald's to get defendant orange juice and a potato cake and then at Burger King, because defendant also wanted a chicken sandwich. They then proceeded to the sheriff's office, arriving at 8 a.m. They placed defendant in an interview room. At about 8:40, defendant vomited in a waste paper basket. Figiel entered the room and asked defendant if he was all right. Defendant stated that he had a $20-a-day heroin habit.

Figiel went to Kenyatta's mother's house on February 19, 2002. The purpose of the visit was to listen in on a telephone conversation between Kenyatta and defendant. Prior to this time, Figiel had never mentioned to defendant that a clothes hanger may have been used as a weapon against

Steven. During the conversation, defendant told Kenyatta that she had hit Steven on the leg with a clothes hangar on February 7. She replied that she did not and that was the occasion on which she asked defendant for a belt to scare Steven. Defendant then stated, "Well, you came out of the closet with a hanger in your hand." This was the first time Figiel heard defendant say anything about a clothes hanger. Defendant called Kenyatta a second time that same day. Defendant asked if anyone was listening in on the call, which only lasted about 10 seconds. Defendant then called a third time a few minutes later. He told Kenyatta that he suspected that someone was listening in on their conversation, so Kenyatta should stay on the line and call him with a different phone at a different number. Kenyatta declined, and the two continued to speak. In all, defendant called Kenyatta nine times that day.

On cross-examination, Figiel agreed that, as he and defendant repeatedly went over the defendant's story, the substance of the story remained, for the most part, consistent. Figiel also acknowledged that defendant never told him that he struck Steven in any way. The court also permitted the State to elicit rebuttal testimony, out of order, from Figiel. The rebuttal testimony concerned the testimony of Martinez. Figiel testified that Martinez told him that Martinez had had a conversation with defendant wherein defendant stated that Kenyatta had hit Steven with a clothes hanger and that defendant had removed the hanger from the apartment as he knew police would be searching the apartment.

Thomas Filipiak, a Chicago police officer, testified that he was dispatched to Mount Sinai Hospital with regard to an injured child on February 8, 2002. Child abuse was suspected. He arrived at the emergency room around 6:30 p.m. Filipiak spoke with defendant. Defendant stated that Steven had choked on a hot dog and that he had slapped Steven in the back in an attempt to dislodge

it. Defendant denied striking Steven in the head. Defendant also replied "no" when asked if there was anyone else who could have harmed Steven. Filipiak described defendant's demeanor as aloof and nonchalant. As the evening progressed and it became clear that Steven's injuries were serious, defendant "became very very [sic] scared." Responding to questioning by defense counsel, Filipiak acknowledged that he did not know how much information defendant possessed regarding Steven's condition at the time he appeared nonchalant and aloof.

The State next called Dr. Paul Severin. Severin is a physician who is board certified in pediatrics and pediatric critical care, and the court recognized him as an expert in those areas. Severin provided treatment to Steven after Steven was transferred to Rush Presbyterian. Severin noted that Steven's Glasgow Coma Scale score was quite low and that Steven was posturing. Due to the interventions provided by the doctors at Mount Sinai, Steven's Glasgow Coma Scale score did improve. Severin explained that one of these procedures was to increase Steven's rate of breathing to relieve pressure in his head. Increasing breathing causes more oxygen and less carbon dioxide to be present in the blood. This, in turn, causes an organ, such as the brain, to shrink its blood vessels which, in turn, decreases blood flow and pressure in the organ. Severin further explained that such "auto-regulation" is not possible where the brain has been injured for a prolonged period of time. Thus, according to Severin, that Steven's brain was able to adjust indicated a more recent injury.

Severin testified that he observed numerous bruises and marks on Steven's back, scrotum, and thigh. The injuries appeared to be of approximately the same age. Steven's right pupil was larger than his left one, indicating that his brain was about to herniate. Severin later learned that the pressure in Steven's head measured 90 millimeters of mercury. The normal range is between 15 and 20 millimeters of mercury. Steven's abdomen was soft and there were bowel sounds which indicated

that no abdominal injury was likely. Due to elevated enzyme levels, Severin ordered additional testing. Steven's pancreatic enzyme level was in the thousands, while the normal level for a child of his age is 200.

Severin continued to provide care to Steven on February 9, 2002. He examined Steven that morning. Steven's abdomen appeared distended and he had "defuse redness around his belly button." That, coupled with elevated pancreatic enzyme, constitutes a "Cullen's sign." A "Cullen's sign" is an indication of hemorrhagic pancreatitis. At this point, Severin could no longer detect bowel sounds. Severin testified that a child that had sustained the type of injuries Steven had received would not be able to eat. At best, the child would throw up anything he ate. Severin opined that Steven's injuries resulted from nonaccidental, external trauma. Additionally, Severin stated that they occurred within four to six hours prior to Steven's admission to Mount Sinai. Further, Severin opined that Steven's injuries could not have been caused the night before, as there was "no way anybody would be able to survive a whole day later to get to the hospital." Additionally, choking on a hot dog was not consistent with Steven's condition.

Defense counsel then cross examined Severin. Severin acknowledged that he is not typically called upon to determine the timing of an injury. Further, Severin agreed that the history received from the family is a "critical factor" in determining the timing of an injury. The bruises Severin observed on February 9 could have occurred within the last 48 hours; however, bruising is difficult to time. Also, even if Steven had suffered no head injury, the injuries to his abdomen were potentially fatal. Severin further stated that the discovery by the pathologist, Dr. Mileusnic, of signs of healing would not change his opinion regarding the timing of the injuries due to his findings during his physical examination of Steven and the acuteness of Steven's injuries.

Janet David then testified that defendant came to the McDonald's where she worked between 2:30 p.m. and 3:30 p.m. on February 8, 2002, and "bummed" a cigarette from her. The McDonald's was across the street from defendant's apartment, and he only stayed a few minutes. The State then called Thomas Szalinski.

Szalinski testified that he had been a law enforcement officer for 19 1/2 years. He spoke with defendant in an interview room at the Du Page County sheriff's office. Defendant told Szalinski that, on the night of February 7, he and Kenyatta were in Steven's bedroom and Kenyatta disciplined Steven because he was crying. Defendant stated that Steven was wearing a diaper and Kenyatta struck him a few times on the buttocks with a belt. Kenyatta did not strike Steven on any other part of his body. Defendant stated that, in his opinion, Kenyatta was not hitting Steven hard enough, as the discipline was not having its intended effect. Defendant stated that he did not think Steven could feel it. Kenyatta then pulled the diaper down and struck Steven with her bare hand. At about midnight, defendant told Szalinski, Steven was crying and he and Kenyatta went into Steven's room. Kenyatta "popped" Steven in the head a few times. By "pop," defendant meant a sort of slap with the palm of the hand. Defendant denied striking Steven.

Defendant related that the next morning he and Kenyatta woke about 9:30 a.m. Neither disciplined Steven. Defendant fed Steven breakfast (cereal, juice, and water) after Kenyatta left for work at 10 a.m. Steven ate all of his cereal and drank his orange juice, but did not touch his water. Defendant then took Steven out of his high chair and placed him on the floor. Steven later played with the dog under the kitchen table. Szalinski asked if Steven could have been injured while playing with the dog, and defendant stated that he did not think that that was possible. Defendant changed Steven's diaper, and he did not observe any injuries while doing so. Later, defendant, Steven, and

Angelique watched the television together. Steven appeared dazed. Defendant stepped out to get a cigarette at about 1 p.m. while the children were asleep. Szalinski asked if anyone could have been in the apartment while defendant was gone. Defendant stated that he doubted so because he was only gone five minutes and the children were still sleeping when he returned. Szalinski testified that defendant told him that at about 3 p.m., he gave Steven a hot dog and some water. Steven choked on the water. Detective Raymond Bradford, who the State called next, then entered the room and took over the interview.

Bradford testified that he was an investigator for the Du Page State's Attorney's office. Bradford told defendant that he had just received information from the hospital that Steven likely would not survive his injuries. Bradford also stated that they were sure that Steven's injuries occurred while Steven had been in his care and that defendant was responsible for them. Defendant stated that he had not hurt Steven. Defendant was crying. Bradford stated that he understood that things like this could happen by accident and that defendant did not mean to hurt Steven. Defendant, resting his head in his hands, appeared to be listening very intently and nodded in agreement. Defendant then asked to use the telephone and to see Kenyatta. He was allowed to do so.

The State then called Robert Liebich. Robert is a police officer with the Roselle police department and is the cousin of defendant. At about 4:30 p.m. on February 14, he was on patrol and he received a dispatch that he had a family member waiting for him in the lobby of the police department. When he arrived there, he saw defendant and another cousin named Dion. The three went into a private room. Defendant asked if it was safe to talk in the room, meaning was the room "bugged." Robert said that it was not. Robert asked defendant about a LEADS message that had been sent out throughout the State that defendant was missing and suicidal. Defendant stated that

he had not been running and that he had in fact been speaking with the detectives investigating the case. Defendant showed Robert a small cut on the right index finger and told Robert that Steven had bitten him and drawn blood. Robert said, "So what?" Defendant explained that he was worried about his DNA being inside Steven's stomach. Defendant also told Robert that he saw Kenyatta hit Steven in the head four times because Steven would not stay in his room. The next morning Steven was lying in his bed awake, which is unusual because Steven usually gets right up. Later, when defendant was feeding Steven a hot dog, Steven started to choke. Defendant hit Steven on the back in an attempt to dislodge the food. When that did not work, defendant inserted his finger into Steven's mouth. It was at this point that Steven bit defendant. Defendant "smacked" Steven twice on the right side of his head so that he would let go. As defendant removed his fingers from Steven's mouth, Steven vomited. Steven was "kind of dizzy, wasn't walking right." Defendant asked Robert what Robert thought he should do. Robert advised him to tell the truth. During cross-examination, Robert acknowledged that he asked defendant if he would "swear on his father's grave" and then defendant looked him right in the eye and stated that he did not hit Steven that hard.

Dr. Darinka Mileusnic Polchan, a forensic pathologist, testified next for the State. Mileusnic explained that there was a difference of opinion in her field as to whether children sometimes experience a lucid interval following a traumatic head injury prior to becoming symptomatic. Studies that extrapolate from car accidents are inherently flawed, as the mechanism of injury is quite different from child abuse. Some studies purporting to demonstrate that no lucid interval can occur suffer from this or similar flaws. She has twice been hired to testify for defendants in criminal cases. Mileusnic performed a postmortem examination of Steven on February 12, 2002.

Her external examination revealed that Steven appeared to be a well-developed, well-nourished child. She noted a healing contusion on the right side of Steven's forehead. Dating the injury based on appearance was not possible. There was also a contusion on the nose and two on the left side of the head as well as one on the inner aspect of the ear. A healing abrasion was present on the back of Steven's head. A sixth injury--a bruise--existed on the right temple, and there was a seventh near the right ear. There were also bruises under Steven's chin. Mileusnic noted a superficial abrasion on Steven's neck that appeared to have been caused by his medical treatment, since it was much fresher than his other injuries. His right wrist was bruised. Mileusnic counted five bruises upon Steven's lower back. A photograph taken at Rush Presbyterian showed a cluster of bruises on Steven's back that were no longer present at the time of the autopsy. In fact, Mileusnic testified that "many of the injuries already healed and was [sic] obscured by the time that [she] saw Steven on the 12th." His scrotum was swollen.

Mileusnic documented three injuries to the right leg. One was a cluster of ten bruises, the second was a cluster of two larger bruises, and the third was another cluster of smaller bruises. She also found four injuries to the left leg. On the thigh was a cluster of four bruises, which Mileusnic listed as the 14th injury she found. Also, she found a bruise on the inner side of the left ankle, seven linear bruises on the foot, and a bruise on the top of the foot.

Mileusnic was shown a blue plastic clothes hanger and asked whether being whipped by it could have made the linear bruises found upon Steven (such as were found on his back, thigh and foot). She was of the opinion that it could. Further, the injuries would not be consistent with being struck with a belt. She did, however, note that there was one mark on Steven's buttock which could have been made by a belt. Outside of some preexisting marks on Steven's back and the abrasion

caused by medical treatment, the rest of the injuries appear to have occurred at approximately the same time.

Mileusnic also performed an internal examination of Steven. She noted a three inch hemorrhage into the soft tissue under the skin of the head. This is indicative of some sort of blunt force trauma. She also noted some residual subdural blood in the cranial cavity. On the left side of the head, which was not touched by the surgeon, she found a significant subdural hemorrhage. Additionally, a subarachnoid hemorrhage enveloped the brain (this comprised a thin layer). The right cerebral hemisphere showed a contusion, hemorrhage, and necrosis;[1] however, she stated she could not comment on the relevance of these findings due to the surgery performed on the right side of Steven's brain. She found blood descending "passively along the dura[2] downwards," which is consistent with blunt force trauma to the head and a severe brain injury. An examination of the eyes revealed an accumulation of blood, which is another sign of severe head trauma.

An internal examination of Steven's body cavity revealed peritonitis, a form of inflamation. Fibrin deposits, which are a sign of healing, were present over the proximal bowel or jejunum. The first segment of the jejunum was perforated and appeared hemorrhagic and necrotic. There was blood in the bowel, which was in the process of dying. There was a hemorrhage around the head of the pancreas. Mileusnic opined that these injuries were also the result of blunt force trauma. Also,

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[1] "Necrosis" is defined as the "[p]athological death of one or more cells, or of a portion of tissue or organ, resulting from irreversible damage." Stedman's Medical Dictionary 1185 (27th ed. 2000).

[2] "Dura," short for "dura mater," refers to "a tough, fibrous membrane forming the outer covering of the cental nervous system." Stedman's Medical Dictionary 548 (27th ed. 2000).

"there was a collection of blood under the capsul [sic] of the liver that corresponded to the same level of injury." The retroperitoneum (which is the soft tissue behind the bowel that "sort of interfaces between the abdomen and the back") had blood tracking down it. In fact, what had been perceived as bruising to Steven's testicle was actually from blood tracking all the way down to his scrotum from this area. Mileusnic examined the testes and found no blunt injury to them during the autopsy; however, she later stated that a photograph of Steven nearer to the time of his admission showed "swelling, redness and a linear abrasion on the scrotum." Furthermore, that there was hemorrhaging on both the right and left side of Steven's body indicated blunt force trauma to both sides. Typically, the bowel is "pink/tan and glistening." Steven's bowel was dark red in areas indicating hemorrhaging, and yellow, which shows necrosis.

In cases of suspected child abuse, the usual practice is to make incisions through the skin of the upper and lower extremities to look for bruises that are deep and not visible superficially. Such an examination revealed three areas of hemorrhaging in the arms and three in the legs and feet. Mileusnic also found evidence of hemorrhaging in the back and buttocks through a similar examination. These injuries are also indicative of blunt force trauma. Mileusnic agreed that injuries like these would not be caused by "normal corporal punishment," but would require "something much more forceful." Again, Mileusnic was of the opinion that all of these injuries occurred at about the same time and that none were related to medical treatment Steven received. Moreover, these injuries were consistent with child abuse and with Steven being beaten to death. Finally, Mileusnic opined that Steven's death was caused by blunt force trauma.

On cross-examination, defense counsel asked Mileusnic about an article upon which she was working. One of the premises of the article concerned lucid intervals in cases of child abuse. Head

trauma does not have to manifest immediately. In the case of child abuse, a child may not become significantly symptomatic for up to 48 hours after the injury. The doctor also authored another case study where a lucid interval occurred where a child died a "couple of days" following head trauma. Mileusnic also explained that there was a difference of opinion in her field regarding the existence of lucid intervals. One school holds that such intervals never occur; the other, less well defined, questions the first school's tenet. The first school normally holds that the last person with a child when symptoms of a head injury manifest is the perpetrator of abuse. Mileusnic finds this proposition problematic and believes that knowing a history from a nonmedical source is important. She expressly was not giving an opinion regarding whether Steven experienced a lucid interval in this case.

Mileusnic agreed that timing is an issue she frequently confronts as a forensic pathologist. Generally, she gives times in terms of days and would never give an interval for the timing of an injury such as four to six hours. Moreover, giving an interval requires looking at microscopic evidence.

Mileusnic did perform a microscopic examination during her autopsy of Steven. Establishing when the abdominal injuries occurred was problematic due to the degree of necrosis. However, she testified that she was "more comfortable" with regard to the head injury as that area of the body is "kind of sequestered" and "not exposed to a lot of decay." Mileusnic opined that Steven's head injuries were inflicted approximately five days, plus or minus a day, from the time of his death. She later clarified that the day of Steven's death would count as a day, so, February 7, plus or minus a day, was, in her opinion, when the injuries occurred. Mileusnic believed the injuries "could have occurred on the 8th, but they could have easily occurred before that," even as early as the 5th. She further explained that abdominal injuries often were slow to manifest. The symptoms are not instantaneous

and, though there would be pain, a two year old would likely have difficulty expressing what he was going through. Necrosis, bleeding, and pancreatitis would follow "hours after the injury." Mileusnic saw signs of healing in Steven. One would usually see a fibrin layer[3] within the first day. However, the peritoneum ("a thin layer of irregular connective tissue[] that lines the abdominal cavity" Stedman's Medical Dictionary 1353 (27th ed. 2000)) responds to an injury differently. In Steven's head, Mileusnic found mononuclear cell fibrin, which is a "third kind of level of defense that happens in the body." Typically, these occur five to seven days after an injury.

Mileusnic agreed that finicky eating, lack of appetite, inconsolable crying for no apparent reason, lethargy, and excessive sleeping could all be symptoms of the injuries Steven sustained. Head injuries could result in seizures, and clenching one's jaw is a sign of a seizure. A seizure could be mistaken for choking. However, during redirect, she explained that it would be difficult for a person to force a finger between the teeth of a child clenching his or her jaw during a seizure. Furthermore, such a child would be experiencing severe pain and would be vomiting within hours. She would not expect a child with these injuries to eat breakfast if he had sustained the injuries the night before.

The final witness to testify for the State was Dr. Lorenzo Munoz. Munoz is board certified in neurosurgery. Munoz was on duty on February 8, 2002, when Steven was transferred to Rush Presbyterian from Mount Sinai. Munoz was informed that Steven was "neurologically very sick", that he had required intubation, and that a CT scan[4] had revealed a lot of blood in his head. Munoz first

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[3] Wikipedia defines "fibrin" as "protein involved in the clotting of blood. It is a fibrillar protein that is polymerised to form a 'mesh' that forms a hemostatic plug or clot (in conjunction with platelets) over a wound site." See http://en.wikipedia.org/wiki/Fibrin.

[4] Munoz explained that " 'CT' stands for computerized tomogram."

saw Steven at around 8 p.m. or 9 p.m. He conducted a gross visual examination of Steven's head and observed bruises. Steven was nonresponsive but not medically paralyzed. Munoz stated that it was obvious that Steven had not been given medication to relax his muscles because he was posturing. Posturing, according to Munoz, "is a very ominous sign that there's something very bad going on with the central nervous system." The CT scan revealed what Munoz believed was a fresh subdural hematoma. It also showed a "rather diffused and very severe subarachnoid hematoma." "Diffused" means not localized, which is significant because it "speaks of a trauma to the whole brain." Indeed, there were other indications of generalized swelling and injury to the entire brain. Munoz stated that Steven's brain was "extremely swollen."

After examining Steven, Munoz decided that surgery was the best course. Munoz testified that because Steven's pupils were reactive, he felt he had to "give this kid a chance." The goal was to decompress a blood clot and relieve the pressure on Steven's brain. The operation commenced at approximately 10 p.m. Munoz opened Steven's skull. The dura (the outermost layer covering the brain) was taught. Upon opening the dura, Munoz observed a subdural hematoma. Steven's brain was "red with a massive amount of subarachnoid blood" and swollen. The brain started to herniate, that is, to come out of the opening in the skull that Munoz had made. Munoz stated that herniation is also an ominous sign "because that tells you the brain is so swollen *** that it's trying to find a way out to decompress itself." He continued, "The problem with that is that as the brain starts coming out there is not much you can do about it at that point anymore." The blood Munoz observed in Steven's head was bright red, as opposed to "currant, jelly color" or "motor oil color." This coloration indicated that the blood constituted a fresh clot. Munoz characterized the amount of blood

as "massive." In Munoz's experience, "the longer removed the injury that brings about the bleeding in your brain is, the darker the blood looks."

Munoz testified that at this point, there was not much he could do except evacuate the subdural blood and close up Steven's skull. Subarachnoid blood is too intimately associated with the brain to be removed. Munoz also installed a "intracranial pressure monitor bolt," which is a device that measures pressure inside the skull. The device gave a reading of over 90, which is incompatible with life. After surgery, Munoz again evaluated Steven. His eyes were fixed and were no longer reactive to light (Munoz later explained that had Steven's eyes been fixed prior to surgery, Munoz likely would have regarded surgery as futile). Subsequently, Steven was declared brain dead by the pediatric staff of the intensive care unit. He was then taken off of his ventilator and died.

In the course of treating Steven, Munoz was provided a history that had been taken at Mount Sinai regarding Steven choking on a hot dog. Munoz testified that that history was "impossible." He also opined that Steven's injuries were not accidental and that they occurred no more than six hours prior to the time Steven arrived at Mount Sinai. Munoz further opined that it would have been impossible for Steven's injuries to have occurred during the evening of the day before he was brought in to Mount Sinai. Additionally, Munoz opined that Steven could not have walked, talked, eaten, or drunk anything after sustaining these injuries. He later testified that it was not possible that Steven had a lucid interval after sustaining his head injury.

When cross examined, Munoz acknowledged that he had only been board certified for nine months and that this was the first criminal case in which he had testified. However, he had given his opinion in approximately six depositions regarding the timing of an injury. Munoz agreed that history played a large role in determining when an injury occurred. By "history," he meant information

gathered by hospital staff from family members and eyewitnesses to the injury. However, Munoz testified that, in the instant case, he really did not have to look at any history because of the appearance of Steven's brain. The history that Munoz did consider was that Steven was "doing okay" before Kenyatta left for work and that he was not when she returned from work. Furthermore, Munoz has contacted DCFS "hundreds" of times regarding suspected child abuse, and on each occasion, DCFS personnel have asked him to give an opinion regarding the timing of the injury. When asked whether he "extrapolate[d] a lot of [his] opinions on what happened to Steven from car accidents," Munoz replied, "No[,] I extrapolate my opinion from having seen hundreds of children like Steve." However, he did disagree when asked whether it was necessarily scientifically invalid to equate head injuries resulting from motor vehicle accidents with head injuries caused by abuse. Later, he added that the majority of cases of pediatric trauma he has seen arise out of automobile accidents rather than child abuse, but both involve similar mechanisms of injury. Finally, Munoz agreed that a seizure could be a symptom of severe head trauma and that nonmedical personnel could mistake a seizure for choking. After Munoz's testimony concluded, the State offered its exhibits and rested.

The first witness presented by defendant (except for those taken out of order for scheduling reasons) was Officer Figiel, who had testified earlier for the State. Figiel sat in on an interview of Kenyatta Brown on February 15, 2002. Three other individuals were also present. Figiel testified, and the State stipulated, to several impeaching statements. Additionally, Figiel testified that he spoke with Dr. Severin at Rush Presbyterian during the late evening of February 8, 2002. Severin told Figiel that Steven's injuries were 24 to 48 hours old. However, Severin also characterized this estimate as a guess.

Defendant then called Joseph DelGiudice, a detective from the Du Page County sheriff's office. DelGiudice interviewed Kenyatta on February 9, 2002, at about 1:10 a.m. DelGiudice also testified regarding several impeaching statements. DelGiudice spoke with Dr. Green. Green pointed out a "knot" on Steven's head, and Kenyatta stated that Steven had had knots on his head since birth. However, after Green showed Kenyatta the "knot," Kenyatta looked at defendant and asked him what he had done. DelGiudice spoke with Nurse Beasley that day as well. Beasley indicated that Kenyatta's behavior was strange and that she believed Kenyatta "may have been in shock and did not realize the magnitude of the situation." She characterized defendant's behavior as, inter alia, "defensive."

Defendant then called Sergeant Michael Price, also of the Du Page County sheriff's office. In the early morning of February 9, 2002, Price went to the apartment of Kenyatta and defendant. He observed a vomit-stained pillow. Price took a picture of a closet in the bedroom. He also visited the apartment on February 10, 2002, and took more photographs, primarily of the closet area. Several items of clothing were missing. There were two additional empty hangers in the closet at this time.

Denise Foster next testified on defendant's behalf. Foster testified that she is defendant's sister and had known defendant for 24 years. She stated that she had seen Kenyatta strike Steven on two occasions. One occasion occurred at Foster's home in the December or January preceding Steven's death. Kenyatta and Steven were in the bathroom. Foster heard Kenyatta spanking Steven and heard Steven crying. According to Foster, Kenyatta then left Steven on the toilet for about 20 minutes. Foster also witnessed another, earlier incident. Kenyatta slapped Steven, back-handed, at least three times because Steven was crying. During cross-examination, she denied that she did not like

Kenyatta. She later clarified that she got along with Kenyatta fine, but did not like the way Kenyatta treated Steven.

Defendant then called Frank Belpedio. Belpedio is defendant's cousin. He testified that he observed Kenyatta strike Steven on three occasions. The first time was in June or July of 2001. Belpedio was driving a car; defendant was in the front seat, and Kenyatta and Steven occupied the back seat. Steven was crying, and Belpedio heard a loud slap. He then looked in his rear-view mirror and saw Kenyatta slap Steven in the cheek and shoulder. He saw Kenyatta strike Steven a second time at Karen Clark's apartment. The second incident occurred a few weeks after the first one. Belpedio was sitting on the couch with Steven and three other children. Steven started to cry. Kenyatta came into the room, told him to be quiet, and slapped him twice. One slap landed on Steven's head and the other on his back. The slaps were delivered with an open hand. Steven fell off of the couch. The third incident happened sometime after Angelique was born, about three or four days before defendant was arrested (Belpedio also stated it occurred in the summer of 2001). Belpedio testified that Steven was crying. Kenyatta walked over to him, shook him causing his head to flail about, and slapped him. Belpedio heard defendant ask Kenyatta, "what are you doing that for?". She slapped Steven "[o]pen hand[ed] right in the face." On cross-examination, Belpedio acknowledged that he and defendant would "hang out" together socially and that they were "tight." Further, Belpedio added that the third incident left a half-inch scratch upon Steven's face.

Defendant next called Karen Clark--Kenyatta's mother. Clark testified that Steven spent half his life living with her and half of it living with his great aunt, Dorothy Herron. He "never really permanently" lived with Kenyatta. Clark stated that she had never seen Kenyatta slap Steven in the face. However, she acknowledged telling a detective that "Kenyatta slapped Steven in the face a few

times in the past due to his whining and crying." She later clarified that she did not actually see Kenyatta do this.

Defendant then recalled Detective DelGiudice. He testified that he spoke with Clark on February 8, 2002 at Rush Presbyterian at about 11:30 p.m. In that conversation, Clark told DelGiudice that "Kenyatta slapped Steven in the face a few times in the past due to his whining and crying." The State also inquired of DelGiudice at this time regarding a statement made by Denise Foster. Foster told him that she did not like Kenyatta and did not think that Kenyatta was right for her brother. When asked to explain herself, Foster declined to discuss the matter further.

Crystal Zeis (formerly Crystal Holdmann) testified next. She had known defendant for five or six years, and she also knew Steven and Kenyatta. Zeis and her boyfriend lived with Kenyatta and defendant for a while. Zeis saw Kenyatta strike Steven on several occasions. One time, Kenyatta told Steven to throw his diaper away. Steven threw it in the kitchen sink. Kenyatta grabbed him by the arm and threw him out of the kitchen. After cleaning up the mess, Kenyatta shoved Steven into the bedroom and told him to take a nap. Zeis stated that she did not note any injuries. Zeis added that, though she would not say that Kenyatta struck Steven on a daily basis, it did happen on a regular basis or "quite often." "Every time he did something wrong," Zeis testified, "he ended up getting hit for it." Often, this involved an open-handed blow to the side or back of the head. Zeis never saw Kenyatta use an object to discipline Steven, but she did observe Kenyatta strike Steven on various parts of his body including his back, butt, arms, legs, and "wherever [she] could reach at the time."

On another occasion, Zeis stated, Kenyatta flung Steven out of the kitchen after he spilled some juice. Zeis described yet another incident where Kenyatta threw a fan which landed about two or three feet from Zeis's daughter. Zeis never saw Randy strike Steven. Steven "always had bruises

on him." Zeis once saw a mark on Steven's back that looked like it was made by a ruler or some similar object. On a few occasions, Zeis saw defendant attempt to intervene to try to stop Kenyatta "when she got out of hand," but Kenyatta would yell at him and tell him that he was not Steven's father so he should stay out of it. In response to questioning by the State, Zeis acknowledged that she had dated defendant for a short time. She further acknowledged that she did not like Kenyatta.

The last witness to testify for defendant was Dr. Shaku Teas. The trial court recognized Teas as an expert in the area of forensic pathology and child abuse. Teas had previously testified for the State "[p]robably hundreds of times" and on behalf of defendants only about 20 times. Teas reviewed Mileusnic's autopsy report, photographs, and histology[5] slides. She also examined the records from Mount Sinai and Rush Presbyterian as well as DCFS records, police reports, and witness's statements. Teas spoke with Mileusnic prior to her testifying. Regarding the cause of Steven's death, Teas agreed with Mileusnic's opinion that he died as a result of multiple blunt-trauma injuries. Further, she opined that a combination of head and abdominal injuries caused Steven's death.

Teas testified that the type of injury Steven sustained to his abdomen was caused by punching, hitting, kicking, or some sort of crushing mechanism. A person sustaining such an injury, she explained, might experience pain for a while and then be fine for a while. Then, as the ulceration and peritonitis "sets up," pain would become more general and the person might become septic and lose consciousness. Further, a person with such an injury would be able to eat, initially. Injuries like these are not necessarily painful; a person might only feel a little discomfort. She later added that it takes time for a perforation to occur and peritonitis to set in. There is usually a delay between the injury

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[5] "Histology *** is the study of tissue sectioned as a thin slice, using a microtome. It can be described as microscopic anatomy." See http://en.wikipedia.org/wiki/Histology.

and the onset of symptoms. Further, Teas felt that abdominal injuries were easier to time than head injuries.

Regarding the head trauma, Teas stated that she reviewed the CT scan from Mount Sinai. There was no mention of "midline shift." "Midline shift" refers to a state where "there is a mass lesion on that side of the brain and so it is shifting the brain to the opposite side," and its absence indicated that there was not "just one localized area." Additionally, histology slides of the testicles revealed no evidence of trauma. Teas explained that the discoloration in the scrotum was likely due to blood trickling down to that area from the abdomen.

Teas testified that, as a pathologist, assessing the timing of injuries is something she is familiar with doing. Timing is "an integral part of pathology." The best way, according to Teas, to address timing is histology; however, she did also state that it is "imprecise." Thus, estimates of the timing of an injury are usually given in days. She testified that she would never give, nor has she ever seen given in any textbook or paper, an estimate in terms of hours. She did believe that all of Steven's injuries occurred within a 24-hour period.

Teas found evidence of acute inflammatory cells in Steven's gastrointestinal tract. These typically appear about 12 to 24 hours after an injury. She did not recall seeing any in the subdural hematoma, however. On about the second day following an injury, mononuclear cells (also called lymphocytes) start to appear and grow in numbers. On the third day, and possibly on the second, spindle-shaped cells called fibroblasts begin to lay down collagen. As time passes, Teas explained, layers of fibroblasts increase; hence, the more that are present, the older the injury. Also, at about the same time, new capillaries form, making the surface of the area very granular. In Steven's case, Teas saw early granulation of tissue and several layers of fibroblasts. After between four and five

days, there are three to five layers of fibroblasts, and at 7 to 10 days, the capillaries become very prominent. "In this case," said Teas, "you could almost see the capillaries."

Teas opined that Steven's injuries were about five days old. She said they could have been six days old and it was possible that they were only four days old. However, in her opinion, due to the amount of healing that had taken place, they could not have been less than four days old. Steven was pronounced dead on February 11, 2002, at about 12:30 p.m. Discounting the fact that Steven was on a respirator for a while, which may have slowed healing, Steven's injuries would have occurred, according to Teas, "on or around February 6." Teas testified that she also found evidence of injuries that could have been a month old. In a histological slide, she found a new blood vessel forming in the dura, which requires four to five days after an injury to occur. Teas also stated that it was more likely that Steven's injuries were seven days old as opposed to three days old. Later, Teas testified that children heal a bit faster than adults, but that would be offset by the respirator.

Teas further testified that Steven's purported choking on a hotdog could be secondary to either his abdominal injuries or his head injuries. She also stated that a seizure resulting from a head injury could result in clenching of the teeth and would involve shaking in other areas of the body. Furthermore, timing an injury based on bruising is problematic, as "each person responds differently." A person who suffered the type of head injury experienced by Steven could remain conscious. Teas stated that she reviewed the postoperative reports of Munoz, and they did not change her opinion that Steven could have remained conscious. That Steven's intercranial pressure was 90 and rapidly progressing (normal is 10 to 20) was consistent with an injury occurring days before, as the brain does not necessarily start to swell at the time of an injury.

Also, having a history of the injury is important in determining timing. Furthermore, extrapolating data and opinions from automobile accidents and applying it to a nonaccidental injury is problematic. Teas explained that she typically sees patterns of injuries that are "a little different" in car accidents and instances of child abuse. When asked again about timing, Teas stated, "The subdural--I can't say anything about the other head injury, but the subdural and abdominal were consistent with five days." These injuries, she added, were the cause of death. Red lines on Steven's foot noted in a Rush Presbyterian record from 3:30 a.m. on February 9, according to Teas, could have been caused by tubes from a blood pressure cuff on Steven's leg.

During cross-examination, Teas explained that calcification (which she observed in the dura) could indicate that the injury is older than the presence of fibroblasts would indicate. It could also be evidence of a second, earlier injury. Teas acknowledged that Mileusnic was of the opinion that the calcification was an artifact of the surgery on the right side of Steven's head. She further acknowledged that her report stated that Steven was transferred to Rush Presbyterian on February 9, 2002, at 11 a.m., which is incorrect as to both date and time. Teas did not differentiate between who reported what injury in her report; that is, the report does not specify that certain injuries that manifested themselves later were not apparent at the time Steven was brought in to Mount Sinai. Later, she stated she "sort of *** tried to separate" what was observed at Rush Presbyterian. She also explained that there were some discrepancies in the records of the anesthesiologist and the surgeon. However, these discrepancies did not affect her opinions. According to Teas, posturing is a type of a seizure. Following Teas testimony, defendant rested.

The trial count found defendant guilty of first-degree murder (see 720 ILCS 5/9--1 (West 2002)). The trial court also found that Steven was under the age of 12, but declined to find that the

murder was accompanied by exceptionally brutal and heinous behavior indicative of wanton cruelty" (see 730 ILCS 5/5--5--3.2 (West 2002)). In so ruling, it made the following findings. Steven was born on April 17, 1999 and was a normal, healthy child. He had been raised primarily by his grandmother, Karen Clark, and his aunts, Dorothy Herron and Sadie Brown. None of these women had ever seen any marks or injuries on Steven indicative of child abuse after Steven had returned from being with Kenyatta. The court found them "extremely credible." It also found that they clearly loved Steven and "would have done everything in their power to keep him from Kenyatta had they thought for a moment that he was in danger."

The cause of Steven's death was blunt trauma to the head and abdomen. The court stated that the sole issue was who caused that trauma. The court explained that the defense's theory was that Kenyatta beat Steven so severely that he was mortally wounded early in the week of February 2, 2002, "perhaps as early as the 4th, 5th, or 6th." Then, Steven experienced a "lucid interval," which lapsed about an hour before Kenyatta returned home on February 8, 2002. Kenyatta's purported history of abusing Steven was presented through the testimony of Zeis, Martinez and Beldepio. However, the trial court expressly found that two of the witnesses (Zeis and Belpedio) were not credible. The court reiterated that Clark, Brown, and Herron never observed an signs of abuse after Steven was with Kenyatta. The pathologists that defendant offered in support of his position, dated Steven's injuries, based on the rate of healing, as occurring between the fourth and eighth or between the fifth and ninth of February. The court observed that these estimates, while "hardly an exact measurement," were further complicated by other factors that affected the rate of healing such as oxygenation, Steven's youth, and the use of a respirator. It is clear that the court did not find the opinions of the pathologists particularly useful and did not attribute great weight to them. In sum,

the trial court found that "[t]he evidence *** does not support any allegation that Kenyatta Brown chronically abused Steven Quinn."

The State's theory, on the other hand, was that Steven was in the sole care of defendant starting at about 10 a.m. on February 8, 2002. When Kenyatta left for work at that time, Steven was healthy; when she returned at 4 p.m., Steven was nonresponsive, " 'breathing funny,' " eyes locked in a cold stare, with vomit in his mouth.

The trial court found the testimony of the medical personnel who treated Steven most compelling. By 6:18 p.m. on February 8, 2002, Steven was posturing. At this time, a treating nurse did not see any marks on Steven's legs or ankles. Nurse Smith at Rush Presbyterian later observed fresh red bruises and marks on Steven's legs. At about 11:30 p.m., welts were noted on his left foot. Moreover, the marks were changing and becoming more defined. Dr. Green observed red marks on his head. That they were red, she testified, indicated that they were fresh. Dr. Boykin testified that Steven could not have eaten a hot dog, as described by defendant, after he sustained these injuries. Because Steven could not have eaten after sustaining such injuries, he had to have sustained them after he last ate. Further, he could not have sustained these severe injuries on February 7, for he would not have survived until the time he was brought to Mount Sinai. At Rush Presbyterian, injuries that were not visible at Mount Sinai continued to appear. Steven's abdomen was not distended until the following morning. Dr. Severin observed that Steven's autoregulation system was intact during the evening of February 8, indicating that the brain injury was recent rather than days old. Amylase and lipase levels had doubled between the time blood tests were taken at Mount Sinai and later at Rush Presbyterian. The only explanation for such an increase, found the trial court, was the recency

of the injury. A Cullen's sign appeared on the morning of February 9. Severin stated that these appear relatively quickly after a severe injury to the pancreas.

Dr. Munoz observed bright red blood, which also indicated a recent injury. He added that it was impossible for the injury to have been sustained on the previous evening, for Steven would have been unable to walk, talk, eat, or drink. A head injury of this magnitude would preclude a lucid interval.

The court noted that the sudden appearance of all of Steven's injuries on the evening of February 8 was inconsistent with a chronic pattern of abuse by Kenyatta. One would expect to see injuries of a different age. The court also noted that while defendant had failed to change Angelique's diaper, he did change Steven's. It also relied upon Robert Liebich's testimony that defendant told him that he "didn't hit the kid that hard," noting that this was an admission that he did, in fact, hit Steven. The court further noted defendant's changing demeanor as the night progressed and the extent of Steven's injuries became clear.

On August 27, 2004, the trial court heard defendant's motion for a new trial. At the beginning of that proceeding, defense counsel informed the trial court that defendant was in the process of attempting to hire private counsel because he had raised issues pertaining to the ineffective assistance of counsel. Defense counsel also told the trial court that the attorneys defendant contacted would not proceed unless they were either retained by defendant or appointed by the court. The trial court, after asking the State for input, simply stated:

"The Court has appointed the public defender to represent you, and if the public defender--If you chose to to [sic]dismiss the public defender, you have the right to hire your

own attorney, but the Court will not give you an additional attorney because you do not wish to proceed today."

Defendant responded, "Okay." The court then turned to the motion for a new trial, which it ultimately denied. In the course of arguing the motion, defense counsel, at defendant's request, raised the following issues regarding ineffective assistance of counsel: (1) that counsel should have brought in additional witnesses to impeach Kenyatta regarding her purported abuse of Steven; (2) that counsel should have had a witness testify that defendant had changed Steven's diapers in the past; (3) that counsel should have brought in additional witnesses to testify regarding other inconsistencies in Kenyatta's testimony; (4) that other detectives should have been called to testify--in addition to Figiel--regarding a "hollow noise" Kenyatta stated she heard while defendant was in the bedroom with Steven; (4) that counsel should have conducted more thorough cross-examinations; (5) that counsel failed to present evidence that no clothes hangers were found in the garbage on February 9, 2002. Counsel then stated, "I believe that covers everything that Mr Liebich wanted us to bring up for ineffective assistance." Later during the hearing, when defense counsel was discussing the testimony of Dr. Severin, defendant interjected, "When they said the bruising was zero to 24 to 48, which was not said. That was never cleared up."

Also, on August 27, 2004, the State called to the trial court's attention the fact that Dr. Teas had sent the court a letter. Copies were also sent to the State and defense counsel. The State asked that the trial court not view it, as it was an "improper ex parte communication." Defense counsel agreed. The court impounded the letter and made it part of the record. On September 9, 2004, the trial court sentenced defendant to 65 years' imprisonment.

Defendant--by counsel--filed a motion to reconsider sentence. He also filed a pro se motion alleging the ineffective assistance of counsel. The latter motion was filed on November 8, 2004. On November 10, the court asked defendant what he was asking for in his motion. Defendant replied that he wanted new counsel and "hopefully" a new trial. In a hearing on November 29, the trial court stated to defendant, "[Y]ou've indicated to your attorney that you wish to make your argument in writing, and you have a copy?" Defendant replied that the copy was illegible. The trial court then made arrangements for defense counsel to make and file a legible copy.

On December 7, 2004, the final hearing in this cause was held. The trial court asked defendant about the papers he had filed and whether they constituted separate motions. Defendant explained that one was a motion, and two packets were arguments in support of that motion. The following colloquy then ensued between defendant and the trial judge:

"THE COURT: What is your position then

MR. LIEBICH: I just feel that I didn't receive a fair trial because my attorneys weren't fully prepared to try this case. And I pretty much stand on my written argument.

THE COURT: A lot of what you say in there are conclusions. Do you have anything to back up those conclusions?

MR. LIEBICH: No.

THE COURT: If you say somebody wasn't prepared, what do you mean?

MR. LIEBICH: Well, there's no way he could have been prepared by failing to present most of this evidence that was critical to my defense. Mr. Holman wasn't even aware of a lot of the stuff that I brought to his attention during the trial. So I don't feel there's any way he could have been fully prepared not knowing about this information.

THE COURT: Again, do you have any examples? I read your, the longer presentation.

MR. LIEBICH: Basically a lot of it's just evidence that he didn't bring forward that was in discovery.

THE COURT: For example?

MR. LIEBICH: Let's see. There were other possible witnesses that should have been brought forward that he didn't call.

THE COURT: For example, who would that be and what would they have testified to?"

Defendant then listed the following purported deficiencies: (1) counsel failed to call Richard O'Brien, a polygrapher, to testify that Kenyatta admitted striking Steven with a belt and comb; (2) counsel failed to call Dion Liebich to impeach Robert Liebich by testifying that defendant never asked Robert whether the room in the Roselle police station was "bugged" during their conversation; (3) counsel failed to bring forth evidence from Kenyatta's diary that Lee Clark (Karen's husband) beats the children in the Clark home and Karen does not interfere, to undermine the notion, cited by the trial court in its adjudication of guilt, that Karen would have done anything to protect Steven; (4) counsel failed to bring forth evidence that Steven had Tylenol and aspirin in his system to undermine the proposition that Steven was feeling fine prior to February 8, 2002; (5) counsel failed to bring forth evidence that Steven lost five pounds between November 6, 2001, and February 8, 2002; (6) counsel failed to bring forth evidence that Steven had been given drugs to sedate him prior to the time he was first examined at Rush Presbyterian, which caused his stomach to be soft and not distended; (7) counsel failed call Dorothy Herron to testify that February 8, 2002 was not the first time Steven was

left home alone with defendant; and (8) counsel failed to bring forth evidence or investigate information regarding a child in Rockford, whose mother's name was Kenyatta Brown, "mysteriously end[ing] up with a broken leg."

After defendant set forth these complaints, the trial judge asked, "Anything else?" Defendant replied, "That's pretty much it, your Honor." The trial court then inquired of one of defendant's attorney--John Casey--regarding whether he wished to address any of these issues. Casey stated that the facts defendant set forth were correct; however, he maintained that the decision regarding whether to present them was a matter of trial strategy. The court then requested that defendant's other attorney, Ricky Holman, be brought to the court room.

The court then asked Holman whether he wished to offer any explanations regarding the issues raised by defendant. Holman explained that, as the trial court had previously observed, it would be unreasonable to expect him to call defense witnesses to offer additional impeachment after the court assessed testimony of certain state witnesses, particularly where those state witnesses were in fact impeached. Holman stated that he reviewed reports from additional witnesses and made a strategic decision not to present them. Evidence of aspirin and Tylenol was contained in Teas's ex parte letter, and thus, though he was now aware of it, he could no longer use this evidence. Investigators from both the public defender's office and the State determined that the Kenyatta Brown in Rockford was not the same person as the Kenyatta Brown involved in this trial.

The trial court then asked Holman whether he failed to communicate a plea agreement to defendant. Holman stated that he did communicate one posttrial offer to defendant, which they agreed to reject. Prior to trial, said Holman, no offers were made, and defendant and Holman agreed to maintain defendant's innocence. Holman did tell defendant that the State had made overtures

regarding a plea, even though no firm offer was ever made. The court also inquired of Holman regarding defendant wishing to testify, which Holman denied. The court then ruled, finding that defendant's allegations pertained to evidence that was cumulative, collateral, or not of any particular relevance. The court also found that no offer was made before trial. It then ruled that it was not necessary to appoint new counsel, and it denied defendant's pro se motion. It further ordered that defendant's pro se motion be incorporated into defendant's original posttrial motion for the purpose of preserving these issues for appellate review. The trial court specifically asked whether either party objected to this final ruling, and neither side did. It also denied defendant's motion to reconsider the sentence it had imposed. Defendant now appeals.

II. ANALYSIS

Defendant raises a number of issues on appeal. First, he asserts that the trial court did not conduct an adequate inquiry into his pro se allegations of ineffective assistance of counsel and that this cause should be remanded for such an inquiry. Next, he contends that he was not proven guilty beyond a reasonable doubt because (1) the trial court considered evidence that was inadmissible under Frye v. United States, 293 F. 1013 (D.C. Cir. 1923); (2) the State failed to prove the essential elements of first-degree murder and instead only proved involuntary manslaughter; and (3) the trial court made inconsistent findings regarding defendant's mental state. Third, defendant argues that the testimony of Dr. Munoz and Dr. Severin should not have been admitted under Frye. Fourth, defendant claims trial counsel was ineffective. We will address these arguments as defendant presents them in his brief.

A. ADEQUACY OF THE INQUIRY INTO DEFENDANT'S PRO SE ALLEGATIONS OF INEFFECTIVE ASSISTANCE OF COUNSEL

Defendant first argues that the trial court did not conduct an adequate inquiry into his pro se claims of ineffective assistance of counsel. See People v. Moore, 207 Ill. 2d 68, 81 (2003). Defendant point out that, on August 27, 2004, he made an oral motion for a new trial alleging trial counsel's ineffectiveness. The trial court, after asking only the State for input, stated:

> "The Court has appointed the public defender to represent you, and if the public defender--If you chose to to [sic]dismiss the public defender, you have the right to hire your own attorney; but the Court will not give you an additional attorney because you do not wish to proceed today."

The trial court made no inquiry of defendant, or, for that matter, defense counsel.

When a defendant makes a pro se charge of ineffective assistance of counsel, the appointment of new counsel is not always necessary. Moore, 207 Ill. 2d at 77. Instead, the trial court must conduct an inquiry into the factual basis of the defendant's claim. Moore, 207 Ill. 2d at 77-78. Where a claim lacks merit or only touches upon trial strategy, new counsel need not be appointed. People v. Williams, 147 Ill. 2d 173, 251 (1991). Only where the claim shows possible neglect of the case is new counsel necessary. Williams, 147 Ill. 2d at 251.

On review, the inquiry focuses upon the adequacy of the trial court's inquiry into a defendant's claims. People v. Johnson, 159 Ill. 2d 97, 125 (1994). The inquiry may take three forms. Typically, some discussion between the trial court and trial counsel, including counsel simply answering questions and explaining the circumstances surrounding defendant's allegations, is usually necessary. Moore, 207 Ill. 2d at 78. Additionally, the trial court may discuss the allegations with the defendant. Moore, 207 Ill. 2d at 78. Further, the trial court may rely on its own recollection of defense counsel's performance. Moore, 207 Ill. 2d at 79. A defendant's allegations may also be insufficient on their

face. Moore, 207 Ill. 2d at 79. Whether the trial court made an adequate inquiry into a defendant's pro se claims of ineffective assistance of counsel presents a question of law subject to de novo review. People v. Strickland, 363 Ill. App. 3d 598, 606 (2006).

In this case, the trial court did not discuss defendant's allegations with either defense counsel or defendant during the hearing on August 27, 2004. There is also no indication in the record that it was relying on its own recollection of defense counsel's performance. The record does not reveal, and the State does not suggest, that the claims were facially insufficient. Indeed, defense counsel informed the court that defendant was raising the issue of ineffectiveness, but counsel did not relay to the court the nature of those claims and the court did not make any inquiry into what, precisely, they were, much less their factual basis.[6] The trial court simply dismissed the claim, stating defendant

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[6] In this section of his brief, defendant states that "many or most of [his] pro se claims dealt with counsel's failure to do things at trial, such as call witnesses, so it is unclear how the trial court's recollection would shed any light on this issue." Later during the hearing, after the trial court denied defendant's motion, defense counsel set forth defendant's claims with some specificity. These issues were (1) that counsel should have brought in additional witnesses to impeach Kenyatta regarding her purported abuse of Steven; (2) that counsel should have had a witness testify that defendant had changed Steven's diapers in the past; (3) that counsel should have brought in additional witnesses to testify regarding other inconsistencies in Kenyatta's testimony; (4) that other detectives should have been called to testify regarding a "hollow noise" Kenyatta stated she heard while defendant was in the bedroom with Steven; (4) that counsel should have conducted more thorough cross-examinations; (5) that counsel failed to present evidence that no clothes hangers were found in the garbage on February 9, 2002. The trial court expressly directed that these issues, though presented orally, had

had an attorney, had the right to hire private counsel, but would not be appointed an additional attorney. The State agrees that the trial court did not make an adequate inquiry into defendant's claims during the August 27, 2004, hearing.

However, the State contends that, under the unique facts and circumstances of this case, a remand is not necessary. The State points out that defendant filed a written motion alleging ineffective assistance of counsel on November 10, 2004. On that date, the State reminded the court of its obligation to examine the factual basis of defendant's allegations. A hearing was ultimately held on defendant's motion on December 7, 2004. During this hearing, the trial court conducted an extensive inquiry into defendant's allegations, asking defendant to clarify and exemplify them. Defendant, in fact, articulated eight distinct complaints about defense counsel's conduct. After defendant specified his complaints, the trial judge asked, "Anything else?" Defendant answered, "That's pretty much it, your Honor." The trial court also inquired of both of defendant's trial attorneys. The State asserts that this inquiry cured any deficiency in the trial court's earlier handling of defendant's claims. We agree.

Essentially, defendant is asking that we remand this cause so the trial court can ask "Anything else?" yet one more time. The remedy where a trial court fails to make an adequate inquiry into a claim of ineffectiveness is a remand to allow the court to make the proper inquiry. Moore, 207 Ill. 2d at 81. Neither a full evidentiary hearing on the question of trial counsel's purported incompetence nor the appointment of new counsel to aid in the inquiry is required. Moore, 207 Ill. 2d at 81-82. On December 7, 2004, the trial court inquired, defendant explained the factual basis for his claims, defendant stated that there were no additional issues he wished to raise, and the court ruled. Insofar

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been sufficiently raised and preserved.

as ineffectiveness is concerned, it is not apparent to us how a hearing on remand would differ in any appreciable way from the hearing conducted on December 7. Defendant raised all the issues he wanted to at that time, as he indicated when he stated, "That's pretty much it." A remand so that this issue could be rehashed one more time would be both a meaningless gesture and a waste of judicial resources. The trial court already ruled on the issues defendant raised, and defendant provides no basis for supposing that a different result would obtain on remand. See People v. Blair, 215 Ill. 2d 427, 446-47 (2005) ("To hold otherwise, we would be forcing courts to waste judicial resources by merely delaying the dismissal of a petition which the judge knows could never bear fruit for the petitioner").

In short, we will not engage in such a meaningless gesture. As defendant was given an adequate opportunity to present his claims to the trial court and indicated that he was satisfied that he had presented them all, and further because the trial court made an extensive inquiry of defendant and both his trial attorneys, we conclude that any error resulting from the trial court's summary dismissal in August was cured in the December hearing. No remand is necessary because the trial court already made the inquiry we would be directing it to make on remand. Finally, we find no error regarding the trial court's determination that new counsel was not necessary. Following a proper inquiry, such a decision will be disturbed only if it is manifestly erroneous (People v. Young, 341 Ill. App. 3d 379, 382 (2003). Here, the trial court's point-by-point explanation of its reasons for not appointing new counsel are clearly sufficient under that standard.

B. PROOF OF GUILT BEYOND A REASONABLE DOUBT

Defendant makes three arguments as to why he was not proven guilty beyond a reasonable doubt. He argues that the trial court considered inadmissible evidence, that the State failed to prove

the essential elements of the crime, and that the trial court made inconsistent findings. We will address these contentions in turn.

1. Inadmissible Evidence under Frye

While defendant characterizes this argument a Frye issue (see Frye v. United States, 293 F. 1013 (D.C. Cir. 1923)), in actuality, he is simply asking that we reweigh the evidence and accept the testimony of two expert witnesses that opined favorably to him rather than the treating physicians upon whom the trial court relied. Frye is, in fact, only cited once, in the course of a three-sentence discussion, and its tenets are not discussed or applied in a sustained or coherent form.

Defendant begins this argument by setting forth the strengths of the two favorable experts--Dr. Mileusnic Polchan and Dr. Teas. Both are forensic pathologists with impressive credentials and are certainly qualified to opine as to the timing of the injuries inflicted upon Steven. Further, both relied upon histological analysis, a well-accepted methodology, in coming to their conclusions.

Before proceeding further, we note that Mileusnic stated that the injuries "could have occurred on the 8th," which is consistent with the State's position and the testimony of Dr. Munoz and Dr. Severin. Thus, even if we were to accept defendant's invitation to reassess the evidence, it is unclear to whom we would deem Mileusnic's testimony favorable. In addition to setting forth the credentials and testimony of Mileusnic and Teas, defendant also points out that no witness saw him strike Steven and no physical evidence links him to Steven's death (we cannot help but note that such observations have nothing to do with the propriety of the testimony of Munoz and Severin under Frye, which reinforces the notion that defendant is actually asking us to reweigh this evidence). We also note the Mileusnic stated that she would defer to a treating physician on the issue of timing and

that there was testimony that Steven was fine on the morning of February 8 before being left alone with defendant for over six hours and injured after that.

Defendant attacks Munoz's qualifications to determine the timing of an injury, incorrectly stating that Munoz "offered no expert experience in determining the timing of injuries." To the contrary, Munoz testified DCFS had consulted with him "hundreds" of times regarding suspected child abuse, and on each occasion, he was asked to give an opinion regarding the timing of the injury. Defendant also disingenuously states that neither Munoz nor Severin "had ever testified in court about the timing of injuries before." (Emphasis omitted and added.) Munoz, in fact, testified that he had given his opinion in six depositions previously. While, strictly speaking, these opinions were not given "in court," they were obviously given in the course of legal proceedings.

Defendant does complain of Munoz's testimony regarding the timing of Steven's injuries based on the color of the blood Munoz found when he opened Steven's head. The color of the blood was bright red, which Munoz took as a sign of a recent injury. The State asserts that this deduction is not subject to Frye. Scientific evidence is that which derives from the "application of scientific principles, rather than on skill or experienced-based observations, for the basis of his opinion." Jackson v. Seib, 372 Ill. App. 3d 1061, 1073 (2007). The Sixth Circuit Federal Court of Appeals illustrated the distinction between scientific and nonscientific opinion evidence thusly:

> "The distinction between scientific and non-scientific expert testimony is a critical one. By way of illustration, if one wanted to explain to a jury how a bumblebee is able to fly, an aeronautical engineer might be a helpful witness. Since flight principles have some universality, the expert could apply general principles to the case of the bumblebee. Conceivably, even if he had never seen a bumblebee, he still would be qualified to testify, as

long as he was familiar with its component parts.

On the other hand, if one wanted to prove that bumblebees always take off into the wind, a beekeeper with no scientific training at all would be an acceptable expert witness if a proper foundation were laid for his conclusions. The foundation would not relate to his formal training, but to his firsthand observations. In other words, the beekeeper does not know any more about flight principles than the jurors, but he has seen a lot more bumblebees than they have." Berry v. City of Detroit, 25 F.3d 1342, 1349-50 (6th Cir. 1994).

Munoz is more like the beekeeper. His expertise is not derived from the abstract application of scientific principles; rather, it is based upon what he has observed in his years as a doctor. Hence, his observation that bright red blood is indicative of a recent injury is not subject to Frye because it is grounded in his own experience. Defendant was, of course, free to attack Munoz's experience regarding whether it was a sufficient basis to render this opinion, but such arguments go only to the weight to which the opinion is entitled, not its admissibility. People v. Swart, 368 Ill. App. 3d 614, 633 (2006).

Defendant attacks Severin to the extent that he relied upon Munoz's conclusions. As we have determined that Munoz's testimony was proper, defendant's derivative attack upon Severin must also fail. Accordingly, we find defendant's assertion that the trial court relied on testimony that was not admissible under Frye ill founded. Moreover, to the extent that the opinions of Munoz and Severin conflicted with those of Mileusnic and Teas, it was for the trial court, in the first instance, to resolve that conflict, (People v. Harrison, 366 Ill.App. 3d 210, 219 (2006)), and its resolution of this issue is amply supported by the evidence.

2. Elements of First-Degree Murder

Defendant next asserts that the State failed to prove him guilty of first-degree murder. In reviewing the sufficiency of the evidence to sustain a verdict, we must construe the record in the light most favorable to the State. People v. Tabb, 374 Ill. App. 3d 680, 691 (2007). At issue is whether "any rational trier of fact could have found the essential elements of the crime beyond a reasonable doubt." People v. Bush, 214 Ill. 2d 318, 326 (2005). A reviewing court will not set aside a conviction unless the evidence is so unsatisfactory or the possibility of a defendant's guilt so improbable as to raise a reasonable doubt regarding that guilt. People v. McGee, 373 Ill. App. 3d 824, 832 (2007).

Defendant focuses his argument upon the State's evidence concerning mens rea. The first-degree murder statute provides:

> "A person who kills an individual without lawful justification commits first-degree murder if, in performing the acts which cause the death:
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> (1) he either intends to kill or do great bodily harm to that individual or another, or knows that such acts will cause death to that individual or another; or
>
> (2) he knows that such acts create a strong probability of death or great bodily harm to that individual or another; or
>
> (3) he is attempting or committing a forcible felony other than second degree murder." 720 ILCS 5/9--1 (West 2002).

Defendant was in fact indicted on four counts of first-degree murder. The first count alleged defendant acted "knowing said act would cause the death of Steven Quinn." The second count alleged that defendant intended to do great bodily harm to Steven. The third count alleged that defendant knew his actions created a strong probability of death. Finally, the fourth count alleged

defendant knew his actions created a strong probability of great bodily harm. The State apparently nolle prossed the first count, and the trial court convicted defendant on the remaining counts but merged the last two into the second for purposes of sentencing. Relevant here, "intent" is defined as "when [a person's] conscious objective or purpose is to accomplish that result or engage in that conduct." 720 ILCS 5/4--4 (West 2002). "Knowledge" with respect to the result of conduct exists when a person is "consciously aware that such result is practically certain to be caused by his conduct." 720 ILCS 5/4--5(b) (West 2002).

> Defendant contends that the State only proved recklessness, which is defined as follows:
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> "A person is reckless or acts recklessly, when he consciously disregards a substantial and unjustifiable risk that circumstances exist or that a result will follow, described by the statute defining the offense; and such disregard constitutes a gross deviation from the standard of care which a reasonable person would exercise in the situation." 720 ILCS 5/4--6 (West 2002).

Defendant contends that the trial court has an independent duty to ascertain whether a defendant is guilty of a lesser-included offense, even if defense counsel did not advance such an argument. Indeed, the law is clear that, in a bench trial, "the judge determines from the evidence whether the defendant is guilty of murder or of some lesser included offense, and the defendant has no 'right' to restrict the judge's determination to the question of his guilt or innocence of murder." People v. Garcia, 188 Ill. 2d 265, 273 (1999), quoting People v. Taylor, 36 Ill. 2d 483, 488-89 (1967); see also People v Turner, 337 Ill. App. 3d 80, 90 (2003).

In support of this argument, defendant points to the trial court's findings that he was calm, nonchalant, aloof and relaxed, rather than tearful or nervous during the initial period after arriving

at Mount Sinai. It further stated that he had a "flat affect." At this time, the court observed, the seriousness of Steven's injuries had not been communicated to defendant. As defendant learned of Steven's condition, he became scared and his demeanor changed. The court also noted defendant's statement to his cousin, Officer Robert Liebich, that he "didn't hit the kid that hard." This statement, the court found, was consistent with defendant's lack of concern when they first arrived at the hospital. As the trial court put it, "It also explains why he was not concerned, originally, being at Mount Sinai because it is his belief he didn't hit the kid that hard, what could be wrong." These findings, according to defendant, show only recklessness.

The State counters that it is sufficient to prove that defendant "voluntarily and willfully committed an act, the natural tendency of which is to destroy another person's life, with the intent being implied from the character of the act, and the disparity in size and strength between the defendant and the victim." See People v. Reeves, 228 Ill. App. 3d 788, 798 (1992). Further, knowledge is often proven by circumstantial evidence rather than direct proof. People v. Brogan, 352 Ill. App. 3d 477, 493 (2004). The State points to the quantity and severity of the injuries Steven sustained, and we agree that, from this alone, it is possible to infer an intent to kill or do great bodily harm as well as knowledge on defendant's part that his acts created a strong probability of great bodily harm. See People v Tye, 141 Ill. 2d 1, 16 (1990) ("In the present case, the defendant was an adult male, and the victim was a three-year-old child. In beating the child, the defendant used first a belt and then an extension cord. According to the defendant's own statement, the beating lasted about an hour. The injuries sustained by the child were severe and too numerous to count. Considering the disparity in size between the defendant and the victim, the brutality and duration of

the beating, and the severity of the victim's injuries, we conclude that the trial judge could infer that the defendant acted with the necessary mental state in bringing about the child's death").

In People v. Rodriguez, 275 Ill. App. 3d 274, (1994), the First District of this appellate court confronted a situation similar to the one present here. Rodriguez involved the beating death of a three-year old child perpetrated by an adult male. The medical examiner testified that during the autopsy, he observed numerous bruises on the child's face, ears, back, left buttock, arms, and legs. The victim's stomach was distended, and the medical examiner discovered a large amount of blood in her abdominal cavity. There were three lacerations to the child's intestines. It would have required significant force to inflict such injuries, opined the medical examiner. Additionally, multiple areas of bleeding were discovered in the soft tissue over the victim's skull. There was evidence that defendant struck the victim twice in the abdomen with an open hand. Unlike this case, there was also evidence of previous abuse by the defendant; however, the Rodriguez court expressly noted that it would have come to the same conclusion even without such evidence. Rodriguez, 275 Ill. App. 3d at 286. Similarly, there was evidence that the defendant had struck the victim out of anger, but the court found this to be additional, supporting evidence. Rodriguez, 275 Ill. App. 3d at 285.

Relevant for our purposes is the Rodriguez court's analysis of the victim's injuries as it bore upon the defendant's mental state. The court began by observing, "With respect to whether a defendant intends to kill or knows that his actions are probable to cause death or great bodily harm, an accused's intent or knowledge can be reasonably inferred from, and are often proved by, the circumstances surrounding the incident including the nature and severity of the victim's injuries." Rodriguez, 275 Ill. App. 3d at 284. An inference that a defendant intended or had knowledge that his actions created a strong probability of death "arises when a defendant strikes a blow with a bare

hand when a great disparity in size and strength exists between him and the victim even though a bare hand is not ordinarily regarded as a deadly weapon." Rodriguez, 275 Ill. App. 3d at 284. The court further reasoned:

> "[T]he admissible evidence in this case overwhelmingly proved that the defendant possessed the requisite mental state for first-degree murder at the time of the killing. A great disparity in size and strength obviously existed between the three year old, three foot tall, thirty pound victim and the defendant, who admitted to striking two blows to her abdomen. [Citation.] [The medical examiner] testified that 'significant force' would have been necessary to produce the injuries to the mesentery which resulted in the victim's death and Detective Winistorfer described the defendant's demonstration of the manner in which he struck her as delivering 'two forceful blows' with the palm of his open hand which 'reverberated in the room and on the wall.' " Rodriguez, 275 Ill. App. 3d at 285.

It then held: "This evidence showing the great disparity in size between the victim and the defendant, the vital part of the victim's body to which the fatal blows were struck, and the testimony suggesting that they were in fact forceful, together create a strong inference that the defendant either knew that his acts created a substantial probability of, or that he intended to cause, death or great bodily harm." Rodriguez, 275 Ill. App. 3d at 285.

Rodriguez provides sound guidance for the resolution of this issue. Like Rodriguez, in the present case, defendant is an adult and Steven was a child, so there was a great disparity in size between defendant and Steven. Further, a number of the blows suffered by Steven were directed to his head, and, as in Rodriguez, his abdomen. These are vital parts of the respective victims' bodies. In Rodriguez, the medical examiner testified that significant force was required to inflict the injuries.

In ths case, Mileusnic stated that the injuries inflicted upon Steven would not be caused by "normal corporal punishment," but would require "something much more forceful." In sum, knowledge and intent can both be inferred from the severity and quantity of the injuries defendant inflicted upon Steven. As we stated previously, "a conviction will not be set aside on grounds of insufficient evidence unless the proof is so improbable or unsatisfactory that there remains a reasonable doubt as to the defendant's guilt." People v. Doll, 371 Ill. App. 3d 1131, 1135 (2007). Such is not the case here.

3. Inconsistent Findings

Defendant next asserts that the trial court made inconsistent findings in that it found he did not appreciate the gravity of the situation when he first arrived at Mount Sinai. The trial court noted that defendant's demeanor changed as the night progressed, and, as he became aware of Steven's condition, defendant became scared. The court pointed to Robert Liebich's testimony that defendant told him he "didn't hit the kid that hard." This, according to the trial court, explained his nonchalant demeanor when he first arrived at the hospital: "it is his belief that he didn't hit the kid that hard, what could be wrong." This finding, according to defendant, is inconsistent with the trial court's finding that he acted knowingly and intentionally when he killed Steven. In support, defendant relies primarily on cases involving inconsistent verdicts (see e.g., People v. Hoffer, 106 Ill. 2d 186 (1985); People v. Spears, 112 Ill. 2d 396 (1986)), which are not directly on point here. Unlike these cases, the trial court never reached verdicts that were not consistent. Instead, what defendant is complaining about is an apparent discrepancy between one of the trial court's findings and its ultimate verdicts.

Initially, we note the finding of which defendant complains is not entirely inconsistent with all of the verdicts in this case. Defendant points out that "it is legally impermissible to find that a defendant's mental state is both less-than-knowing and knowing." In Hoffer, 106 Ill. 2d at 195, for example, the court determined that the mental state necessary to convict a person of involuntary manslaughter precluded the existence of the mental state necessary for a murder conviction. This is because involuntary manslaughter is defined as the unintentional killing of a human being caused by acts that are performed recklessly. Thus, it expressly excludes an intentional killing from its scope. Further, since recklessness is defined as the conscious disregard of a substantial risk (People v. Barham, 337 Ill. App. 3d 1121, 1130 (2003)), and "knowledge" for the purpose of the murder statute requires awareness of a "strong probability" of death or great bodily harm (see 720 ILCS 5/9--1 (West 2002)), these mental states are mutually exclusive in that they require awareness of different levels of risk. However, in this case, the trial court did not find defendant guilty of involuntary manslaughter, it simply found he was unaware of the gravity of the situation when he arrived at Mount Sinai. In other words, the finding at issue in cases like Hoffer--that the defendant was guilty of involuntary manslaughter--entails a finding that the killing was unintentional, as per the statutory definition of the crime. In this case, the trial court made no finding regarding defendant's intent, and, more importantly, its finding that defendant did not comprehend the seriousness of Steven's condition does not preclude a finding that defendant intended to cause Steven great bodily harm at the time he acted.

Indeed, defendant's argument, in a sense, compares proverbial apples and oranges. Defendant's mental state at Mount Sinai concerned things that had already happened, that is, the past consequences of past actions. The relevant mental state for determining guilt is that mental state that

accompanied the actions that constituted the crime. People v. Grever, 353 Ill. App. 3d 736, 757 (2004), rev'd on other grounds, 222 Ill. 2d 321 (2006) ("Criminal liability requires the conjunction of a culpable mental state (at common law, the mens rea) and a punishable act or omission (at common law, the actus reus)"). Thus, defendant's nonchalance at Mount Sinai may have been because he did not believe he did anything sufficient to fulfil his intent to kill Steven. Similarly, his statement to Robert Liebich that "he didn't hit the kid that hard," could be based on his assessment of his actions made at the time he took them or it could be based on his perception of the results of his actions between the time he beat Steven and the time Steven's injuries began to manifest themselves. Thus, the trial court's finding is not inconsistent with a finding of intent.

Lest defendant charge that we are doing nothing more here than engaging in much hypothetical speculation, we point out the following. Defendant is relying on cases about inconsistent verdicts. Such cases typically involve a certain amount of speculation regarding possible bases for the verdicts, and, if there is some plausible basis that does not require the finding of the existence and nonexistence of some element of the crimes, the verdicts are allowed to stand. See People v. Foley, 152 Ill. App. 3d 354, 357 (1987) ("As in Munday, the verdicts could have been based on compromise or an exercise of lenity. In addition, defendant presented some evidence which tended to indicate that the breathalyzer reading was inaccurate. Thus, the jury might have disregarded it entirely, yet still found sufficient other evidence to find defendant guilty of driving under the influence of alcohol" (emphasis added)). In People v. Munday, 134 Ill. App. 3d 971, 976-77 (1985), the court, after considering a number of possible reasons for two seemingly conflicting verdicts, including lenity, compromise, and confusion, held:

"We do not know, and need not decide, which of these possible explanations is correct; the fact remains that defendant has not carried his burden to demonstrate the acquittal was necessarily based on a finding he had no intent to commit rape. As defendant has thus not demonstrated a legal inconsistency of the verdicts, collateral estoppel cannot be applied and his retrial for residential burglary was proper."

The Munday court resolved the issue based entirely on possible scenarios that would explain the apparent conflict between the two verdicts at issue. Thus, we can do the same here. Since, as explained above, the trial court's factual finding based on defendant's demeanor at Mount Sinai is not inconsistent with a finding of intent, case law regarding inconsistent verdicts is of no help to defendant. Furthermore, as the trial court's findings did not preclude convictions based on intent, and because all counts were merged, any complaint regarding the knowledge-based counts are moot. See People v. Hemphill, 230 Ill. App. 3d 453, 468 (1992) ("Next, McIntyre argues that his due process rights were violated because he was not proved guilty of conspiracy of armed robbery beyond a reasonable doubt. McIntyre was found guilty of armed robbery against Jackson. The trial court stated that the conspiracy conviction merged with the armed robbery conviction and only sentenced McIntyre for the armed robbery conviction; thus, this issue is moot").

Accordingly, the trial court's findings regarding defendant's behavior at the hospital do not preclude a finding that defendant intended to kill or do great bodily harm to Steven. Finally, to the extent that evidence regarding defendant's behavior at Mount Sinai weighs in favor of an acquittal, it merely created an evidentiary conflict for the trial court to resolve. People v. Roberts, 374 Ill. App. 3d 490, 497-98 (2007). Substantial evidence militated for a finding of guilt, not the least of which was Kenyatta's testimony that Steven was fine when she left for work, Steven spent the day alone

with defendant, and when Kenyatta returned, Steven was severely injured. None of defendant's arguments persuade us that he was not proven guilty beyond a reasonable doubt.

C. ADMISSIBILITY OF EXPERT OPINION TESTIMONY

Defendant next alleges error in the trial court's admission of the opinion testimony regarding the timing of Steven's injuries from Dr. Munoz and Dr. Severin. Defendant argues that they had no training and possessed no expertise regarding timing. He also contends that their opinions lacked a "scientifically-recognized foundation." The admissibility of an expert opinion is a matter committed to the discretion of the trial court, and a court of review will not interfere with an exercise of that discretion so long as it is not abused. Volpe v. IKO Industries, Ltd., 327 Ill. App. 3d 567, 576 (2002).

Defendant first baldly and incorrectly asserts that, "Since neither Munoz nor Severin had any prior experience with respect to establishing the time of an injury, their opinions on that issue should not have been admitted." This statement is simply false. Munoz testified that he has been involved in "hundreds" of cases of suspected child abuse, and in each case, DCFS personnel have asked him to give an opinion regarding the timing of the injury. Severin, on the other hand, testified that sometimes, authorities, DCFS personnel, other health care workers, or individuals from Child Protective Services (CHP) would sometimes ask him to "correlate their exam with findings of progression of the disease." However, he did acknowledge that he had never been asked for such an estimate in any of the 20 child-abuse cases in which he has been involved. However, to state that neither of these doctors had any experience is pure hyperbole. Whether they had enough experience to render such opinions is a different question. Defendant cites no case law regarding the quantum of experience necessary to render an opinion. In fact, defendant cites no case law whatsoever in

support of this argument, which waives the issue. See People v. Acevedo, 191 Ill. App. 3d 364, 366 (1989).

Defendant next asserts Munoz and Severin "relied on faulty information" in forming their opinions. Defendant points out that Munoz, upon reviewing the CAT scan from Mount Sinai, believed there was a large subdural hematoma in the right side of Steven's head. Munoz did not find such an injury when he subsequently operated upon Steven. Defendant states, "So Munoz's conclusion about the fact that there was a significant subdural hematoma on the right side of the brain proved to be incorrect." We fail to see the relevance of these observations. Munoz presumably did not formulate his ultimate opinion until after he performed surgery. It is a dubious suggestion indeed, and one we find completely unpersuasive, that Munoz relied on his interpretation of the CAT scan rather than what he learned during surgery in coming to the conclusions to which he testified at trial. Cf. Jeffers v. Weinger, 132 Ill. App. 3d 877, 882 (1985) ("Therefore, any possibility that Dr. Fossier based his opinion on an incorrect assumption was nullified when the defendants [subsequently] clarified the situation on cross-examination"). Defendant also argues that Munoz believed that, prior to surgery, Steven was not paralyzed in any way. Dr. Teas's report indicated that records from Mount Sinai show that Steven had been given medicines to paralyze his abdomen. Defendant does not explain how this gap in Munoz's knowledge affected his opinion, or, for that matter, whether he was still unaware of the use of these medications at the time he rendered his opinion. Moreover, our supreme court has held that "the basis for a witness' opinion generally does not affect his standing as an expert; such matters go only to the weight of the evidence, not its sufficiency." Snelson v. Kamm, 204 Ill. 2d 1, 26 (2003). That principle controls here, and it was for the trial court to determine how any such omissions affected the weight to which Munoz's testimony was entitled (People v. Harrison,

366 Ill. App. 3d 210, 219 (2006)). Defendant attacks Severin's opinion on this same basis, and we find the attack no more persuasive here than when it was directed against Munoz.

Defendant cites People v. Wilhoite, 228 Ill. App. 3d 12 (1991), for the proposition that an expert's opinion must be disregarded where it is without proper foundation. Indeed, the exact language from Wilhoite is: "If the expert's opinion is without proper foundation, particularly where he fails to take into consideration an essential factor, that opinion is of no weight and must be disregarded.' " (Emphasis added.) Wilhoite, 228 Ill. App. 3d at 21, quoting 32 C.J.S., Evidence, § 569(1) at 609. The Wilhoite court spoke of "an essential factor." Defendant, aside from pointing out the alleged flaws in the bases of Munoz's and Severin's opinions, makes no attempt to show why these purported defects were so critical as to constitute a matter of admissibility rather than weight. In any event, we hold that they were the latter.

Finally, defendant contends that Munoz and Severin offered no scientific bases for their opinions that would comport with the Frye standard. See Frye, 293 F. 1013. Defendant again complains that Munoz based his opinion on the color of the blood in Steven's head. Defendant contends that this is not an accepted scientific methodology. We agree. In fact, as we explained earlier, this was not a scientific methodology at all. It is not derived from the "application of scientific principles" but from Munoz's own skill and experience based upon his observations made in the course of his career. Jackson, 372 Ill. App. 3d at 1073. Moreover, "[s]imply because scientific principles relate to aspects of an opinion witness's testimony does not transform that testimony into 'scientific' testimony." Jackson, 372 Ill. App. 3d at 1073. Defendant also points to a number of alleged defects in Munoz's opinions here, such as the fact that much of Munoz's experience was derived from car accidents rather than child abuse and his failure to consider the effect of Steven's

abdominal injury on his head injury. Such things go to weight, not admissibility. Harrison, 366 Ill. App. 3d at 219.

Regarding Severin, defendant states, "Severin likewise offered no scientifically-based explanation regarding how his estimate of the timing of the bruises on Steven's body comported with his later estimate of the timing of the injuries." The absence of a "scientifically-based explanation" is not a basis for objecting to Severin's opinion. Severin is board certified in pediatric critical care. Based on his training and experience, he was certainly entitled to opine regarding Steven's injuries. Scientific testing may have provided additional support for Severin's opinion, but such additional support was not necessary for the trial court to consider the opinion. Indeed, defendant presented countervailing scientific evidence (the histology slides), and it was for the trial court to resolve the conflict between Severin's testimony and that scientific evidence just as it would have been for the trial court to attribute additional weight to Severin's opinion had it been supported by corroborating scientific evidence.

In sum, Munoz's and Severin's testimony was clearly admissible. The sorts of things of which defendant complains typically are matters of weight. Therefore, we reject his arguments on this point.

D. INEFFECTIVE ASSISTANCE OF COUNSEL

Defendant makes five distinct arguments as to why his counsel was ineffective. First, he points to counsels' failure to interpose a Frye objection to the testimony of Munoz and Severin. Based on our discussion of this issue above, any such objection would have been futile, and we will not consider the issue further. See In re Ottinger, 333 Ill. App. 3d 114, 118 (2002) ("The failure of defendant's counsel to make a futile objection does not constitute fundamentally deficient performance"). Second, defendant complains of his trial attorneys' failure to argue that a letter from

Dr. Teas to the court that was received after the trial had concluded should be considered. Defense counsel actually agreed with the State that the letter should be impounded. Third, defendant asserts that trial counsel should have argued that he was guilty of a lesser-included offense, namely involuntary manslaughter. Fourth, defendant claims that "[d]efense counsel neglected to effectively cross-examine and impeach several witnesses." Defendant's entire argument on this point consists of the following: "The Record [sic] contains multiple instances of defense counsel failing to effectively impeach prosecution witnesses." This statement is followed by a string citation to five places in the record, but contains no reference to legal authority. As has oft been stated, "A reviewing court is entitled to have the issues before it clearly defined and is not simply a repository in which appellants may dump the burden of argument and research; an appellant's failure to properly present his own arguments can amount to waiver of those claims on appeal. People v. Chatman, 357 Ill. App. 3d 695, 703 (2005). We deem that to be the case here, and we will not give this issue further consideration. Fifth, defendant contends that a Rule 604(d) (188 Ill. 2d R. 604(d)) certificate filed by trial counsel contained material inaccuracies.

To succeed on a claim of ineffective assistance of counsel, a defendant must show that his attorney's performance fell below an objective level or reasonableness in light of prevailing professional norms and that this deficient performance prejudiced the defendant. People v. Ramirez, 371 Ill. App. 3d 738, 744 (2007). In order to establish deficient performance, a defendant "must overcome the strong presumption that the challenged action or inaction might have been the product of sound trial strategy." People v. Jackson, 205 Ill. 2d 247, 259 (2001). To show prejudice, a defendant must show a reasonable probability that, but for counsel's errors, the outcome of the proceeding would have been different. Jackson, 205 Ill. 2d at 259. A "reasonable probability" is one

sufficient to undermine confidence in the result of the proceeding. People v. Harris, 206 Ill. 2d 293, 304 (2002). Sometimes, it is easier to dispose of a claim on the prejudice prong of the test, and, in such cases, "counsel's performance need not be evaluated." People v. Brooks, 187 Ill. 2d 91, 137 (1999). With these standards in mind, we will now turn to the balance of defendant's arguments.

1. Dr. Teas's Letter

After the trial had concluded, Teas sent a letter to the trial court. The letter set forth a number of purported exculpatory facts that were supposedly not brought out by defense counsel during the trial. She characterized these facts as "significant findings in the medical records." She sent copies of the letter to the State, defense counsel, and Dr. Mileusnic. Defense counsel agreed with the State that the letter constituted an improper ex parte communication and that it should be impounded in the record.

Defendant now claims trial counsel was ineffective for not seeking to have the letter admitted into evidence. Defendant, citing Black's Law Dictionary, 597 (7th ed. 1999), contends that the letter is not an ex parte communication, since it was sent to all parties. Accepting this assertion as true, defendant does not explain how the letter is otherwise admissible. For example, the statements in the letter appear to be hearsay. See People v. Douglas, 362 Ill. App. 3d 65, 70 (2005) ("Hearsay is an out-of-court statement offered to prove the truth of the matter asserted"). A related problem is that, even though the State received a copy of the letter, it was unable to cross-examine Teas regarding its contents. Moreover, the letter was received well after the trial had concluded. Whether to reopen proofs is a matter committed to the discretion of the trial court. People v. Allen, 344 Ill. App. 3d 949, 953 (2003). Defendant makes no attempt to show that the trial court would have granted such a motion, particularly given the hearsay character of the letter.

To the extent the letter might serve as evidence of ineffectiveness, the relevant question is whether defense counsel's failure to bring out this evidence at trial constituted ineffectiveness. In this regard, defendant has not established that he was prejudiced by these purported failures. We see no reasonable probability that the outcome of the trial would have been different had this evidence been presented. Teas's letter addresses four main points: (1) Teas states that all autopsy findings indicate that Steven's injuries were at least five days old, particularly "[t]he specific characteristics of Steven's tissue reaction take at least several days to develop;" "[t]hey do not occur in 3 or even 4 days;" (2) Steven had aspirin and acetaminophen in his system, which undercuts the notion that he was feeling fine prior to the morning of February 8; (3) Steven lost four pounds between November 6, 2001, and February 8, 2002; (4) Steven was sedated before his abdomen was examined at Rush Presbyterian, which would have initially masked signs of abdominal distress and made the injury appear to be evolving to someone (i.e., Severin) not aware of the sedation.

The first point appears to be a recapitulation of Teas's trial testimony, which was, obviously, presented at trial. The remaining three points were called to the attention of the trial court during posttrial proceedings. Specifically, defendant raised them in his pro se motion alleging ineffective assistance of counsel, and the trial court expressly addressed two of them in its ruling on the motion. The trial court stated that the weight loss was not relevant in that Steven was in the custody of Dorothy Herron for most of the time between November 6, 2001, and February 8, 2002. As for the medication that may have affected observations of Steven's abdomen at Rush Presbyterian, the trial court stated "I think far more critical were the initial observations of the abdomen at Mount Sinai Hospital when Steven was initially brought in." Furthermore, we note that this evidence would have served only to impeach Severin regrading timing, and there was considerable other evidence on this

issue. Most notably, the trial court placed great weight on the testimony and observations of all of the treating medical personnel, including Munoz's opinion. Nurse Smith and Dr. Green both testified that they observed fresh bruises. Dr. Boykin testified to the impossibility of Steven eating a hotdog after sustaining these injuries. They must have occurred, therefore, after Steven last ate. According to defendant's statements to officers Figiel and Szalinski, Steven last ate at about 3 p.m., long after Kenyatta went to work. In fact, defendant told Kenyatta that Steven had been exhibiting signs of injuries for about an hour prior to the time Kenyatta returned from work on February 8. Kenyatta testified that Steven was fine when she left in the morning, but not when she returned after he was left in the exclusive care of defendant. Munoz confirmed that Steven could not walk, talk, eat, or drink after sustaining such injuries and that a head injury of this magnitude would preclude a lucid interval. Steven's amylase and lipase levels doubled following his admission to Mount Sinai. Numerous witnesses testified regarding injuries and bruises that continued to appear throughout the night of February 8. Severin observed that Steven's autoregulation system was still functioning during the evening of February 8. Mileusnic stated that she, as a pathologist, would defer to a treating physician on the issue of timing. Teas is not a treating physician.

In light of all of this evidence, we see no reasonable probability that undermining a portion of the basis for Severin's opinion would have led to a different result at trial. Similarly, that Steven had taken a pain killer at some point (we do not know when) certainly does not support an inference that Steven had sustained these massive injuries at an earlier time than is indicated by the weight of the evidence. As soon as Kenyatta observed the state that Steven was in upon her return from work, she wanted to take him to an emergency room, not give him an over-the-counter pain killer. Furthermore, two doctors testified that, after receiving such injuries, Steven would not have been able

to eat. If he could not eat, he could not swallow aspirin or acetaminophen. Thus, he must have taken these drugs prior to sustaining these injuries, and their presence in his system likely had nothing to do with the injuries that manifested themselves on February 8 at Mount Sinai and Rush Presbyterian. We further note that Kenyatta testified that Steven had a runny nose in the days leading up to his death, which could explain their presence. Again, defendant has not shown a reasonable probability that had this evidence been presented, a different outcome would have followed.

Finally, we note that even if we were to conclude that defendant had satisfied the prejudice prong of the ineffective-assistance-of-counsel test, we are not convinced that any purported error regarding the presentation of the material in Teas's letter would fit within the definition of ineffectiveness contained in the first prong of the test. See Ramirez, 371 Ill. App. 3d at 744. Except in extreme cases, decisions regarding what witnesses to call and what evidence to present are typically matters of trial strategy. People v. Enis, 194 Ill. 2d 361, 378 (2000); People v. Ward, 371 Ill. App. 3d 382, 433 (2007). Matters of trial strategy are "outside the scope of review for purposes of establishing incompetency of counsel." People v. Medrano, 271 Ill. App. 3d 97, 101 (1995).

Accordingly, we hold that trial counsel's failure to present the material in Teas's letter does not constitute ineffective assistance of counsel.

2. Lesser-Included Offense

Defendant next argues that trial counsel was ineffective for failing to argue that he was guilty of the lesser-included offense of involuntary manslaughter (720 ILCS 5/9--3 (West 2002)). Indeed, involuntary manslaughter is a lesser-included offense of first-degree murder with a less culpable mental state. People v. Robinson, 374 Ill. App. 3d 949, ___, 872 N.E.2d 73, 77 (2007). Though this case involved a bench trial, we believe that the best guidance for the resolution of this issue comes

from cases involving the decision to tender a jury instruction on a lesser included offense. Jury instructions place an issue before a jury and ask the jury to resolve it. See Drinkard v. Johnson, 97 F. 3d 751, 761 (5th Cir. 1996) ("The challenged instruction itself asks the jury to consider whether the defendant was temporarily insane (or, more specifically, 'did not know his conduct was wrong') as a result of intoxication 'at the time of the commission of the offense' "). In a bench trial, counsel places an issue before the court by arguing the issue. Insofar as jury instructions are concerned, it is well established that the decision to tender a jury instruction on a lesser-included offense is one of trial strategy. People v. Evans, 369 Ill. App. 3d 366, 383 (2006); People v. McIntosh, 305 Ill. App. 3d 462 471 (1999); People v. Balle, 256 Ill. App. 3d 963 971 (1993) ("Whether to tender an instruction on a lesser-included offense is almost always a question of trial strategy"). Similarly, in this case, counsel's decision not to argue defendant was only guilty of involuntary manslaughter was also a matter of trial strategy. As such, this purported failing by defendant's trial attorneys cannot support a claim of ineffective assistance of counsel. Medrano, 271 Ill. App. 3d at 101.

3. The Rule 604(d) Certificate

Defendant's final contention is that defense counsel was ineffective for filing a certificate pursuant to Rule 604(d) (188 Ill. 2d R. 604(d)) that contained "material misrepresentations." Defendant charges that 70 pages of the transcripts were not included in the record. The "material misrepresentation" defendant refers to is counsel's certification that he had reviewed the record, as set forth in the rule. See 188 Ill. 2d R. 604(d). The chief problem with defendant's argument is that Rule 604(d) does not apply in this case. The rule applies to appeals following a judgment entered on a guilty plea. People v. Willis, 313 Ill. App. 3d 553, 556 (2000). The instant appeal follows a trial. Thus, it is hard to see how defendant could be prejudiced by counsel's noncompliance with Rule

604(d) when he was not entitled to the protections of the rule in the first place. In any event, defendant does not attempt to explain how he was prejudiced despite the fact that the State called his attention to this point in its brief.

IV. CONCLUSION

In light of the foregoing, the judgment of the circuit court of Du Page County is affirmed.

Affirmed.

GROMETER, J., with CALLUM, J., concurring.

O'MALLEY, J., dissenting,

As the majority correctly notes, when a defendant makes a pro se claim of ineffective assistance of trial counsel, a trial court must conduct a threshold inquiry into the defendant's claim to determine if the claim lacks merit or touches upon trial strategy. If so, the claim should be dismissed; and, if not, the trial court should appoint counsel to argue defendant's claim. See slip op. at 44. I disagree with the majority's conclusion that the trial court did not err in its decision not to appoint new counsel to present defendant's claim of ineffective assistance of trial counsel.

First, though I do not dispute the majority's correct citation of the rule that a trial judge's decision not to appoint counsel will be disturbed "only if it is manifestly erroneous," (slip op. at 47, citing Young, 341 Ill. App. 3d at 382), I question the appropriateness of that standard. I trace the "manifestly erroneous" standard to the decision in People v. Jackson, 131 Ill. App. 3d 128, 140 (1985), in which the court, after summarizing the posttrial contention of ineffectiveness raised by the defendant, concluded by saying that the trial court's decision was not "manifestly erroneous." Nowhere in the case law do I find a rationale for applying this standard on appeal. While it is true that a trial court sits in a superior position to answer the above-described threshold questions, I am

not convinced that the trial court's answer is entitled to the high level of deference the "manifest error" rule implies. This type of posttrial motion and a petition under the Postconviction Hearing Act (725 ILCS 5/122--1 et seq. (West 2006)) are quite similar (with the admittedly important difference that the trial judge here, unlike a typical postconviction judge, had recently presided over the trial), and it is well-established that a reviewing court will determine de novo whether a trial court correctly dismissed a postconviction petition without appointing counsel to argue a defendant's claim (eg., People v. Coleman, 183 Ill. 2d 366, 388-89 (1998)). In my view, this similarity warrants more searching review than review for "manifest error."

However, even under the "manifest error" standard, I disagree with the trial court's decision here. After defendant's conviction, Dr. Teas sent a letter detailing what she viewed as relevant exculpatory medical evidence that was not adduced at trial: she wondered why Steven had aspirin in his blood if he had been feeling well; she wondered why Steven had lost four pounds in the four months prior to his death, she noted that some of the drugs doctors administered to Steven may have caused his abdomen to be soft, and she noted that it was not uncommon for symptoms of children's abdominal injuries to be delayed as much as 2-3 days after the injury. To me, defendant's allegation that counsel ignored these facts presents a potentially meritorious claim of ineffective assistance sufficient to withstand the trial court's threshold inquiry. Though counsel generally asserted that any challenged decisions were matters of trial strategy, counsel admitted being unaware of at least one of these medical facts despite Dr. Teas's assertion that it was in the medical records. See slip op. at 42 ("Evidence of aspirin and Tylenol was contained in Teas's ex parte letter, and thus, though he was now aware of it, he could no longer use the evidence"). As for the remaining facts, I believe they raise

enough doubt that defendant should have an opportunity to argue their exclusion was not a matter of reasonable trial strategy.

I would remand this cause for the trial court to appoint counsel to argue defendant's claims.

Shaku S. Teas, M.D.
1123 Ashland
River Forest, IL 60305

August 26, 2004

Honorable Ann Jorgenson
DuPage County Court

Re: Randy Liebich

Dear Judge Jorgenson:

I was saddened to hear that you found Randy Liebich guilty for the death of Steven Quinn. I am concerned that your determination may have been based, at least in part, on testimony that strongly suggested that Steven's injuries occurred on February 8, 2002.

The autopsy findings indicate that *all* of Steven's injuries were at least five days old at the time he died. The healing and repair process, identifiable when portions of his brain (subdural hemorrhage) and abdominal (pancreas and gastrointestinal tract) injuries are examined under the microscope, was well established at the time he died. The specific characteristics of Steven's tissue reaction take at least several days to develop. They do not occur in 3 or even 4 days. Five days or 120 hours from the time of death would indicate that the injuries occurred on February 6, 2002 or earlier

Dr. Mileusnic indicated in three different sections of her written autopsy report that the injuries were at least five days old. The Assistant State's Attorney was not even aware of Dr. Mileusnic's determination until he read my report. I had to show him where Dr. Mileusnic had recorded her observations regarding the timing of the injuries when he called me to inquire about it.

There are significant findings in the medical records that were not discussed at trial. For example, why did Steven Quinn have acetaminophen (Tylenol) and salicylates (Aspirin) in his blood if he had been feeling well until the morning or afternoon of February 8th? Why was his weight more than four pounds less on February 8, 2002 than it was on November 6, 2001? (His weight on November 6, 2001 was 35.5 pounds and on admission to Presbyterian- St. Luke's Hospital, the weight was recorded as 14 kilograms or 30.8 pounds.) He was sedated with Versed and Ativan, and paralyzed with succinylcholine, before his abdomen was examined at Presbyterian-St. Luke's. The sedation and paralysis are the reasons that he did not have signs of an acute abdomen and that his abdomen was soft, even though he had peritonitis at the time. It is

not unusual for signs and symptoms of abdominal injury in children to be delayed for as much as 2-3 days after the injury, especially if there is no associated blood loss. In fact, children with documented accidental blunt abdominal injuries often have misleading clinical signs on initial presentation to medical personnel. The body does not differentiate in its reaction and responses to accidental or inflicted injury. Forensic pathologists learn and extrapolate timing and mechanisms of injury from known accidental injuries.

Timing of injuries is difficult and not precise. However, the *best method* available at this time is histological examination, that is reaction of the body to the injury at a cellular level. The amount of reaction and healing seen in the dura mater (brain) and gastrointestinal tract (small bowel and pancreas) tissues of Steven Quinn are inconsistent with the injuries having occurred on the morning or afternoon of February 8, 2002.

Finally, I am enclosing copies of the medial records where the transfer dates and times were incorrect, and the anesthesiology records indicating the incorrect date of the craniotomy. The two copies of the CT scan read by the radiology department do not indicate that the subdural hemorrhage was "large". The term "large" is perpetuated in the physician notes from Rush Hospital. In fact, the operative report states that " there was *no large* subdural blood accumulation"

Thank you for considering the above facts.

Respectfully,

Shaku S. Teas M.D.

cc: Ricky Holman/ John Casey
Joseph A. Ruggiero
Darinka Mileusnic

OFFICE OF THE MEDICAL EXAMINER
COUNTY OF COOK, ILLINOIS

REPORT OF POSTMORTEM EXAMINATION

NAME Steven Quinn **CASE NO.** 202 of February 2002

AGE 2 ½ **RACE** Black **SEX** Male **DATE OF DEATH** February 11, 2002

ADDRESS 714 S. Independence **DATE EXAMINED** February 12, 2002 (8:50am)

CITY & STATE Chicago, Illinois **EXAMINED BY** Darinka Mileusnic, M.D.

EXTERNAL EXAMINATION:

The body is received unclothed. A disposable diaper is present over the hips and buttocks.

The body is that of a well developed and well nourished male child appearing the stated age of 2 ½ years. The body measures 39.0 inches in length, which is at the 97th percentile for age. The body weighs 44 pounds, which is above the 97th percentile for age.

The body is cold to touch. Rigor mortis is present to an equal extent in all joints. Postmortem lividity is fixed and well developed in the posterior dependent portions of the body. The body is in a moderate to severe state of anasarca which is manifested by bloating of the face and swelling of the subcutaneous tissue and the viscera due to accumulation of fluid.

The crown/rump length is 25.0 inches. The head circumference is 20.5 inches. The chest circumference is 21.2 inches. The abdominal circumference is 20.2 inches.

The head hair is black and very short. The scalp is very soft and edematous. The eyes are closed. The eyelids are edematous. The irides are brown. The cornea are cloudy. The sclerae and conjunctivae are edematous. There are no petechiae in the sclerae or the conjunctivae of the eyes. The external ears are well formed. The skeleton of the nose is intact. The lips and frenula are atraumatic. The teeth are natural, deciduous and in good repair. The hard palate is intact. The neck is not hypermobile. On the right posterior neck, there is a slanting scar, 0.8 inch long.

The body is clean. The chest is symmetrical. The abdomen is slightly protuberant. The external genitalia are of a normal

EXTERNAL EXAMINATION: (Continued)

circumcised male child. On the right scrotum, there are multiple superficial abrasions with no surrounding vital reaction consistent with postmortem artifact. The scrotum is mildly to moderately swollen.

The anterior and posterior upper extremities are without special note. On the back of the left shoulder, there is an oval pigmented scar, 0.3 inch in greatest dimension. On the dorsum of the left hand, there are two pale oval scars, each measuring 0.2 inch in greatest dimension. The fingernails are short and clean.

Except for bruising, the back and buttocks are without special note. On the left lower back, in the lumbar region, there is a cluster of six small irregular pigmented scars which range in size from 0.1 to 0.4 inch and cover an area of which measures 2.0 x 1.5 inches. The anus is patent and displays no evidence of trauma.

On the anterior and posterior lower extremities there are scattered contusions. On the medial left knee, there is a slanting scar, 0.4 inch in greatest dimension. On the back of the proximal right leg, there is an oval hyperpigmented discoloration, 0.8 x 0.3 inch. The soles of the feet are without special note. The toenails are short and clean.

EXTERNAL EVIDENCE OF INJURY:

1. On the right forehead, there is a healing purple/brown contusion, 2.0 inches in greatest dimension.

2. On the right and proximal nasal bridge, there is a healing purple/brown contusion, 0.25 inch in greatest dimension.

3. On the left side of the head, posterior and slightly superior to the pinna of the left ear, there is a purple/brown healing contusion, 0.9 inch in greatest dimension.

4. On the superior and inner aspect of the pinna of the left ear, there are two healing purple/brown contusions, 0.2 and 0.3 inch in greatest dimension.

5. On the right back of the head, there is a healing abrasion associated with underlying purple/brown contusion, 0.4 inch in greatest dimension.

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EXTERNAL EVIDENCE OF INJURY: (Continued)

6. On the right temple, there are two faint healing purple/brown contusions which measure 0.2 and 0.7 inch in greatest dimension.

7. On the right back of the head, inferior and posterior to the pinna of the right ear, there is an ill defined healing purple/brown bruise, 1.6 x 0.7 inches in greatest dimension.

8. On the right neck, there is an irregular superficial 0.4 inch abrasion surrounded by minimal skin reaction and appearing fresh, consistent with a treatment artifact.

9. On the posteromedial aspect of the right wrist, there is a purple/brown healing contusion, 1.0 x 0.5 inch in greatest dimension.

10. On the lower back and extending into the lumbar region and slightly to the left, there is a cluster of five purple/brown bruises which range in size from 0.4 to 1.3 inches and cover an area of 4.0 x 2.7 inches.

11. On the medial and distal right thigh, there is a cluster of ten purple/brown bruises which range in size from 0.2 to 0.4 inch and cover an area of 4.0 x 3.0 inches.

12. On the anterior proximal right leg, there are two oval purple/brown bruises which measure 1.2 x 0.8 inches and 0.4 inch in greatest dimension.

13. On the medial aspect of the proximal right leg, there are three purple/brown bruises which range in size from 0.2 to 0.4 inch and cover an area of 0.8 x 0.7 inch.

14. On the medial and proximal left thigh, there is a cluster of four purple/brown bruises which range in size from 0.3 to 0.4 inch and cover an area of 1.4 x 0.9 inches.

15. On the medial left ankle, overlying the medial malleolus, there is an intense purple/brown bruise, 1.0 x 0.7 inch

MR 360

EXTERNAL EVIDENCE OF INJURY: (Continued)

16. On the medial aspect of the left foot, radiating from the medial malleolus distally and anteriorly, there are seven linear purple/red bruises which range in length from 1.2 to 1.7 inches.

17. On the distal aspect of the dorsum of the left foot, proximal to the left great toe, there is a 0.2 inch abrasion surrounded by a purple/blue bruise.

INTERNAL EVIDENCE OF INJURY:

1. On reflecting the scalp, over the left frontoparietal region, there is a healing subgaleal hemorrhage, 3.0 inches in greatest dimension.

2. On opening the cranial cavity, there is a residual bilateral subdural hemorrhage. A small amount of residual clotted blood is present over the right hemisphere. A larger amount of clotted subdural blood is present over the left hemisphere, medially, along the falx, 4.0 x 1.0 inches and 0.3 inch thick. Approximately 30 grams of clotted blood are recovered from the left side. The subdural hemorrhage extends into the interhemispheric fissure and caudally along the entire length of the spinal cord.

3. Over the bilateral cerebral hemispheres and the cerebellum, there is diffuse subarachnoid hemorrhage.

4. On the lateral aspect of the right cerebral hemisphere, there are contusion hemorrhages and necrosis. The rest of the brain is severely edematous with evidence of subtentorial and cerebellar tonsillar herniation.

5. On opening the abdominal cavity, over the proximal loops of the small bowel and adjacent mesentery, there are diffuse hemorrhages and superimposed localized visceral peritonitis. Fibrin deposits are seen over the proximal several inches of the jejunum and the adjacent mesentery. Approximately 7.0 inches of the proximal jejunum are hemorrhagic and necrotic with a 0.1 inch small perforation covered by fibrin which is located at the mesenteric aspect of the bowel. Another focus of visceral peritoneal hemorrhage and the

MR 361

INTERNAL EVIDENCE OF INJURY: (Continued)

adjacent soft tissue is found in the area of the terminal ileum and the cecum. Blood stained fecal material is present in the large bowel.

6. In the area of the head and body of the pancreas, there is thick peripancreatic hemorrhage. Focally in the pancreatic tissue itself, there are small hemorrhages. Surrounding the body of the pancreas, there are small clusters of bright yellow calcium soaps, possibly consistent with early traumatic pancreatitis.

7. On the inferior aspect of the right lobe of the liver, close to the anterior margin and the fundus of the gallbladder, there is a healing subcapsular hematoma, 1.0 x 0.4 inch.

8. In the right and left retroperitoneum, there is a thin layer of hemorrhage which tracks downward in the pelvis and then along the right spermatic cord into the scrotum. The right and left testicle are intact.

9. The section of the skin and the subcutaneous tissue on the front and the back of the body reveals multiple foci of healing subcutaneous hemorrhage:

   A. In the right anterolateral arm, there is focal hemorrhage.

   B. On the lateral aspect of the right wrist, there is extensive hemorrhage.

   C. On the dorsum of the left hand, there is focal hemorrhage.

   D. On the lower back, in the lumbar region, crossing the midline, there are multiple confluent foci of healing contusions.

   E. Over the right and the left buttock and extending into the right hip, there are multiple and focally confluent foci of subcutaneous healing contusions.

   F. On the posterior right thigh, there is a focal subcutaneous hemorrhage

MR 262

INTERNAL EVIDENCE OF INJURY: (Continued)

G. On the anterior right leg, there are three foci of deep subcutaneous hemorrhage.

H. On the left medial ankle and foot, there is extensive hemorrhage.

EVIDENCE OF MEDICAL TREATMENT:

1. The head is wrapped in white dressing. On the right aspect of the scalp, there is a letter C-shaped stapled surgical incision, 9.5 inches long. The incision is protected with adhesive tape. The surrounding scalp is bruised and markedly edematous. On reflecting the scalp, there is extensive subgaleal hemorrhage associated with the surgical site, 5.0 inches in greatest dimension. The craniotomy has been left open, creating a 4.0 inch defect in the skull. The dura is widely cut open and hemorrhagic, necrotic and diffluent brain parenchyma has been oozing through the opening in the skull and under the scalp. A large amount of hemorrhagic pasty brain substance is collected from the surface of the skull.

2. The right portion of the scalp has been shaved. In the vicinity of the surgical incision and within the shaved area, there is a smaller incision with in-situ intracranial pressure monitor.

3. A naso-gastric tube is present in situ and inserted through the left nostril.

4. An endotracheal tube is present in situ and secured with adhesive tape around the mouth. Associated with the endotracheal tube, there is an ill defined bruise on the lower lip in the midline.

5. In the right inguinal region, there are multiple needle punctures with two in situ catheters.

6. In the left inguinal region, there are multiple needle punctures.

7. A pediatric Foley catheter is present in situ.

8. A rectal probe is present in situ.

MM 263

EVIDENCE OF MEDICAL TREATMENT: (Continued)

9. On the right upper chest, in the infraclavicular region, there are multiple needle punctures.

10. On the right lower neck, there is an irregular abrasion, possibly associated with the medical treatment.

11. The wrist and ankles have been loosely tied in the hospital for transportation purposes. The underlying skin demonstrates criss-crossed shallow furrows.

12. On the dorsum of the right hand, there is a needle puncture.

13. On the right lower abdomen, there is a horizontal 1.3 inch surgical incision with in situ drain. The incision is protected by numerous layers of gauze which are secured around the lower abdomen with adhesive tape. On the flank region, the tape leaves small superficial impressions and abrasion.

INTERNAL EXAMINATION:

BODY CAVITIES: The body is entered by a Y-shaped incision. All organs are present in their ordinary anatomic positions and present their ordinary anatomic relationships. In the upper abdomen, there is localized visceral peritonitis and hemorrhage due to blunt abdominal trauma. In the retroperitoneum and tracking down in the pelvis and the right scrotum, there is hemorrhage. Pleural, pericardial and peritoneal cavities display an increased amount of transudate.

NECK ORGANS AND TONGUE: The anterior muscles of the neck reveal no evidence of hemorrhage. The cartilages of the larynx, including the epiglottis are intact. The hyoid bone is intact. Examination of the tongue reveals no evidence of injury.

RESPIRATORY SYSTEM: The tracheal and bronchial mucosa are pale and mildly edematous. The right lung weighs 220 grams (expected average weight for age 88 to 89 grams). The left lung weighs 181 grams (expected average weight for age 76 to 77 grams). The pleural surfaces of the lungs are smooth and pale. The lungs are pale tan anteriorly and purple/red posteriorly. The lungs are heavy and hypocrepitant. On cut section, the lung parenchyma displays marked posterior congestion and generalized severe

MR 764

INTERNAL EXAMINATION: (Continued)

edema. No nodules or granulomas are palpable in either lung. The pulmonary arteries are unremarkable.

CARDIOVASCULAR SYSTEM: The heart weighs 97 grams (expected average weight for age 56 to 59 grams). The epicardial surface is smooth and epicardial fat is adequate. The superior and inferior venae cavae return to the right atrium. There are no atrial or ventricular septal defects. The ductus arteriosus is closed. The coronary ostia are present in their ordinary anatomic locations. The coronary arteries pursue their ordinary anatomic course. The valves of the heart and great vessels are without special note. No congenital anomalies are noted. The myocardium displays no focal pathologic change. The pulmonary veins return to the left atrium. The great vessels at the base of the heart are unremarkable. The aorta displays no abnormalities.

HEPATOBILIARY SYSTEM: The liver weighs 578 grams (expected average weight for age 418 to 516 grams). The surface of the liver is red/brown and smooth. The margins of the liver are sharp. On cut section, the hepatic parenchyma is uniformly red/brown. The gallbladder and biliary tract pursue their ordinary anatomic course and display no evidence of pathologic change.

HEMOLYMPHATIC SYSTEM: The spleen weighs 54 grams (expected average weight for age 37 to 39 grams). The spleen is purple/blue and soft with a wrinkled capsule. On cut section, the splenic parenchyma is red/brown and uniform. No abnormal lymphadenopathy is noted. The thymus gland is uniformly pink/tan and weighs 44 grams. On cut section, the thymic parenchyma is tan with no evidence of petechia.

GASTROINTESTINAL SYSTEM: The esophagus is without special note. The stomach contains approximately 40 ml of green thick liquid material. The mucosa of the stomach has the usual rugosity and displays scattered petechiae. The duodenum, small and large intestines show evidence of contusions and focal peritonitis as already described. The appendix is present and shows no pathological changes.

MM 265

GENITOURINARY SYSTEM: The right kidney weighs 56 grams (expected average weight for age 48 to 58 grams). The left kidney weighs 62 grams (expected average weight for age 49 to 56 grams). The kidneys are red/brown with smooth and lobulated surfaces. On cut

INTERNAL EXAMINATION: (Continued)

section, the renal parenchyma is red/brown and congested with well demarcated corticomedullary junctions. The renal calyces, pelves, ureters, and urinary bladder with prostate display no abnormalities. Traces of urine are present in the urinary bladder.

ENDOCRINE SYSTEM: The thyroid gland displays no abnormalities. The pituitary gland is soft and hemorrhagic due to head trauma. The pancreas is firm with surrounding and focal intraparenchymal hemorrhages. The adrenal glands are soft and slightly dark yellow discolored.

CENTRAL NERVOUS SYSTEM: The scalp displays no lacerations. There is a large hematoma associated with the surgical procedure. On reflecting the scalp, there is right-sided subgaleal hemorrhage associated with the surgical procedure as well as a healing left-sided subgaleal hemorrhage which is located over the left frontoparietal region. The skull is intact. On entering the cranial cavity, there is residual bilateral subdural hemorrhage already described. The leptomeninges show diffuse subarachnoid hemorrhage. The brain weighs 1438 grams (expected average weight for age 1141 to 1191 grams). The brain is very soft, hemorrhagic and focally necrotic and falls apart easily on removal from the cranial cavity. There is severe brain edema as manifested by flattening of the gyri and uncal and cerebellar tonsillar necrosis. The spinal cord is taken out and displays diffuse subdural hemorrhage which is continuous with the intracranial bleed. The brain with the covering and the spinal cord are saved for further neuropathological examination. Both eyes have been enucleated and demonstrate extensive optic nerve sheath hemorrhages. The eyes are sent to the eye pathologist for further examination.

MUSCULOSKELETAL SYSTEM AND RADIOLOGIC EXAMINATION:

1. Ribs, long bones and vertebrae are intact to palpation.

2. Perimortem and postmortem x-rays of the entire body fail to reveal any overt skeletal injuries. The x-rays are sent for additional evaluation by a radiologist.

3. Incisions into the skin, subcutaneous tissue and the muscles of the anterior and posterior body and extremities reveal subcutaneous hemorrhages as already described.

MR 266

ANATOMIC DIAGNOSES:

1. Healing contusions of the scalp, back, buttocks, and the extremities, multiple.
2. Subgaleal hemorrhage.
3. Subdural hemorrhage.
4. Subarachnoid hemorrhage.
5. Cerebral contusions, hemorrhages and necrosis.
6. Brain edema, severe.
7. Small bowel contusion, necrosis and perforation.
8. Mesenteric hemorrhage.
9. Peritonitis, localized.
10. Pericecal hemorrhage.
11. Peripancreatic and pancreatic hemorrhage with superimposed acute traumatic pancreatitis.
12. Subcapsular hematoma of the liver.
13. Blood stained fecal material.
14. Retroperitoneal hemorrhage.
15. Optic nerve sheath hemorrhage, bilateral.
16. Anasarca.

OPINION:

This 2 ½ year old Black male child, STEVEN QUINN, died of multiple injuries due to blunt force trauma which was a consequence of child abuse.

MANNER OF DEATH: Homicide

DARINKA MILEUSNIC, M.D.
Deputy Medical Examiner

February 28, 2002

DM:lcc
2/21/02

MR 267

OFFICE OF THE MEDICAL EXAMINER
COUNTY OF COOK, ILLINOIS

NEUROPATHOLOGY

GROSS & MICROSCOPIC DESCRIPTIONS

NAME Steven Quinn **CASE NO.** 202 February 02

MEDICAL EXAMINER: Dushka Milijanic, M.D., PhD.

GROSS DESCRIPTION:

BRAIN AND SPINAL CORD: The brain with the dura and spinal cord are examined in fixed state. The spinal cord is slightly distorted after fixation and is approximately 6.5 inches long. As already described during autopsy, a significant amount of subdural blood is found diffusely from the rostral all the way to the caudal segment of the spinal cord. No other abnormalities are noted. Residual clotted blood is seen on the inner aspect of the dura. In the dural sinuses, there is clotted blood. The brain weighs 1545 grams in the fixed state. The brain itself is severely deformed due to underlying edema and necrosis. Diffuse residual subarachnoid hemorrhage which remains after washing of the fixed brain covers the bilateral convexities, the brain stem, and the cerebellum. Due to underlying hypoxic-ischemic changes in the [illegible] of the brain, the central structures are poorly fixed. There is extensive necrosis of the midline structures, such as the corpus callosum, fornix, septum, and brain stem. On the left cerebral hemisphere in the region of the left posterior frontal lobe, there is approximately 7 cm in diameter area of contusion necrosis. The contusion necrosis involves the cortex and the underlying subcortical white matter. The base of the brain, specifically, the temporal lobes with the bilateral hippocampi, are almost completely necrotic and their structures cannot be discerned during sectioning.

Serial sections are made through the cerebrum, cerebellum, and the brain stem. There is a striking extensive change associated with hypoxic and ischemic damage. The bilateral basal ganglia and thalami fall out during sectioning due to surrounding necrosis. Distended and congested blood vessels and numerous petechiae are seen through the white matter, especially the

81-6 MP

ORIGINAL

MR 917

centrum semiovale and the basal ganglia with the thalami. As already mentioned, the proximal brain stem, specifically the mesencephalon and the rostral pons are necrotic. The distal pons and the medullae display midline petechiae. The white matter of the tegmentum of the pons and the cerebellum is gray/brown discolored. Cerebellar folia appear atrophic and necrotic throughout. The central cerebellar nuclei cannot be discerned.

ANATOMIC DIAGNOSES:

1. **BRAIN EDEMA, SEVERE.**

2. **SUBDURAL HEMORRHAGE.**

3. **DIFFUSE HYPOXIC/ISCHEMIC DAMAGE TO THE CEREBRUM, CEREBELLUM, AND THE BRAIN STEM WITH SUPERIMPOSED RESPIRATOR INDUCED CHANGES AND NECROSIS.**

4. **CONTUSION NECROSIS OF THE LEFT CEREBRAL HEMISPHERE.**

03/08/02

Dr. M/kl
03/1/02

ORIGINAL

MR918

Office of the Medical Examiner
County of Cook

MICROSCOPIC EXAMINATION

Case No. 02-2-02 Pathologist Darinka Mileusnic, M.D., Ph.D.

Name Steven Quinn

Heart: Post traumatic ischemic changes + secondary changes due to hypo

Lung: Congestion + edema; bronchopneumonia;

Liver: Numerous glycogen nuclei; fine granular basophilic hemosiderin surrounding hepatocyte nuclei

Pancreas: Hemorrhage + numerous chylomicrotic cells c/c fat necrosis in the peripancreatic connective tissue

Spleen: Changes c/c early hypo

Kidney: NMHP's

CNS: Subdural + subarachnoid hemorrhage c admixed macrophages dep J.

Other: Pericolic + mesocolic hemorrhage; mesenteric hemorrhage
Thymic petechiae
Thyroid WNL; Adrenals WNL

D.M.
Initials

Healing subpleural hemorrhages c few admixed macrophages + other mononuclear cells c dep J

Subconjunctival + subcutaneous hemorrhages c admixed macrophages dep J

Intact testicles
Small intestinal wall hemorrhage + transmural inflammation c perforation (traumatic)

Office of the Medical Examiner
County of Cook

MICROSCOPIC EXAMINATION

Case No. 202-2-02 Pathologist Darinka Mileusnic, M.D., Ph.D.

Name Steven Quinn

Heart:

Lung:

Liver:

Pancreas:

Spleen:

Kidney:

CNS: Edema + congestion
SAH + focal perivascular hemorrhages
Hypoxic-ischemic changes & vascular axonal injury
(most prominent in the cerebellum)

Other: Focal axonal necrosis + focal axonal damage in corpus
callosum
Early gliosis surrounding the foci of contusion; less prominent
generalized gliosis due to hypoxic-ischemic changes

D.M.
Initials

Recent SAH + SDH c signs of organization -
absence of infiltration of mononuclear cells,
mainly lymphocytes and scattered fibroblasts (@ 5 days old)
Perivascular discoloration SDH

MED 292 (R)

...member of Sinai Health System

SINAI
Sinai Hospital

(773) 542-2000 - Emergency Department (773) 257-6438

EMERGENCY SERVICES

QUINN, STEVEN
000105638837 2Y 09M M
Green, Paula B. 04/17/99
Non-Staff Physician
Reg: 02/08/02 MR#: 001389281

NAME	DATE	TIME	ACCT. NO
QUINN, STEVEN	02/08/02	1821	000105638837

SEX	BIRTHDATE	AGE	PRIMARY CARE PHYSICIAN	ARRIVAL MODE
M	04/17/99	2Y 09M	Non-Staff Physician	Parent Arms

TIME 6:40
C.C. [illegible]
HPI
*Location [illegible]
*Quality [illegible]
*Severity [illegible]
*Duration [illegible]
*Timing [illegible]
*Context [illegible]
*Modifying Factors [illegible]
*Associated Symptoms [illegible]

ROS	NORMAL	ABNORMALITY
Constitutional	✓	
Eyes	✓	
Ears, Nose, Mouth, Throat	✓	
Cardiovascular	✓	
Respiratory	✓	
Gastrointestinal	✓	
Genitourinary	✓	
Musculoskeletal	✓	
Skin	✓	
Neurological		
Psychiatric	✓	
Endocrine		
Hematologic/Lymphatic		

ROS	NORMAL	ABNORMALITY
Allergic/ Immunologic		
Other Systems Negative		

Med Ø
All [illegible]

FH: DM
SH:
PMH: Ø [illegible]

PHYSICAL EXAMINATION
EYES [illegible] PERRL 3-2 [illegible] lateral gaze
ENT / NECK [illegible] C-spine immob. + gag [illegible]
RESPIRATORY / CARDIOVASCULAR equal B/B grunting c periods of whining [illegible]
BREASTS [illegible]
GENITOURINARY / LYMPHATIC back [illegible] buttocks hyperemic
MUSCULOSKELETAL / SKIN [illegible]
NEURO PSYCHIATRIC Periods of grunting c lateral eye gaze, B UE/LE rigid posturing c
MEDICAL DECISION MAKING periods of flaccid, Ø tonic/clonic movement.

Signature: [illegible]

ATTENDING / CONTINUATION NOTE:
[illegible] intubated [illegible]
[illegible] equal B/B, [illegible] c good placement.
[illegible]
[illegible] to report possible abuse & Police department notified
(DCFS [illegible])
[illegible]
[illegible]

Signature: [illegible] MR 327

FINAL DIAGNOSIS
[illegible] (2) Ped ET intubation (3) [illegible]

MD CONTACT	CALLED/RESPONDED	MD CONTACT	CALLED/RESPONDED	MD CONTACT	CALLED/RESPONDED
[illegible] 6:40	[illegible] 6:40	[illegible] 6:40			

CONDITION ON DISCHARGE: ☐ GOOD ☐ FAIR ☒ POOR ☐ DECEASED   DISPOSITION: ☐ HOME ☐ ADM ☐ OBS ☒ TRANS ☐ DOA ☐ DIED ☐ AMA ☐ LWOT

ADMIT TO DR.	RM #	NURSE SIGNATURE	TIME OF DISCHARGE	ATTENDING PHYSICIAN SIGNATURE
		[illegible]		[illegible]

MED 6

RUSH

RECORD OF OPERATION

Original

PATIENT NAME: QUINN ,STEVEN

PROCEDURE DATE: 2/8/02

MED. REC. NO.: 550-05-84

ATTENDING SURGEON: BASS, KATHRYN D M.D.

PAT. HOSP.SERV: WPI

ASSOC. SURGEONS: -NONE-

ASSISTANTS: TREDWAY, TRENT L M.D.

2-11-02

PREOPERATIVE DIAGNOSIS:

Multiple trauma with severe head injury.

POSTOPERATIVE DIAGNOSIS:

Multiple trauma with severe head injury.

PROCEDURE:

Abdominal drain placement.

DESCRIPTION OF PROCEDURE:

ANESTHESIA:
None.

INDICATIONS:
The patient is a 3-year-old who was brought to the Rush Children's Hospital from Mt. Sinai for emergent neurosurgical intervention of a closed head injury. He had decorticate positioning at Mt. Sinai and decerebrate positioning on arrival at Rush. He was brought to the operating room emergently under Dr. Munoz' care for an evacuation of a right-sided subarachnoid and subdural bleed. Following the procedure, the patient returned to the ICU with fixed and dilated pupils. He currently has no response to pain, and has had an apnea test which showed no respirations at 14 minutes.

A request for a peritoneal drain placement was initiated by the ICU team in order to maintain appropriate ventilation.

PROCEDURE:
The patient was prepped and draped in a sterile fashion in the right lower quadrant. A 3-cm incision was made through skin, subcutaneous tissue, and fascia of the external oblique. The muscle was then spread. The internal oblique was incised and spread. The peritoneum was opened and a large amount of serosanguinous pink fluid was drained. A total of 500 cc drained. There was no evidence of succus. A 1-inch Penrose was inserted, and three 3-0 silks were used to secure the Penrose bilaterally. A 4 x 4 dressing was placed.

The patient tolerated the procedure well. No response occurred to painful stimuli. No improvement occurred in ventilation.

The patient remained under the care of the ICU team with a preliminary diagnosis of brain death. ROBI will be contacted for organ donation.

MR 614

MED 84A

RUSH

RECORD OF OPERATION

Original

PATIENT NAME: QUINN ,STEVEN PROCEDURE DATE:2/8/02 MED. REC. NO.: 550-05-84

ATTENDING SURGEON: BASS, KATHRYN D M.D. PAT. HOSP.SERV: WPI

SURGEONS: -NONE-

ASSISTANTS: TREDWAY, TRENT L M.D.

---

cc: Paul N. Severin, M.D.
Lorenzo F. Munoz, M.D.

Dictated by: BASS, KATHRYN D M.D.

Electronically Approved by:BASS, KATHRYN D M.D.
On:Feb 20, 2002

RU 140/60043
D:02/09/2002
T:02/12/2002

MR615

MED 84B

RUSH

RECORD OF OPERATION

Original

PATIENT NAME: QUINN ,STEVEN PROCEDURE DATE: 2/8/02 MED. REC. NO.: 550-05-84

ING SURGEON: MUNOZ, LORENZO F M.D. PAT. HOSP.SERV: WPI

C I SURGEONS: -NONE-

ASSISTANTS: TREDWAY, TRENT L M.D.

05224

Pt. Died 2-11-02

PREOPERATIVE DIAGNOSIS:

1. Right subdural hematoma.
2. Diffuse subarachnoid hemorrhage.
3. Severe head trauma.
4. Decerebrate posturing.

POSTOPERATIVE DIAGNOSIS:

1. No subdural hematoma found.
2. Diffuse subarachnoid hemorrhage.
3. Severe head trauma.
4. Decerebrate posturing.

PROCEDURE:

Right frontal temporal parietal craniectomy for decompression of severely edematous brain.

DESCRIPTION OF PROCEDURE:

INDICATIONS:
This is the case of a 2.5 year old boy who was emergently transferred to our service at Rush after he presented to Mt. Sinai Hospital with mental status changes and abnormal posturing. At that time he was readily intubated. A CT scan was performed which revealed the aforementioned findings. For that reason, we will transfer the patient immediately to Rush for further care.

The patient came to the pediatric intensive care unit where good vascular access and an arterial line were procured. At that time the patient was having decerebrate posturing but the pupils were equal, round and reactive to light. Upon looking at the CT scan, there was a question of a right frontal parietal subdural hematoma. However, it was also thought that this could also be a rather profuse subarachnoid hemorrhage. Furthermore the patient had ecchymosis in the right frontal parietal part of his head. For that reason, an after talking to the patient's mother and grandmother, we brought the patient to the operating room in order to undergo the aforementioned emergency procedure.

TECHNIQUE:
The patient was brought to the operating room and the patient was placed in the supine position after the head was secure in a horseshoe head holder. This was done after the usual pressure points were addressed. Upon shaving the right side of the head, we were able to readily visualize areas of ecchymosis located in the right frontal parietal area.

MRC

PATIENT NAME: QUINN ,STEVEN PROCEDURE DATE:2/8/02 MED. REC. NO.: 550-05-84

ATTENDING SURGEON: MUNOZ, LORENZO F M.D. PAT. HOSP.SERV: WPI

CO-SURGEONS: -NONE-

ASSISTANTS: TREDWAY, TRENT L M.D.

At this point, we proceeded to perform a standard trauma flap on the right side. Raney clips were then applied to the scalp and the scalp was then reflected anteriorly. Using the Midas M8, multiple bur holes were placed along the circumference of the scalp flap. The underlying dura was then readily coagulated. Using the Midas B1 we then proceeded to perform the aforementioned large right frontal temporal parietal craniectomy.

Upon elevating the bone flap, which was done without lacerating the underlying dura, we were able to realize that the brain appeared to be under tremendous pressure. The intraoperative judgment was made that in order to give this child a chance we will proceed to perform an opening of the dura throughout in order to perform a decompressive craniectomy.

Upon doing this we found there was no large subdural blood accumulation. Although there was some subdural blood, it did not appear to be as severe as it appeared to have been on the CT scan that the patient brought in from Mt. Sinai Hospital. However, there was a large and significant amount of subarachnoid hemorrhage throughout indicating a severe injury.?? Also in spite of our best medical efforts in the operating room the brain was severely swollen. At this point after having performed the aforementioned decompression, we then proceeded to close the scalp using a combination of inverted 3-0 Vicryl for subcutaneous tissue and a running 2-0 and 3-0 nylon for the skin.

secondary to shaken

At the end of the procedure, the instrument and sponge count was complete. For the estimated blood loss please see anesthesia note.

In summary this patient had a severely swollen brain with a large amount of subarachnoid hemorrhage and a small thin subdural hematoma. The brain appeared to be tremendously edematous.

Dictated by: MUNOZ, LORENZO F M.D.

Electronically Approved by:MUNOZ, LORENZO F M.D.
On:Feb 12, 2002

RU 104/60084
D:02/09/2002
T:02/09/2002

MR617

RUSH

DISCHARGE SUMMARY

Original

PATIENT NAME: QUINN ,STEVEN

MEDICAL RECORD NO: 550-05-84

ADMISSION DATE: 02/08/02

DISCHARGE DATE: 02/11/02

ATTENDING PHYSICIAN: SEVERIN, PAUL N M.D.

CHIEF COMPLAINT: Abnormal breathing.

HISTORY OF PRESENT ILLNESS: The patient is a 2 1/2-year-old male with no significant past medical history, who was initially brought to Mt. Sinai Hospital by his mom secondary to abnormal breathing and behavior. Mom reportedly left for work at 10 a.m. and returned at 6 p.m. on February 08, 2002. She had left her son in her boyfriend's care, for the first time in several days. She was returning to work after being postpartum for approximately 2 weeks. On arrival at home she found that the patient was minimally responsive with abnormal breathing. Per mom's boyfriend, the patient had "choked on a hotdog" approximately 12 p.m. Mom immediately took the patient to Mt. Sinai Hospital where he was found to be unresponsive, grunting and having abnormal posturing. He was intubated with initial vent settings of FiO2 of 100%, rate of 40, tidal volume 150, PEEP of 5. He reportedly had seizure-like activity so he was given Ativan X1. Per report from Mt. Sinai, as the patient was being examined and found to have a right erythematous region over the right temporal region, mom immediately turned to her boyfriend suspiciously. An emergent CT scan of the brain showed a large right temporal subdural and intraparenchymal bleed.

PAST MEDICAL HISTORY AND SURGICAL HISTORY: As above.

MEDICATIONS: None.

ALLERGIES: No known drug allergies.

SOCIAL HISTORY: The patient had been living with his mom and her boyfriend and their 2-week old daughter.

FAMILY HISTORY: Noncontributory.

INITIAL PHYSICAL EXAM: Vital signs: Temperature 95.7 rectally. Respirations 40 on a vent rate of 40. Heart rate is 196. Blood pressure is 93/77. Vent settings were rate of 40, tidal volume 150, PEEP of 5, and FiO2 of 100%. General: The patient is unresponsive. His right temporal and parietal areas were erythematous. There was also a 2 to 3-cm diameter area of erythema over his posterior occiput as well. HEENT: There was no Battle sign. No rhinorrhea. Pupils were equal bilaterally, approximately 2 mm, but the left pupil was noted to be sluggish. There were no raccoon eyes. His Tms were clear bilaterally with no evidence of hemotympanum. Mouth: He is orally intubated. Lungs: Clear to auscultation bilaterally. Heart: Tachycardic. No murmurs. Normal S1, S2. Abdomen: Soft, nondistended, hypoactive bowel sounds with small red and somewhat ecchymotic areas around his midepigastric and periumbilical regions

MC 537

ecchymotic areas around his midepigastric and periumbilical regions. There was noted scrotal erythema as well as edema, right greater than left. On his back there were multiple whip marks over his mid to lower back as well as right posterior thigh. His buttocks also had some erythema, right greater than left. Extremities: Capillary refill is less than 2 seconds. Pulse is 1+ bilaterally in the radial pulses and 2+ femoral pulses. Neurologic exam: The patient is unresponsive. There is some decerebrate posturing with extension of the wrists and feet. He was hyperreflexic to approximately 3+ throughout with some increased tone.

His initial laboratories were significant for a BUN and creatinine of 25 and 1.2 respectively and a glucose of 517. His white count was 19.1, hemoglobin 12.6, hematocrit 37.9, platelets 512. His urine drug screen was negative. Tylenol and aspirin levels were negative. His UA showed greater than 300 protein and moderate blood. Lipase was 95, 98, and amylase was 1131. His SGOT was 5429 and SGPT was 3130.

HOSPITAL COURSE:

1) Neuro: Upon arrival to the PICU, the patient had primary and secondary survey as described above. Head CT scan was reviewed and showed a large subdural hematoma involving the right frontal temporal and parietal areas. He was emergently taken to the operating room following placement of a right subclavian line as well as a right femoral A line. In the operating room his intracranial pressure was noted to be greater than 90 with evidence of subdural subarachnoid hemorrhage as well as edema secondary to severe head injury that he had sustained. Immediately following his craniectomy pupils were then noted to be both fixed and dilated. The patient did not receive any sedative medications over the course of his hospitalization and his neurological exam remained unchanged. Specifically, on February 09 and February 10, 2002, he was noted to have absent corneal reflexes bilaterally, fixed and dilated pupils with absent gag reflex. He was unresponsive with no movement noted to deep pain, except with lower extremity spinal reflexes. He was also without dolls eyes reflexes, no response to cold calorics, and he had a positive apnea test off the ventilator for 5 minutes with no spontaneous respirations noted. As a result of the above findings, brain death criteria were met on February 09 and February 10, 2002, and the patient was pronounced brain dead on February 11, 2002. Brain death and his prognosis were discussed extensively with the family and the patient was removed from the ventilator at 12 p.m. on February 11, 2002, and pronounced dead at 12:24 p.m. An ophthalmology exam on February 09, 2002, was consistent with bilateral retinal hemorrhages.

) Social: Given the degree of severity of the injuries that the patient had sustained, non-accidental head, as well as abdominal trauma

was strongly suspected. Child protective services, DCFS and the Chicago Police Department were all notified. Both Dr. Kramer and Dr. Scotellaro both agree that given the evidence of the large subdural and intraparenchymal bleed with diffuse brain swelling and edema, resulting in brain death, bilateral retinal hemorrhages, extensive cutaneous injuries, and intra-abdominal injuries, collectively were diagnostic of child abuse. Both the patient's mom and her boyfriend were detained in Wheaton by the DuPage County Sheriff's Department.

3) SIRS shock with multi-organ dysfunction. The patient was found to have multi-organ dysfunction involving CNS, pulmonary, GI, and cardiovascular systems. As described above, the patient had a large right subdural and subarachnoid hemorrhage and met the criteria for brain death. His initial lipase and amylase were 9,598 and 1,131 respectively. His SGOT was 5429 and SGPT of 3130. Pediatric surgery was consulted on February 09, 2002, and placed a Penrose drain into the right lower quadrant of the abdomen with return of approximately 500 cc of serosanguinous fluid. In addition, on February 09, 2002, ROBI was consulted for possible organ donation. The patient's blood pressure and urine output were monitored closely. He was given fluid boluses as well as packed red cells and fresh frozen plasma to maintain his hemoglobin, hematocrit and his blood pressure in anticipation of a possible organ donation. In discussions with Pam Nelson of ROBI, they decided not to pursue organ harvesting in this case secondary to medical examiner priority. The patient was weaned off dopamine and as described above, ventilatory support was withdrawn on February 11, 2002.

DCFS, central DuPage authorities, as well as the medical examiner were notified of the patient's death at 12:24 p.m. on February 11, 2002.

DICTATED BY: CHENG, LEON Y M.D.

Electronically Approved by:SEVERIN, PAUL N M.D.
On:Mar 11, 2002

MR 535

RU 144/ 64301
D: 03/08/2002
T: 03/11/2002

5500584 1001 2 H
QUINN
STEVEN
SEVERIN, PAUL
RRUSSE

RUSH-PRESBYTERIAN-ST. LUKE'S MEDICAL CENTER
CHICAGO, ILLINOIS 60612
PEDIATRIC TRANSPORT REFERRAL FORM

HISTORY:
2 y/o ♂ BIB parents, eating hot dog → choking & grunting. On admission, grunting & posturing, unresponsive. ↓ crying on blood streaks.
Bruise ⓒ temporal region.
Dad had been taking care of child alone today as mom at work.
Red, small marks on abd / back / buttocks

Date: 8/8/02 Time: 7:05pm
Name: Quinn, Steven
DOB: 4/7/99 Sex: M Weight: ~14kg
Parent(guardian):
Referral MD: Dr. Green.
MD Phone: (773) 257-6241
Referral Hosp: Mt Sinai
Hosp. Phone:
City: Unit:
Diagnosis: epidural bleed.

PMH:
↓

Allergies: NKDA.

MEDS GIVEN:
Med / Dose / Time
mannitol
phenobarbitol
ativan
lidocaine
succinyl

FLUIDS:
Maintenance (Solutions, Rate)

BOLUSES:(Solution, CC/KG)
0.9 NS bolus.

LINES: ☐ PIV ☐ PIA ☐ Central ☐ Intraosseous ☐ Cut Down

PHYSICAL ASSESSMENT

Vital Signs			
	99.2		
P	154		
R	36		
BP	135/69		

CARDIOVASCULAR
Murmur
Rhythm
Color
Pulses
Cap Fill Time
Liver

RESPIRATORY
ABNL Aeration
Retractions
Rales
Wheezes
Supplemental Oxygen
Oxygen Saturation

VENT PARAMENTERS
FIO_2 100%
PIP/PEEP 30/5
TV 150 cc
Rate 30
☒ ETT 4.5 ☐ Trach
☒ Sedated ☒ Paralyzed
Other

NEURO STATUS
Mental Status
Seizures
Focal Signs
Glascow Coma Score
Other

LAB DATA pending.

Na 139	Cl 105	BUN 25	Ca 9.5	Mg	Alb	ABG's 7.40/42	
K 4.3	CO_2 (17)	Cr 1.2	Glucose (517)	PO_4	TP	pH 7.234	
HGB 12.6	WBC 19.1	DIFF S B L M			SGOT	pCO_2 34.7	
HCT 37.9	PLT 512	OTHER: serum acetaminophen, amylase, lipase, UDS ⊖, + ASA ⊖, UA: Cath, mod bld, 1 Sqet, 1 WBC, 1 SRBC, gluc ⊕			SGPT	pO_2 246	
PT	FIBRINOGEN				Bili	Bicarb 143	
PTT					NH3	BE -12.1	

EKG:

CXR:

Head CT: ⓒ epidural subdural & intraparenchymal

CULTURES: ☐ CSF ☐ Urine ☐ Blood ☐ ETT ☐ Other

GI/GU
NPO
Vomiting:
Diarrhea:
Urination:
NG GT
Foley
Other:

Problem List:

Recommendations:
① add amylase, lipase, coags to labs
② Head CT scan p/o intra-abd bleed.

MR 367

Patient Accepted: ✓
Chart Copied: ✓
Verbal Consent:
Signature/Title:

PATIENT TRANSFER DATA
Time team left: 8 pm
Time of arrival: 8:
Agency of Transport:
Time leaving referral:
Time arriving RPSLMC:
Signature/Title:

Form No. 1863 Rev/01
White - Chart Copy | Yellow Transport Committee Copy | Pink - Attending Copy

[illegible]

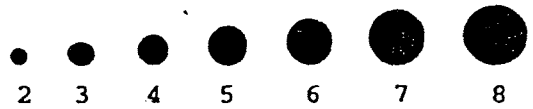
[illegible] 1001 2 H
QUINN
STEVEN
SEVERIN, PAUL
RRUSSE

RUSH-PRESBYTERIAN-ST. LUKE'S MEDICAL CENTER
Rush Children's Hospital
Chicago, IL 60612
Pediatric Transport Nursing Note

GENERAL INFORMATION
Patient Name Steven Quinn
Diagnosis/Procedure
Referring Physician Dr Green
Receiving Physician Dr. Severin
Transport Physician Dr Subramanian.
Vitals: Temp 94.3 ® Pulse 134 Resp 40%RA BP 96/63 (64)

Date/Time of Transfer 2/9/02 850pm.
Allergies NKDA
Referring Hospital Mt Sinai ER
Receiving Hospital RPSLMC - PICU
Transport Nurse Tammy Smoot.

NEUROLOGICAL
LOC: Alert ____ Oriented x ____
Lethargic ____ Stuporous ____ Unresponsive X
Movement: S=Strong W=Weak (N=Nil)
Upper Rt ____ Lt ____ ; Lower Rt ____ Lt ____
Speech: WNL for age ____ Nonverbal ____
Pupil Size: Rt 4 Lt 4 ; React Rt Ø Lt 4→3 sluggish



RESPIRATORY
Pattern: Regular X Irregular ____
Quality: Easy X Labored ____
Rapid ____ Shallow ____
Breath Sounds:

	Clear	Congested	Wheezing	Absent
Rt	X			
Lt	X			
Other				

Cough: Productive ____ Nonproductive ____
Sputum ____
Airway: ET 4 Soral Trach ____ Other ____
Oxygen Therapy: ____
ABG Date/Time: ____
pH ____ PCO_2 ____ PO_2 ____
HCO_3 ____ BE ____ O_2 Sat ____
Vent: Mode ____ Rate 40 FiO_2 100
(V) PIP 150 PEEP +5

CIRCULATION
Pulses:

	Rt	Lt
Femoral	(G) F P N	(G) F P N
Pedal	G F (P) N	G F (P) N
Radial	G (F) P N	G (F) P N

G=Good F=Fair P=Poor N=Nil

Skin:
Pink X Pale ____ Other ____
Warm Trunk Cool extremities Diaphoretic ____
Appearance: Bruising on ® side head across abd
Normal ____ Intact red marks diagonally across back x 2
Tenting ____ Edema red marks across inner ® thigh
Rash ____ Other ® scrotum reddened + swollen

ABDOMEN
Flat ____ Distended ____ Firm ____ Soft ____
Bowel Sounds ____ Last BM ____

CONDITION OF WOUNDS/DRESSINGS
Site: (L) Ankle PIV /Cond. of Wound: soft, flat
Drainage: Ø noted
Type of dressing: Tape Time changed: ____

Site: ® hand PIV /Cond. of Wound: soft, flat
Drainage: Ø noted
Type of dressing: tape Time changed: ____

Site: ____ /Cond. of Wound: ____
Drainage: ____
Type of dressing: ____ Time changed: ____

TUBES/DRAINS

	Condition of Site	Type of Drainage
Mediastinal		
Pleural		
Hemovac/JP		
NG/Keofeed		
Gastrostomy		
Jejunostomy		
Other		
® Foley →	gravity	clear orange colored urine

Time Voided ____

MR 7:57

Signature: Tammy Smoot RN

MR Form No. 2616 9/96

White: RPSLMC Service Yellow: Receiving Hospital Pink: Transport Team, Supervising Attending

0006

5500364 1001 2 M
QUINN
STEVEN
SEVERIN, PAUL
RRUSSE

RUSH-PRESBYTERIAN-ST. LUKE'S MEDICAL CENTER
Rush Children's Hospital Chicago, IL 60612
Pediatric Transport Summary

TRANSFER DATA
Depart RPSLMC
Arrive Referring Hosp.
Depart Referring Hosp.
Arrive Receiving Hosp.

PATIENT INFORMATION			
Name			M F
Date	DOB	Age	WT
GA	BWT	Recent infectious exposure? Y N	
Time of Birth	VtK? Y N	Specify:	
Apgars 1 5 10		Info given to parents? Y N	
Neonatal Screen? Y N Eye Care? Y N		Consent? Y N	Chart/x-rays? Y N
Mother's blood? Y N Cord blood? Y N		Call to family? Y N referring hospital? Y N	

TRANSPORT INFORMATION	
Referring Unit	Referral Diagnosis
Referring Hospital	Receiving Hospital
Referring MD	Receiving Unit
Primary MD	Transport MD
	Transport RN

LABORATORY DATA				MOST RECENT BLOOD GAS		
Na	Cl	BUN	Ca	Time	Type	
K	CO_2	Cr	Gluc	pH	PCO_2	PO_2
Hg	WBC	S	B	HCO_3	BE	O_2Sat
Hct	Plt	L	M	ON VENTILATOR SETTINGS		
CULTURES OBTAINED				Rate	FiO_2	I time
Blood	Urine	CSF	Sputum	TV	PIP	PEEP

IV/SOLUTION/ADDITIVE CONCENTRATION	RATE	SITE
A.		
B.		
C.		
D.		

MEDICATIONS GIVEN	DOSE	TIME
1.		
2.		
3.		
4.		

PRESENT COMPLAINT:

PREVIOUS MEDICAL PROBLEMS:

PREVIOUS SURGERY:

ALLERGIES:

TIME	VITAL SIGNS				RESPIRATORY THERAPY				NEURO	MEDICATIONS	INPUT				OUTPUT		
	T(pt)	HR	NBP	ART	FiO_2	PIP	TV	Device	GCS	Type/Dose	A	B	C	D	Urine	Stool	Other
	T(env)	RR			Rate	PEEP	Mode	Flow	LOC	Route/Time							
8:05	94.3	124	96/(61)		100		150										
		40			40	5		02 Sat 97%									
										Totals							
										Total In/Out							

Signature: _____ Title

MR Form No. 2615 Part 1 9/96

White: RPSLMC Service

Yellow: Receiving Hospital

Pink: Transport Team Supervising Attending

MED 44

MR 359

200009

1101 2 M

RUSH-PRESBYTERIAN-ST. LUKE'S MEDICAL CENTER
Rush Children's Hospital Chicago, IL 60612
Pediatric Transport Summary Continuation

Patient Name: Steven Quinn
Date: 2/8 Page Number:

Additional Comments:
Arrived in MH Small ER, to find pt orally intubated + mechanically ventilated. Pt to have access breath movements of chest. Multiple bruise within noted on abd, R) side head of head. Red marks also noted on inside of L) thigh. 2 diagonal red marks across back & bruising also noted. IV in NaHCO3 infusion started over 30". L) antecube PIV retained. Placed on transport monitor + transported to RPSLMC Bill 5 incident

Tammy Smith RN

TIME	VITAL SIGNS				RESPIRATORY THERAPY				NEURO	MEDICATIONS	INPUT				OUTPUT		
	T(pt)	HR	NBP	ART	FiO_2	PIP	TV	Device	GCS	Type/Dose	A	B	C	D	Urine	Stool	Other
	T(env)	RR			Rate	PEEP	Mode	Flow	LOC	Route/Time							
										Totals							
										Total In/Out							

Signature: Tammy Smith RN Title

MR Form No. 2615 Part 2 9/96

White: RPSLMC Service Yellow: Receiving Hospital Pink: Transport Team, Supervising Attending

MED 45

SS[illegible] 1001 2 M
QUINN
STEVEN
SEVERIN, PAUL
RRUSSE

RUSH-PRESBYTERIAN-ST. LUKE'S MEDICAL CENTER
Rush Children's Hospital
Chicago, IL 60612

TRANSPORT NOTE * NOTE HX LIMITED PARENT BEING QUESTIONED BY POLICE	PHYSICIAN'S ORDERS
Patient Name: STEVEN QUINN	Weight: 14 kg
Referring Hospital: MT. SINAI	Allergies: NKDA
Referring Hospital Phone: (773) 257-6241	Date: 2/8/02
Referring Physician: PAULA GREEN, MD	Time
Referring Physician Phone: ( )	8:35PM NaHCO3 7mEq IV over 30min
Primary Physician: ANN KELLER, MD (Madison Fam Clinic)	
Primary Physician Phone: ( )	
Receiving Physician Phone: ( )	
Date/Time: 2/8/02 8:30PM	

2yo ♂ s/p PMH. Mom reports that when she left for work ~10AM, child was behaving normally; when she returned home ~6PM, child minimally responsive ~~AMS~~ abnormal breathing. Child left with mom's boyfriend alone during day while mom at work. No h/o trauma of any kind volunteered by mom; says marks on his belly are "old." Ø known ingestions. Ø symptoms preceding. Moms boyfriend told her that the child choked on a hot dog around lunchtime (~12PM). Pt taken by Ambulance to Mt. Sinai - there found to be unresponsive - abnl posturing (decerticate). Resp irregular, Ø grunting. Pt intubated; vent setting; TV 150 cc, FiO_2 100%, R 40, PEEP +5. Had sz-like activity x several seconds → received Ativan 0.1 mg/kg x 1. Pt received Lidocaine 15mg, Aux 15mcg and Versed 2mg when intubated. Received a 2nd dose of Versed ~10p intubation (7:45 PM; got 2mg). CT brain showed large (R) subdural & intra-parenchymal temporal hemorrhage.

ativan 6:15 PM intubated 7:30 PM

MD 760

Signature: U Subra MD SUBRAMANIAN #3933 MD/DO

MR Form No. 2613 Part 1 9/96

White: RPSLMC Service Yellow: Receiving Hospital Pink: Transport Team, Supervising Attending

SSDSoA 1001 2 H
QUINN
STEVEN
SEVERIN, PAUL
RRUSSE

RUSH-PRESBYTERIAN-ST. LUKE'S MEDICAL CENTER
Rush Children's Hospital
Chicago, IL 60612

TRANSPORT NOTE CONTINUATION PAGE 2/3

Patient Name: STEVEN QUINN

Date/Time: 2/8/02 8:30PM

Labs drawn (see below). Skeletal survey done → ø reading. C-spine not yet cleared. CT abd ordered but not yet done. Mannitol 7gm (0.5 mg/kg) IV started; pt on IVF of D.5 NS (received 20cc/kg bolus). Ⓐ Hypothermic (T 94) → bair hugger blanket.

PMH: ø Birth Hx: FT, NSVD ø complications.

Meds: ø All: NKDA Fam Hx: ø Hx dz d/c, Ⓐ asthma, Ⓐ DM, ø CV dz ~~ø pmtx~~

PE: Wt 14 kg VS: T 94 R 40 on vent HR 130's SaO2 100%

Neuro: Intubated, sedated; withdraws to pain.

HEENT: Ⓐ extensive bruising over Ⓡ temporal + occipital areas. C collar in place. PERRLM: Ⓡ pupil ~4mm and reactive to 3mm; Ⓛ pupil ~4mm and poorly reactive (sluggish) to 3mm. MMM: ETT in place; ø OGT/NGT. ø Raccoon eyes or Battle's sign.

CV: RRR Ⓐ S1, S2, ø m. Fem + radial pulses 2+ Ⓑ.

Resp: Good AE equal Ⓑ; CTAB ø W/R/R (vent).

Abd: Soft; Ⓐ bruising (red 2-3cm patches) in epigastric region. ND; hypoactive BS; ø HSM; ø masses palpated.

Ext: Ⓐ decorticate posturing of hands (Ⓡ hands fisted + wrists flexed); Ⓑ ankles hyperextended (plantar). ~~Withdraws~~ Ⓑ Ext cool x 4.

Neuro: Pupils – see above. ø ~~stp~~ Eye opening. Withdraws extremities to pain R more than L. Ⓐ Occasional spont. movements of Ⓡ side during exam. Ⓑ patellar and achilles reflexes 4+ (TCD).

GU: Ⓐ Ecchymotic Ⓡ scrotum. Ne ↑ testes bl. Anus: ø trauma Ⓐ dry blood

Back: Ⓐ Rope marks (horizontal) x 3 lower back (~5-10cm long x 3). Ⓐ Bruising over sacrum. sacrum

Skin: Ⓐ Ecchymoses (see above) on head, back, abd; also between legs on upper thighs (medial surfaces upper thighs).

LABS: ~~4.7/12.6/512~~ 37.9 , N81/L13/M6/E0.2/B0.2

139 | 105 | 25 / (517)
4.3 | 17 | 1.2

Ca 9.5 Serum ketones ⊖ Serum tyl + ASA nl

Urine tox ⊖ UA (cath): prot >300, gluc 250, ket 15, mod blood, RBC 14, WBC 1

Signature: [signature] MD SUBRAMANIAN #3933 MD/DO

MR Form No. 2613 Part 2 9/96

White: RPSLMC Service Yellow: Receiving Hospital Pink: Transport Team, Supervising Attending

MR 359 MED 47

ISCSS# 1001 2 H
QUINN
STEVEN
SEVERIN, PAUL BRUSSE

RUSH-PRESBYTERIAN-ST. LUKE'S MEDICAL CENTER
Rush Children's Hospital
Chicago, IL 60612

TRANSPORT NOTE CONTINUATION PAGE 3/3

Patient Name: STEVEN QUINN
Date/Time: 2/8/02 9:40 PM
Amylase / lipase - drawn; Ø results.
ABG (on current vent settings): 7.234/34.7/246/14.3/-12.1
A/P: 2yo ♂
#HM → bruises appear to be all of the same age.
#Marks of extensive trauma c (R) temporal + subdural + intraparenchymal hemorrhage and ex of ↑ICP. likely 2° to child abuse.
- Transport + admit to Rush PICU (consent obtained)
- D/W Dr. Severin
- ABCs → keep pt intubated @ current vent settings: hyperventilate; IVF 0.9 NS @ maint c NaHCO3 @ 0.5 mEq/kg.
- Neurosurgical consult (Dr Munoz aware) — likely to go to OR for emergent evacuation
- Continue mannitol
- Review skeletal survey; clear C-spine
- CT abdomen + Chest - Ophtho exam (R/o retinal hemorrhages)
- √ amylase / lipase (R/o pancreatic injury)
- Child Protective Services (Dr Scotellaro) to be notified.
- Mom + mom's boyfriend being questioned.
- DCFS to be notified.
- Further management in Rush PICU.

Tp) Metabolic acidosis, hyperglycemia: likely 2° to extensive trauma + stress. Cont. NaHCO3 in IVF; rec'd BMP.

MR 761

Signature: [signature] MD SUBRAMANIAN #3933 MD/DO

MR Form No. 2613 Part 2 9/96

White: RPSLMC Service Yellow: Receiving Hospital Pink: Transport Team, Supervising Attending

IMAGED 2-21-03

DuPage County Sheriff's Police
Follow up Report

COPY

Incident # 02-4531 Page 2

	TYPE OF INCIDENT	DATE OF FOLLOW-UP	TIME	Complainant's name
0110	Homicide	02-12-02	0815	Chicago PD

02-13-02 1105hrs. This detective, Ray Bradford from the DuPage County Children's Center and A.S.A. Dave Imielski went over to the Hollywood address and met with Randy. He was asked if we could talk over at Hanover Park PD. He agreed and the four of us drove over to Hanover Park PD.

02-13-02 1120hrs. In one of the departments interview rooms, this detective and Ray Bradford spoke with Randy. He was admonished Miranda, he indicated he understood his rights and agreed to speak with us after signing the waiver form.

Randy was advised of the medical evidence against him. That we knew he was responsible for the tragedy to Steven. That he was at the apartment by himself with the children. That he probably didn't think the injuries Steven sustained were as serious as they were. How this could have been an accident on his part and that he didn't intend for this to happen. That he was the only one who could help himself. Randy would sit and listen for long periods of time and on occasion when the subject of harming Steven was brought up he would say that he'd never hurt Steven like that. Randy would say that he loved Steven ~~wouldn't do that to him. Various other subjects were covered which included how a judge, a jury and th~~ ~~ates Attorney would view him.~~ How his family would view him. Randy was told that nothing could be promised to him and that we could not make any deals but that his cooperation and any regret on his part could be passed on to the prosecutor's office. This was the general conversation with Randy. It was discussed in a very repetitive manner, which lasted two and a half hours. In addition to denying he hurt Steven, Randy indicated that just before he and Kenyatta left for Mount Sinai hospital with Steven, he took his dog out to relieve itself. When he returned back to the apartment they left.

02-13-02 1350hrs. Ray Bradford left the interview room, this detective stayed with Randy. The conversation continued on about how his family would view him. Randy was asked and indicated that he had only eaten twice since last Saturday and that he wasn't getting the proper rest he should. This detective expressed the fact that he needed to release this from within because it was built up inside of him, that he should to do this for his daughter Angelique and especially for Steven. Randy started to cry but continued to deny hurting Steven.

02-13-02 1410hrs. Ray Bradford returned back inside the interview room, he asked Randy if he was willing to be wired when he spoke with Kenyatta, he related he would. (This was only asked to find ~~hat Randy's reaction would be, there was no intention on going through with this).~~ Randy was also

MR 72

OFFICER #1 CLEARED

DuPage County Sheriff's Police
Follow up Report

COPY

Incident # 02-4531 Page 3

	TYPE OF INCIDENT	DATE OF FOLLOW-UP	TIME	Complainant's name
0110	Homicide	02-12-02	0815	Chicago PD

asked if he would take the polygraph today, Randy said he wouldn't take it today because he was still sick due to his heroin habit. Randy was asked if we could audiotape his account of events that occurred on Friday the 8th, he agreed to do this. This detective left the room to get the tape recorder, Ray Bradford remained with Randy. This detective returned to the interview room after a few minutes to obtain the car keys from Ray Bradford. The door to the interview room was opened and Ray Bradford indicated that Randy had asked for an attorney.

Randy was brought out to the lobby, his mother Linda Liebich was there waiting for him. Before leaving with his mother, Randy was asked if he would take the polygraph tomorrow. He related that if he could obtain his methadone supply he would definitely do so. Randy was advised to call this detective (supplied him a business card) tomorrow at 0900hrs so the examine could be set up. Randy said he would call.

02-14-02 0955hrs. Randy Liebich had not called. This detective called the cell phone number of y Sicosinski, the stepfather of Randy Liebich. No one answered the Nextel phone.

02-14-02 1000hrs. Denise Liebich was called, it was indicated that her brother Randy didn't call this detective at the previously arranged time of 0900hrs. Denise related that she would call a neighbor by Wally Sicosinski's residence and have them go to their door to deliver the message to Randy.

02-14-02 1010hrs. Randy Liebich called this detective on Wally's cell phone. He was advised that the polygraph examiner was scheduled to be in our office between 1400-1430hrs today. Randy was asked if he needed a ride to our office, he related he didn't, that he had a ride. Randy related that he would be in at 1400-1415hrs.

02-14-02 1419hrs. Randy Liebich called and related that he called an attorney, a Dennis Born (847-501-3388) who told him not to come in and take the test. Randy was advised that the test was voluntary, that the decision to take one was entirely up to him and any attorney would tell him not to take one. An offer was again made to supply him with a ride. It was indicated that even if he wasn't feeling that well he should take it. Randy indicated that he wasn't going to come in today and that he hasn't been able to get his methadone. Randy was further advised that Kenyatta took the polygraph test when asked and that this detective wished he would reconsider. Randy related he wasn't coming in today.

MK 73

OFFICER #1

CLEARED

DATE 2/12/02

Miranda Rights

Date 2-9-02

I have been informed by Richard D. O'Brien, polygraph examiner, that he wants to question me about the inquiries to Steve Quard's on 8 Feb 02

He has told me that I have the right to remain silent and that I do not have to agree to be questioned at this time unless I wish to do so. He has also told me that even though I agree to be questioned I have the right to change my mind at any time during the questioning and can refuse to answer. I may also request that the questioning be stopped and he will abide by any such decision. He has further advised me that anything I say may be used against me in court at some future time.

He has further advised me that I have a right to consult with an attorney or in the event that I can not afford to retain an attorney one will be appointed to represent me. I have a right to have said attorney present if I wish during the time I am being questioned.

I fully understand what I have been told and I hearby agree that I am willing to discuss the ABOVE

I agree to be questioned at this time and I do not wish to consult with, retain, or have an attorney appointed to represent me.

I have been requested to read the above statement, which I have done, and hearby state that there has been no interrogation about the ABOVE prior to my having read and signed this statement.

MR 2-11

X Signature Randy Cook

DU PAGE COUNTY SHERIFF
FIELD INTERROGATION WAIVER

A. You have a right to remain silent and do not have to say anything at all.

B. Anything you say can and will be used against you in Court.

C. You have a right to talk to a lawyer of your own choice before we ask you any questions, and also to have a lawyer here with you while we ask questions.

D. If you cannot afford to hire a lawyer, and you want one, we will see that you have a lawyer provided to you, before we ask you any questions.

E. If you are willing to give us a statement, you have a right to stop any time you wish.

F. Do you agree to answer our questions here and now?

YES ✓ NO .....

G. [signature] .....
Signature

Witness [signature] .....

Witness [signature] .....

Time and Date 1:05 A.M. 2-9-02

SO-00251

W-223

DuPage County Sheriff's Police
Follow up Report

ORIGINAL

Incident # 02-4531

	TYPE OF INCIDENT	DATE OF FOLLOW-UP	TIME	Complainant's name
0110	Homicide	2-8-02	1950	Chicago PD

Cod e	NAME: LAST, FIRST, MIDDLE	ADDRESS	CODE	D.O.B.	S	R	TX HOME	TX BUS
C	Officer Sullivan John #8479	10th District Chicago PD 2259 S Damen	165	Adult	M	W	Cell 773-203-1779	773-747-5028
W	Officer Filipiak Thomas #19723	Chicago PD 10th District 2259 S Damen	165	Adult	M	W		773-747-5028
V	Quinn Steven	16 W 505 Mockingbird Apt 204	793	041799	M	B	No Phone	
S	Liebich Randy R	16 W 505 Mockingbird Apt 204	793	071279	M	W	330-1432	
W	Brown Kenyatta M	16 W 505 Mockingbird Apt 204	793	100683	F	B	773-722-7976	Cell 708-646-5801
W	Liebich Angelique Marie	16 W 505 Mockingbird Apt 204	793	012702	F		No Phone	
W	Lt. Szalinski	D.P.S.O.	512	Adult	M	W		682-7279
W	Det Delgiudice	D.P.S.O.	512	Adult	M	W		682-7865
	Det Sgt Kunz	D.P.S.O.	512	Adult	M	W		682-7278
	Det Sgt Price	D.P.S.O.	512	Adult				682-7802
W	Investigator Vrbos Boris	DuPage County Children's Center	512	Adult	M	W		681-2432
W	Smith Tammy Registered Nurse	Rush Presbyterian St. Lukes	165	021964	F	W	708-366-3635	312-942-6191
W	Dr. Severin Paul N	Rush Presbyterian St. Lukes	165	Adult	M	W	Pager 312-333-4251	312-942-6194
W	Dr. Munoz Lorenzo Pediatric Neural Surgeon	Rush Presbyterian St. Lukes	165	Adult	M	W	Pager 877-665-4050	
W	Assist S.A. Guerin Dan	DuPage County States Attorney Office	512	Adult	M	W		682-7760
W	Assist S.A. Brennan Liam	DuPage County States Attorneys Office	512	Adult	M	W		682-7669
W	Assist S.A. Reidy Michael	DuPage County States Attorney Office	512	Adult	M	W		682-7669
W	Deputy Chief Bradford Ray	DuPage County Children's Center	512	Adult	M	W	Pager 722-8582	681-2426
W	O'Brien Richard T	15 Spinning Wheel Road	796	Adult	M	W		325-4404
W	Clark Karen L	714 S Independence Blvd	165	101564	F	B	773-722-7976	773-736-9636
	Herron Dorothy J	4937 W Augusta Blvd	165	111049	F	B	773-379-9440	773-419-8317

MR 10

DuPage County Sheriff's Police
Follow up Report

ORIGINAL

Incident # 02-4531

	TYPE OF INCIDENT	DATE OF FOLLOW-UP	TIME	Complainant's name
0110	Homicide	2-8-02	1950	Chicago PD

W	Liebich Denise M	1231 Prairie Brook Drive Palatine	999	040670	F	W	847-991-0731	847-991-4646
W	Patio Restaurant	7440 Rt 83	722					920-0211
W	Mileusnic Darinka	Cook County Medical Examiner 2121 W Harrison	165	Adult	F	W		312-666-0500

POLICE ACTION: REFER TO STATES ATTORNEY [ ] COMP. SIGNED YES [ ] NO [ ] ARRESTS: TYPE____ # OF PERSONS____ OTHER ____

NAME OF ARRESTEE	CHARGE	STATE #	I. R. #	C.C OR TICKET #

NARRATIVE

Lead# 1

2-8-02 1950hrs. This detective received a telephone call from Lt. Szalinski at the office and indicated that there was a report of an injured infant, possibly a shaken baby syndrome case from Willowbrook. The child was currently at Mount Sinai Hospital in Chicago. The Chicago Police Department c"ed our office and two Chicago police officers were at the hospital. It was requested that this detective one of the officers on his cell phone, an Officer Sullivan.

2-8-01 2005hrs. This detective called Officer Sullivan who related what information he had at this point. The victim child, Steven Quinn, a two-year-old male black was in critical condition. This child had trauma to the head, scrotum and bruises throughout the body. It was indicated that the doctors could not tell if the injuries were recent. The mother of the child, eighteen-year-old Kenyatta Brown and her twenty two year old boyfriend, Randy Liebich transported the child to the hospital in their car. Steven Quinn is a child from a previous relationship of Kenyatta Brown not between her and Randy Liebich. Also in the company of this couple was another 11-day-old female infant, Angelique Liebich who is their natural child The explanation Randy Liebich gave to the Chicago Police Officers in relation to what occurred was that he was at home in Willowbrook sleeping this afternoon. When he got up he fed the victim child a hot dog and Steven started to choke. After Steven stopped choking he laid Steven down and the child became unresponsive. The reason given for their drive from Willowbrook to Mount Sinai hospital in Chicago was that Kenyatta Brown felt more comfortable at this particular hospital. Randy Liebich was very vague with his explanation. Officer Sullivan related that he notified D.C.F.S. in this incident. It was also indicated that the Chicago Officers would stay at the hospital until personnel from this office arrived. It was further indicated that Steven may be transferred to Rush Presbyterian St. Lukes due to his serious condition.

2-8-02 2020hrs. This detective called Lt. Szalinski back and informed him of the situation. He indicated he would be coming to the office.

2-8-02 2100hrs. At the office Lt. Szalinski paged A.S.A. Dennis Harrison who was on call.

2-8-02 2115hrs. This detective phoned Officer Sullivan who related that Steven Quinn had already been transported to Rush Presbyterian Hospital and was on floor 5 (Kellogg) in pediatrics.

2-8-02 2120hrs. Lt. Szalinski contacted the DuPage County Children's Center and was advised an agent would respond to our office.

MR 11

DuPage County Sheriff's Police
Follow up Report

ORIGINAL

	TYPE OF INCIDENT	DATE OF FOLLOW-UP	TIME	Complainant's name
0110	Homicide	2-8-02	1950	Chicago PD

2-8-02 2145hrs. Criminal Investigator Boris Vrbos from the DuPage County Children's Center arrived at the office. He was advised of the incident and he called Assistant States Attorney Dan Guerin of the domestic violence section to relate what had transpired and information known at the time.

2-8-02 2150hrs. This detective and Investigator Vrbos left our office and rode together to Rush Presbyterian St. Lukes Hospital. Lt. Szalinski, Det Sgt Kunz and Det Delgiudice also drove out to the hospital.

2-8-02 2240hrs. These investigators arrived at the John L. & Hellen Kellogg Pavilion pediatric critical care unit floor of the hospital and met with the Chicago Police Officers, registered nurse Tammy Smith and attending physician Paul Severin in a conference room marked 537. The Chicago Police Officers related further that the mother, Kenyatta Brown was at work all day and her boyfriend Randy Liebich was watching the kids at their apartment. Around 1500hrs the victim child Steven Quinn had choked for a short period on his hot dog and at 1700hrs the mother came home from work. It was at this time that they decided to take Steven to a hospital. The mother and boyfriend had Steven for the last 3 to 4 days, prior to this the child was being taken care of by his grandmother.

Attending physician Paul Severin spoke of the injuries the child sustained. It was indicated that there was a bleed in the brain on the right side. There was internal abdominal injury. The child was showing signs of severe brain injury. Steven had bruising about the head, he had marks on his back and on the area of his inner legs. There was a little blood in his urine. He was brought into the hospital at hrs and went into surgery at 2158hrs., he was still currently in surgery. Randy Liebich and Kenyatta Br n originally brought Steven into Mount Sinai Hospital at 1800hrs. Nurse Tammy Smith related that Steven weighed almost thirty pounds and when brought into the hospital the child had a low body temperature. The surgery was in an effort to reduce blood swell to the brain. According to Doctor Severin, the child was grunting and not crying, he was also posturing, meaning he had abnormal movement. Doctor Severin demonstrated this type of movement by holding his arms at the sides of his body then he pushed his arms forward up around his chest. He said that this type of movement was consistent with severe brain injury. It appeared the child had injury of a vein that is below the outer portion of the skull and brain. There were no broken bones observed by a visual check. The bruising on his body looked relatively recent and all bruising appeared to be of the same age. An opinion by Doctor Severin which he indicted would be a guess on his part is that the bruising occurred sometime between 24 to 48 hours. None of the bruising stood out more than the other. Pediatric Neural Surgeon Doctor Lorenzo Munoz was performing the surgery.

2-8-02 2320hrs. Nurse Tammy Smith was requested to bring the boyfriend Randy Liebich to the conference room for the purpose of conducting an interview with him. She said at least she could get him away from Kenyatta Brown's family members who were making comments that he was probably involved in this. Tammy also noted that Randy Liebich clenched the infant Anglique close to him in his arms while at the hospital.

2-8-02 2325hs. Tammy Smith brought Randy Liebich into the conference room. This detective and investigator Boris Vrbos then conducted an interview with him. Randy Liebich related that he was watching Steven this morning. The child's mother left for work at 1030hrs., she works at Car-Lene Market Research inside the York Town Mall in Lombard. Randy Liebich said that Steven sat down at the kitchen and ate a bowl of Apple Jacks cereal but didn't drink the milk. Randy said that Steven didn't want to eat anything yesterday. After eating his cereal, Steven then played around with the small dog they have.

M T Dolan

MR12

CLEARED

DuPage County Sheriff's Police
Follow up Report

ORIGINAL

Incident # 02-4531

	TYPE OF INCIDENT	DATE OF FOLLOW-UP	TIME	Complainant's name
0110	Homicide	2-8-02	1950	Chicago PD

Randy related he (Randy) was on the mattress in the living room and fell asleep around 1430hrs. and woke up at 1500hrs. When he got up he made Steven a hot dog. Randy said that Steven only ate half of his hot dog. Steven had a glass of water and when he began drinking the water he started to choke and then threw up. Randy related that he patted Steven on the back because he was breathing funny, he was wheezing. Randy asked Steven if he was OK, Steven verbally said, "Yea" and shook his head up and down. Randy laid Steven down by him on the bed which is on the floor. This bed is a mattress on the floor in the living room. Randy said that Steven moaned a little then he fell asleep. Kenyatta came home from work at 1600 or 1630hrs and woke him up. Randy informed Kenyatta of what happened with Steven. Because of the difficulty Steven experienced in his breathing they decided to take him to the hospital. Steven at this time was not talking, he just looked around. Randy carried Steven to the car, Kenyatta drove to the hospital. Randy related that Steven hadn't been eating, he wasn't saying much and he was not as active as he usually was. It was indicated that he still moved around and played but not as much. Steven was at his grandmother's residence for two weeks, he's been back with Randy and Kenyatta for the last three days. The last time Steven was at the doctor was two weeks ago, this was a general examine and he was fine.

Randy and Kenyatta have been living at the apartment on Mockingbird in Willowbrook for the last three months. Kenyatta's name is on the lease and they both contribute to the rent. They moved there because the rent is cheap, they only pay ninety-nine dollars a month. Before this, Kenyatta lived with Randy's parents in Hanover Park and then they moved to Palatine. Randy's mother didn't get along with Kenyatta. Randy has known Kenyatta for the last two years. They have plans on being married within the next couple of months. They met at a friend's house of Randy's in the city of Chicago, the friends name is Charles.

Randy works at the Patio Restaurant in Darien, he has been employed there for the last two and a half weeks. Prior to this job he worked at Foot Locker in the York Town Mall in Lombard as a manager. He lost that job after Christmas.

Randy indicated that Steven is an active kid and that he does cry a lot. His little girl Anglique cries too and sometimes Randy is unable to get any sleep. Randy said that Steven is a bed wetter. They place a diaper on him at night because he sometimes has accidents, usually they need to remind him to go. They have to ask Steven about going poop to, sometimes he goes in his diaper. Sometimes he goes on himself and other times not.

Randy was asked what kind of clothing Steven had been wearing yesterday. It was indicated he wore black sweatpants and a blue Rock a Wear sweatshirt.

The following were some points that were gone over. Steven was still sleeping when Kenyatta left for work. When Kenyatta left, Randy called out for Steven to get up, he did and came over to the kitchen table to eat his cereal. Going back to the time Randy said that Steven threw up, Randy indicated he picked Steven up and Steven's body went limp into Randy. Randy did ask Steven if he was OK, Steven nodded his head that he was. Randy was asked if Steven fell or bumped his head at all in the apartment. Randy related that Steven did not fall in the apartment at anytime that morning. Randy fed infant Angelique baby formula around 1030hrs and again fed her formula when Steven was eating his hot dog. It was indicated that Anglique sometimes spits up her formula. Randy cleaned up Steven's throw up with napkins and the napkins were thrown in the garbage can at the apartment. After eating his hot dog, Steven laid down on the mattress in the living room and watched television.

MR13

OFFICER #1 | CLEARED

DuPage County Sheriff's Police
Follow up Report

ORIGINAL

	TYPE OF INCIDENT	DATE OF FOLLOW-UP	TIME	Complainant's name
0110	Homicide	2-8-02	1950	Chicago PD

Going over the story again, Randy related that everyone woke up at 1000hrs. Kenyatta got dressed and walked out the door after filling a bowl of cereal for Steven. Steven did eat all of his cereal in about five to ten minutes. Randy knew about the times he has indicated because there is a clock in the apartment. Steven then sat down on the mattress in the living room and watched television and patted the dog. Around 1400-1430hrs. Randy fell asleep on the living room mattress with Anglique who was in his arms. Steven was still roaming around. Sometime around 1430hrs or just after, Randy got up from sleeping on the mattress and made Steven a hot dog. Randy cut the hot dog up and poured ketchup on the plate too because that is what Steven liked. Steven ate half the hot dog, he had orange juice in a can also. Randy held his daughter during this period. Steven finished the can of orange juice and Randy poured him a glass of water. Steven started to drink the glass of water and started to choke. Randy indicated that Steven was gulping the water down fast. Because of the choking, Randy patted Steven on his back. When this was under control, they all laid down on the mattress in the living room and watched Jurassic Park 2 that was on tape in the recorder. The end of the movie was never seen, they fell asleep. Kenyatta came home between 1600-1630hrs and woke them all up. Kenyatta discovered that something was wrong with Steven and they all left the apartment at 1630hrs or shortly thereafter to go to the hospital. The ride to Mount Sinai was about thirty minutes long. Randy related that Steven never stopped breathing nor was he ever unconscious. Randy was asked if he ever left the apartment at all between the time Kenyatta left for work and the time she returned back. Just after the time Steven threw up and was down on the mattress in the living room, Randy said he left the apartment to go to the McDonald's to a girl there for a cigarette. He said he didn't know this girl personally but has seen her on occasion having a cigarette outside. He described this girl as being on the chunky side who has blonde hair and was about 18 years old. This girl gave him a cigarette and he returned back to the apartment, he was gone for less than 5 minutes. Randy left the door unlocked to the apartment and both kids were asleep during the time he was gone. When Randy returned back to the apartment everything was as he left it. Randy was asked about his dog. His dog is the type that is friendly and doesn't bark at all. Randy related that he and Kenyatta had not been involved in any arguing recently, that they have been getting along and were pretty much in love. The only two children at the apartment were Steven and Anglique. No one else stopped by the residence when Randy was there with the children, he was home with the kids by himself. Randy said he basically laid around the residence most of the day. He said he changed Steven's diaper that morning sometime after Kenyatta left for work because it was wet. Throughout the interview, Randy was asked if he wanted anything to drink and or eat.

2-9-02 0015hrs. This detective and Investigator Vrbos left the conference room and were told by a nurse to view Steven who had recently come out of surgery. Steven was seen in a critical unit room, nurse Tammy Smith who was inside the room said that Steven was declared brain dead, he was on life support.

2-9-02 0025hrs. This detective went to his unit and retrieved a tape recorder and cassette tapes.

2-9-02 0105hrs. This detective and Investigator Vrbos went back inside the conference room where Randy Liebich was seated and admonished him Miranda. Randy related that he understood his rights and signed the waiver form indicating so. He agreed to speak with us about what occurred with Steven again. These investigators stepped out of the room again. A taco salad was supplied to Randy.

2-9-02 0115hrs. These investigators went back inside the conference room and again spoke with y, his story was gone over again. He had denied harming Steven. His story was the same except for

OFFICER #1 [signature] CLEARED BY 775#170 DATE 3/5/02

DuPage County Sheriff's Police
Follow up Report

ORIGINAL

Incident # 02-4531

	TYPE OF INCIDENT	DATE OF FOLLOW-UP	TIME	Complainant's name
0110	Homicide	2-8-02	1950	Chicago PD

the following. Randy was told that the bruises on Steven's head were inconsistent with what he was telling us. Randy was asked how these bruises got there and if he ever fell. Randy said that Steven fell o his right side when he patted him on the back during the time he choked on the water. Steven fell to his right side and hit his head on the floor but Randy indicated it wasn't hard. Randy was asked if Steven appeared OK prior to him eating his hot dog and having his orange juice. Randy indicated that he was.

2-9-02 0210hrs. The interview with Randy ended. Randy wanted to go to the room where Steven was, he was instructed that he couldn't at his time. Randy then went with Lt Szalinski and Sgt Kunz to th family area room.

2-9-02 0230hrs. This detective, Investigator Vrbos, Det Delgiudice and Assistant S.A. Liam Brennan discussed this incident in the conference room.

2-9-02 0315hrs. Randy was approached again by this detective and Investigator Vrbos in the family area waiting area. Randy was brought over to see Steven and to view the injuries on Steven's body. Nurse Tammy Smith was also in the room. A conversation with Randy concerning the condition of Steven was conducted in this room. Randy was advised that Steven was clinically brain dead. These investigators questioned him as to why this happened and that his story on the events that occurred Friday were inconsistent with the severe injuries sustained by Steven.

2-9-02 0325hrs. Randy returned back to the family waiting room.

2-9-02 0340hrs. This detective, Investigator Vrbos, Lt Szalinski and A.S.A. Liam Brennan spoke in in the conference room discussing this incident. Lt. Szalinski was on the telephone with Det Sgt who was at the Willowbrook apartment.

2-9-02 0405hrs. Hospital waivers were filled out for a full set of medical records on Steven Quinn, these were signed by Randy and Kenyatta. Based on the information received, A.S.A. Brennan indicated that Randy Liebich and Kenyatta Brown were to accompany us to the Sheriff's Office for further investigation into this matter.

2-9-02 0515hrs. This detective spoke with Price on the phone, he read small portions of Kenyatta's diary. Det Sgt Kunz had supplied information on this. He had spoken with Kenyatta who told him that the diary was in her purse inside the apartment and gave permission to retrieve it.

2-9-02 0550hrs. This detective and Det Degiudice checked Randy Liebich's car, a red 1989 Grand AM, Illinois plate of 218-5639, vin 1G2NE54D81C257240 parked outside in front of the emergency room. The keys were obtained from Kenyatta. Nothing of evidentiary value was located within. The vehicles trunk would not open. Kenyatta had told us that the trunk would not open even with the key.

2-9-02 0700hrs. This detective left Rush hospital with Investigator Vrbos and Randy Liebich.

2-9-02 0750hrs. This detective initiated a stop for food at McDonald's on the corner of Rt 38 and County Farm Road in Wheaton. An orange juice and potato cake was purchased for Randy. After the order was made, Randy related that he didn't like McDonald's so we drove across the street to Burger King where Randy was ordered a chicken sandwich by his request.

2-9-02 0800hrs. Arrived at the Sheriff's Office, Randy was provided with a seat in the interview room where he ate.

2-9-02 0830hrs. A.S.A. Michael Reidy arrived at our office. Deputy Chief Ray Bradford from the DuPage County Children's Center was notified and was en route to our office.

MR 15

OFFICER #1

CLEARED

DATE 2/9/02

DuPage County Sheriff's Police
Follow up Report

ORIGINAL

Incident # 02-4531

	TYPE OF INCIDENT	DATE OF FOLLOW-UP	TIME	Complainant's name
0110	Homicide	2-8-02	1950	Chicago PD

2-9-02 0840hrs. Randy threw up in a waste paper basket inside the interview room. Randy indicated that he was addicted to a twenty dollar a day heroin habit. Randy said that he has been on the synthetic narcotic Methadone for the last three weeks and his supply was out.

2-9-02 0900hrs. Lt Szalinski paged polygraph examiner Richard O'Brien.

2-9-02 1025hrs Richard O'Brien answered the page and indicated that he would be on his way to our office.

2-9-02 1045hrs. This detective paged Det Ledogar and spoke with him by phone. Det Ledogar was requested to stop by the McDonald's across the street from Randy and Kenyatta's apartment in Willowbrook and locate the chunky eighteen-year-old blonde girl who Randy said gave him a cigarette yesterday afternoon.

2-9-02 1150hrs. Richard O'Brien arrived at our office. O'Brien was briefed by Lt. Szalinski on what had transpired in this incident.

2-9-02 1215hrs. Richard O'Brien made his original contact with Randy Liebich in the interview room. Randy indicated to him that he didn't want to take the test today.

2-9-02 1330hrs. Lt. Szalinski conducted an interview with Randy Liebich in the interview room.

2-9-02 1355hrs. Richard O'Brien made original contact with Kenyatta Brown in the second interview room. It was decided by O'Brien that the polygraph test would not be given to Kenyatta today. Kenyatta did agree to take the test tomorrow.

2-9-02 1400hrs. Investigator Ray Bradford from the DuPage County Children's Center joined Lt Szalinski and Randy Liebich in the interview room.

2-9-02 1455hrs. Kenyatta Brown asked Richard O'Brien if she could speak with Randy Liebich about what happened to her son. Richard O'Brien brought Kenyatta into the interview room where Randy was seated.

2-9-02 1510hrs. Both were separated, Kenyatta returned back to the interview room she was originally in.

2-9-02 1640hrs. It was decided by the State's Attorney Office that both Kenyatta Brown and Randy Liebich would not have any criminal charges filed against them at this time. They both left our office several minutes apart from one another.

MR16

OFFICER #1 B. Jay Paxson

CLEARED BY 175#176

DATE 2/5/02

DuPage County Sheriff's Police
Follow up Report

ORIGINAL

Incident # 02-4531

ODE	TYPE OF INCIDENT	DATE OF FOLLOW-UP	TIME	Complainant's name
10	Murder	020802	2012	.Chicago PD

code	NAME: LAST, FIRST, MIDDLE	ADDRESS	CODE	D.O.B.	S	R	TX HOME	TX BUS
V	Quinn, Steven M	16w505 Mockingbird #204	793	041799	M	B	None	None
S	Liebich, Randy R	16w505 Mockingbird #204	793	071279	M	W	None	3237427
W	Brown, Kenyatta M (Keke)	16w505 Mockingbird #204	793	100683	F	B	None	7051303
W	Clark, Karen L	714 S. Independence #3fl	165	101564	F	B	7737227976	773-736-9636
W	Sgt. Kunz #718	DCSO	512					N/a
W	Rush-Presbyterian St.Luke's Medical Center	1653 W. Congress Parkway-622 Murdock	165					312-942-6194
W	Herron, Dorothy J	4937 W. Augusta Blvd	165	111049	F	B	7733799440	Cell-773-419-8317
W	Liebich, Angelique M	16w505 Mockingbird #204	793	012702	F	B	none	None

POLICE ACTION: REFER TO STATES ATTORNEY [ ] COMP. SIGNED YES [ ] NO [ ] ARRESTS: TYPE ____ # OF PERSONS _____ OTHER ____

NAME OF ARRESTEE	CHARGE	STATE #	I. R. #	C.C OR TICKET #

NARRATIVE:

LEAD 6

020802 2330hrs

Sgt. Kunz and myself interviewed Karen Clark, Steven Quinn's grandmother and Kenyatta Brown's mother, in an empty exam room on the 5th floor of the Pediatric Intensive Care Unit at Rush-Presbyterian- St. Luke's Medical Center.

Karen said she first learned of this incident when Kenyatta called her when they arrived at Mt. Sinai Hospital in Chicago this evening. She was asked what Kenyatta and Randy Liebich had told her about how Steven had received his injuries today. Karen said Kenyatta told her when she arrived home from work this afternoon she found Steven unresponsive and Kenyatta said he appeared to be very sick. Kenyatta indicated Steven was lying on the floor on a blanket at the time. His eyes were rolling around and he had thrown up. Kenyatta also told her mother Randy was alone with the two children

OFFICER #1	CLEARED BY	DATE
Det. J. Del Giudice #141	#176	2/18/02

MR17

DE	TYPE OF INCIDENT	DATE OF FOLLOW-UP	TIME	Complainant's name
0	Murder	020802	2012	.Chicago PD

(Steven Quinn and Angelique Liebich) all day. Karen said Kenyatta worked from 1000-1600hrs today at Car-Lene's Marketing Research in Lombard. Karen said Kenyatta told her that when she found Steven unresponsive, she asked Randy what had happened to him. Randy responded by saying he had no idea what happened to Steven. Kenyatta also told Karen that Randy fed Steven a hotdog for lunch and Steven choked on the hotdog, but that was all she knew at this time.

Karen said Kenyatta was 15 years old when she had Steven and Steven had a trace of marijuana in his system when he was born. Karen became Steven's legal guardian due to the circumstances. Karen also said her sister-in-law, Dorothy Herron, helped take care of Steven for the past 2 years. Dorothy would watch Steven up to 2-3 weeks at a time. Kenyatta would see the child and have him for a couple of days at a time, but either Dorothy or Karen would take Steven back to their residences. Karen said Kenyatta had her newborn child (Angelique) at Hinsdale Hospital. Karen last cared for Steven approximately 2 weeks ago. Since then, Dorothy and Kenyatta had taken turns caring him. Karen spoke to Kenyatta via the phone on Thursday (020702) and learned Steven had been her since Saturday (020202) when he was picked up at Dorothy's residence. Karen said Kenyatta sounded fine when she talked to her.

Karen was then asked about her relationship with her daughter, Kenyatta. Karen indicated Kenyatta had a behavior problem when she was young and she ran away a lot. Kenyatta had become an "excellent liar" and got into trouble continuously. As of late, their relationship had progressed in a positive way. Karen said they would talk and see each other on a more frequent basis. The last time Karen saw Kenyatta was on Sunday (020302), when Kenyatta had come to her house during the afternoon hours. They met in the hallway when Karen was leaving to run an errand. Karen said Kenyatta looked real funny like she had just been fighting with Randy. Randy was in his car with Angelique, although she did not see Steven because she did not go over to the car to talk to Randy. Kenyatta went into the residence to retrieve some food and Karen went on her way. That was the last time she saw them until today.

Karen told us Kenyatta and Randy had been together for the past 2 years. At one point, they were separated for approximately 3 months. Karen could not provide the approximate dates of their

DE	TYPE OF INCIDENT	DATE OF FOLLOW-UP	TIME	Complainant's name
0	Murder	020802	2012	.Chicago PD

separation. She went on to describe their relationship as a violent one. She said Kenyatta had called her several times during their relationship complaining about Randy beating her and physically abusing her. Karen said she did not see Randy hit Kenyatta at any time while she was around them. Kenyatta told her about an incident at their Joliet residence in the summer of 2001. Apparently, Kenyatta told her Randy had either tied her up and beat her or locked her in a closet during a fight. Kenyatta described to her how she had to climb out of a window to escape Randy's violent advances towards her. Karen believed the Joliet Police were summoned for that particular incident. Kenyatta also told her about another incident in Joliet involving Steven. One morning Kenyatta woke up and found Steven's face swollen and she described it as if someone had beaten him. Kenyatta confronted Randy about the situation and he told her the dog must have injured Steven. Karen did not see Steven's face at that time. Karen said Randy has a bad temper. She has observed him yell at Kenyatta and seen him get red faced when he is upset. Karen said Randy has no patience and is very possessive of Kenyatta. One ample she gave of his impatience was when Randy came over to Karen's house on one occasion to ve Kenyatta, he pressed the doorbell and kept ringing it until she came out.

I asked Karen if she ever observed or knew of either Kenyatta or Randy ever physically striking Steven in any way. She said Kenyatta has slapped Steven in the face a few times in the past due to his whining and crying. In regards to Randy, she never saw him hit Steven. She remembered one occasion when Steven cried and did not want to go with his mother because Randy was with Kenyatta when she came to pick him up. Her explanation for his outburst that time was because he saw Randy hit his mother in the past and he was afraid. She went on to describe Steven as a calm "cuddler" type. She said he was not "wild" but he did cry and whine a lot for whatever reasons. I asked her if she has ever seen any injuries or bruising on Steven when he came back from staying with Randy and Kenyatta. Karen said no. The only marks she knows about are the faded pink spots on his lower back that were here when he was born. I then asked Karen what she believed happened to Steven to cause the extensive injuries he suffered. She thought Steven may have been crying and Randy became upset nd could not deal with it and he hurt Steven.

MR 19

CER #1	CLEARED	
Det. J. Del Giudice #141	BY 775#726	DATE 2/18/02

E	TYPE OF INCIDENT	DATE OF FOLLOW-UP	TIME	Complainant's name
J	Murder	020802	2012	.Chicago PD

At this point, Karen could not think of anything else to add, so the interview ended at 1220hrs on 020902.

MR20

CER #1	CLEARED BY	DATE
Lieut. J. Del Giudice #141	TJS #774	2/18/02

DuPage County Sheriff's Police
Follow up Report

ORIGINAL

Incident # 02-4531

	TYPE OF INCIDENT	DATE OF FOLLOW-UP	TIME	COMPLAINANT'S NAME
01	Homicide	02/14/02	1000	Chicago P.D.

CODE	NAME: LAST, FIRST, MIDDLE	ADDRESS	CODE	D.O.B	S	R	TX HOME	TX BUSINESS
W	Herron, Dorothy J.	4937 W. Augusta Blvd.	165	11/10/49	F	B	773)379-9440	Cell 773)419-8317

POLICE ACTION: REFER TO STATES ATTORNEY [ ] COMP. SIGNED YES [ ] NO [ ] ARRESTS: TYPE ____ # OF PERSONS _____ OTHER ____

NAME OF ARRESTEE	CHARGE	STATE #	I. R. #	C.C OR TICKET #

NARRATIVE:

LEAD #19 On 02/09/02 0020 hrs. I spoke to Dorothy Herron, Steven Quinn's Great Aunt, in an empty room on the 5th floor of Pediatric Intensive Care Unit at Rush-Presbyterian-St. Luke Medical Ctr. She advised me that Kenatta and Randy picked up Steven between 1900 – 2000 hrs on Saturday 02/02/02. Dorothy advises that when Steven left with Kenyatta and Randy, Steven had no scratches or bruises. Dorothy advised that she had Steven with her for approximately two weeks prior to Kenyatta and Randy picking Steven up. Dorothy advised that in her opinion Steven seemed a little apprehensive to go with Kenyatta and Randy. Dorothy also advises that in the past Steven has made statements such as 'Randy Fight' or 'Randy hit'. Dorothy is not sure which statement Steven said. Dorothy advises that when she does have Steven, after he has been with Kenyatta and Randy, she doesn't remember seeing any bruises, but does remember one time when Steven had a scratch on his neck. Dorothy doesn't know what caused the scratch, but did add that it wasn't very big. Dorothy states she hasn't talked to Kenyatta or Randy since they picked Steven up, but added that this is normal. Dorothy advises that she has heard Randy has a bad temper, but she has never witnessed it. Dorothy advises she has never seen Kenyatta or Randy hit Steven. Dorothy states she thinks Randy uses drugs, because he "sometimes looks wacey". Dorothy advises that while in the Family waiting area in the hospital, she asked Randy what happened to Steven and Randy said, "I don't know, he fell". Also while in the Family waiting area, Dorothy states that family members were questioning Randy about Steven's condition, Randy stated to Kenyatta 'why don't you tell them you whooped him the other day?' Dorothy also adds that while in the

OFFICER #1		CLEARED BY	DATE
Det. Sgt. Kunz #718	02/21/2002	TS #176	2/22/02

EK 718

	TYPE OF INCIDENT	DATE OF FOLLOW-UP	TIME	COMPLAINANT'S NAME
0	Homicide	02/14/02	1000	Chicago P.D.

Family waiting area; Kenatta observed what appeared to be blood on the infant's (Angelique) blanket. Kenyatta asked Randy, where the blood came from? Randy stated, "That's not blood, that's make-up." The evidence technician collected the blanket. Dorothy had nothing more to add. Interview concluded at 0100 hrs.

MR 32

OFFICER #1		CLEARED BY	DATE
Det. Sgt. Kunz #718	02/21/2002	TS #176	2/22/02

S K 718

INCIDENT #
02-4531

DUPAGE SHERIFF'S POLICE
FOLLOW-UP REPORT

ORIGINAL

CODE	TYPE OF INCIDENT
0110	Homicide

	N	OFFICER - RANK Initial. Name & #	Agency or Address	Code	CODE	ON	OFFICER - RANK Initial. Name & #	Agency or Address	Code
					O2				

CODE	ON	NAME LAST FIRST MIDDLE	ADDRESS	CODE	D.O.B.	S	R	TX HOME	TX BUS
S		Liebich, Randy R.	16W505 Mockingbird Ln. Apt 204	793	071279	M	W	none	920-0211
W		Bradford, Ray (Investigator)	DuPage Children's Center						784-3727

NAME OF ARRESTEE	CHARGE	C.C. OR TICKET #

DATE OF FOLLOW-UP:	TIME OF FOLLOW-UP:

NARRATIVE:

LEAD #3

2/9/02 1330hrs

I met with Randy Liebich in interview room 2095 at the Sheriff's Office. Introductions were made and I asked Randy if we could review the events of Thursday evening and Friday. Randy agreed and the following is a summary of that interview:

Randy was asked about the usual routine for getting Steven to sleep at bedtime. Randy advised that Keke (Keke is the nickname that Randy used for Kenyatta) takes care of Steven and specifically mentioned that he does not change diapers. On Thursday evening, at about 8pm Randy was in the bedroom of their apartment along with Keke and Steven. Randy advised that Steven was crying and that Keke was disciplining him by striking him with a belt across the buttock. Steven was wearing a diaper at the time and it was Randy's opinion that Steven was not feeling the strikes due to the padding of the diaper. Steven could not recall how many times Keke struck Steven other than to say "a few times". Keke then removed the diaper and spanked Steven on the buttock with her bare hand. Randy was asked if Keke struck Steven anywhere else on his body with the belt besides his butt and he said that she did not. Randy also advised that Keke did not strike Steven anywhere other than his butt. Steven then was left alone until around midnight when he started to cry again at which he and Keke entered the bedroom. Randy then offered that he observed Keke "pop" Steven in the head. When asked what he meant by "pop" Randy demonstrated a motion similar to an an open hand slap but with the impact coming from the palm of the hand. Randy said that Keke gave a few strikes of this type but could not be specific on the number of strikes. Randy said that during this time Keke did not strike Steven anywhere else on his body other than in the head. Randy advised that he considered the earlier strikes with the belt and the open hand spanking as normal discipline, but that the "pops' in the head made him uncomfortable. Randy advised that he did not strike Steven at any time.

Randy advised that he and Keke awoke around 9:30am and Keke was getting ready to go to work. Keke left just before 10am. Randy was asked if Steven was disciplined by himself or Keke on Friday morning before Keke left for work. He advised that he did not and that he did not see or hear Keke discipline or otherwise strike Steven on Friday morning.

MR 48

ORIGINAL

S advised that Keke had put a cereal bowl out on the kitchen table and told Randy that he should fee even. Randy advised that shortly after Keke left the apartment he picked up Steven and put him in the car seat that they have on a chair at the kitchen table. Randy poured milk into a bowl of Apple Jacks cereal which was in front of Steven. Randy also said that Steven had a glass of orange juice and a glass of water at his place with the cereal. Randy was asked specifically how much of the breakfast Steven ate. Randy replied that Steven ate all his cereal, drank most of the orange juice, but that he did not drink the water. Randy estimated that the breakfast lasted only about five minutes. Randy advised that he cleared the table of the dishes and lifted Steven out of his chair and set him on the kitchen floor.

After breakfast Randy advised that he went over to the area of the living room where he and Keke sleep on a mattress on the floor. He had turned on the TV and a VHS tape which contained a recording of *Jurassic Park 2* and some other movies. Steven walked over to where Randy was, sat down and the two of them began to watch the video. Randy estimated that approximately 30 minutes into the movie, he dozed off for approximately 30 minutes. When he awoke Steven was under the dining room table with the dog. I asked Randy if Steven could have hit his head on the table or been pushed into it by the dog and he advised that he did not think so and that Steven was quiet and was just "chillin with the dog." I asked Randy if the dog had a leash that Steven could have gotten tangled in. Randy said that they don't own a leash and don't use one when the dog is let outside. At this point, Randy advised that he changed Steven's diaper. I asked if he noted any marks on Steven while he was changing him and Randy said that he did not. Randy said he gave Angelique a bottle and that he lied back down on the mattress in the living room with her and continued to watch the movie. Randy said Steven walked over to him and sat down and was watching the as well. I asked Randy how Steven was. Randy advised that Steven seemed "dazed". When to explain what he meant by "dazed" Randy said that Steven had been quiet all day and that he was st ng up with his eyes open staring at the TV.

Randy said that he must have fallen asleep again while the movie was on. I remarked that *Jurassic Park 2* must have been over at about that time. Randy said other movies came on after *Jurassic Park 2* but that could not remember what they were titled or what they were about. Randy said that about 1pm he woke and Steven and the baby Angelique were sleeping so he decided to go out for a smoke. Randy said he went over to the near-by McDonald's Restaurant were he saw a girl whose name he did not recall. (See Detective Ledogar's report on LEAD#5) Randy described her as a "neighbor" and said she gave him a cigarette. Randy then returned to the apartment and found the children still asleep in the same position as when he left. I asked Randy if he locked the apartment door when he departed to smoke and he related that the door does not lock. I asked if he thought someone might have entered the apartment while he was away at which he said that he did not think so because he was only gone five minutes and the children were asleep as they were when he left.

asked Randy what he and the children did after he returned from the trip to McDonalds. He said that he gave Steven a hotdog and some water at about 3pm and that Steven started choking on the water. I told Randy that we would talk about the 3pm meal, but first I wanted to talk about what he and the children did have almost two hours between the time he got back from the McDonalds and the 3pm hotdog meal.

MR 49

ORIGINAL

2/9/02 1400hrs

At this point Investigator Ray Bradford joined me in the interview room. In summary, Bradford said that he had heard form the hospital regarding Steven's condition. Bradford told Randy that Steven was not expected to survive and asked Randy to help explain Steven's injuries. Bradford told Randy that there is no doubt that the injuries to Steven had occurred during the time that he was caring for him and he (Bradford) needed Randy to explain how they occurred. Randy denied that he caused any injury to Steven and further stated that he has never struck Steven. Bradford told Randy that the evidence is clear and that now is the time to tell us what really happened. Randy listened intently to Bradford and leaned forward in his chair and put his face in his hands for a short period. Randy's eyes welled with tears and he turned his head down and to the right in an apparent attempt to wipe a teary eye on his jacket collar. Bradford continued to ask Randy to explain how Steven was injured and that we (detectives) know that he didn't mean to hurt him. Randy was still slummed forward and was nodding his head as if in agreement but would not confirm or acknowledge his involvement in Steven's injury.

/9/02 1415hrs

fter several minutes of interview by Ray Bradford, Randy stated that he wished to talk to a lawyer at 415hrs and asked to use a telephone. I left the room to obtain a phone. Shortly thereafter I handed radford a phone and left the room. Randy made several telephone calls and then asked Mr. Bradford if e 'd see Keke.

2/9/02 1437hrs

r. Bradford left the interview room.

/9/02 1456hrs

. O'Brien brought Keke into room 2095 where Randy was seated. In summary, Keke implored Randy to her what he did to Steven. Randy repeated several times "I didn't do all that to him". This went on for veral minutes with Keke crying and asking Randy to tell her what happened. Randy continued to say he not hurt Steven and told Keke that he needed to hug her and said that he loved her. Keke declined the and continued to asked Randy to tell her what happened to no avail.

'02 1512hrs

O'Brien and Keke left the room.

MR50

Szalinski #176

	OFFICER #2	SUPERVISOR	DATE
T. Szalinski			022102

ON 02-14-02 AT 1630 HRS, I WAS ADVISED BY DISPATCH THAT I HAD A FAMILY MEMBER IN THE LOBBY.

MY COUSINS, DION AND RANDY LIEBICH WERE WAITING FOR ME. I BROUGHT THEM INTO THE ROOM NEXT TO DISPATCH, AND RANDY WAS ASKING IF IT WAS SAFE TO TALK IN THIS ROOM. I GAVE HIM A QUIZZICAL LOOK, AND HE ASKED IF IT WAS "BUGGED". I SAID "NO".

RANDY SAID HE WANTED TO TALK TO ME AND LET ME KNOW HE WASN'T RUNNING, AND THAT HE HAS BEEN TALKING WITH THE DETECTIVES HANDLING THE CASE. RANDY ALSO WAS CONCERNED ABOUT THE CUT ON HIS FINGER (RIGHT INDEX), SAYING THE KID BIT HIM AND DREW BLOOD. I SAID, "SO WHAT". RANDY WAS WORRIED ABOUT HIS BLOOD BEING INSIDE THE KID'S BODY. I ASKED RANDY WHAT HAPPENED, AND HE STARTED EXPLAINING EVENTS ON 02-07-02 AND 02-08-02.

RANDY SAID HIS GIRLFRIEND "KIKI", WAS HITTING HER CHILD IN THE EVENING OF 02-07-02 BECAUSE HE KEPT COMING OUT OF THE BEDROOM. RANDY SAID HE LIT A CIGARETTE AND WENT INTO THE ROOM. RANDY SAW KIKI HIT STEVEN IN THE HEAD FOUR(4) TIMES. RANDY TOLD HER THAT SPANKINGS SHOULD BE ON THE BUTT. KIKI TOOK A BELT AND STRUCK STEVEN ON THE BUTT. MR 10d

IN THE MORNING, RANDY SAID STEVEN WAS LYING IN BED WITH HIS EYES OPEN. RANDY SAID STEVEN NORMALLY

KIKI TOLD STEVEN TO GET OUT OF BED. STEVEN DID GET OUT OF BED AND WALKED OUT ON HIS OWN. KIKI WENT TO WORK, AND LATER RANDY WAS FEEDING STEVEN A HOT DOG. RANDY SAID STEVEN WAS CHOKING AND HE HIT STEVEN ON THE BACK TO DISLODGE THE FOOD. RANDY PUT HIS FINGERS IN STEVEN'S MOUTH TO PULL OUT THE FOOD AND STEVEN BIT DOWN ON RANDY'S FINGER (RIGHT INDEX). RANDY COULD NOT GET STEVEN TO LET GO, SO HE HIT STEVEN IN THE HEAD WITH AN OPEN HAND. RANDY SAID HE HIT HIM ON THE OTHER SIDE THAN WHERE KIKI HAD HIT HIM THE NIGHT BEFORE. RANDY SAID HE HIT STEVEN AGAIN AND THAT'S WHEN STEVEN LET GO. STEVEN THREW UP AFTER THIS AND JUST WASN'T ACTING RIGHT. KIKI CAME HOME A SHORT TIME LATER, AND THEY BOTH TOOK STEVEN TO THE HOSPITAL.

RANDY TOLD ME THAT HE DIDN'T TELL THE DETECTIVES ABOUT THE BITE OR HITTING STEVEN. RANDY ~~ASKED WHAT HE SHOULD DO, AND I TOLD HIM TO TELL~~ THE TRUTH. I ASKED RANDY SEVERAL TIMES IF HE HIT STEVEN HARD ENOUGH TO DO THIS. THE LAST TIME HIS EYES LOOKED AT THE FLOOR, AND I WAS UNCOMFORTABLE BELIEVING HIS ANSWER. I ASKED HIM TO SWEAR ON HIS ~~FATHER'S GRAVE, RANDY LOOKED ME RIGHT IN THE EYE AND~~ THEN SAID HE DIDN'T HIT THE KID THAT HARD.

~~RANDY ASKED IF I HEARD ANYTHING TO LET HIM KNOW.~~ I SAID NO, IF HE WAS LYING TO ME, THEY WILL COME FOR YOU.

MR 103

FAGUE 20-10-0007

DION SAID AN UNMARKED CAR WAS WAITING WHEN THEY ARRIVED. WE BOTH ASSUMED RANDY HAD BEEN ARRESTED. IT WASN'T UNTIL A WEEK LATER THAT I FOUND OUT RANDY HAD NOT BEEN ARRESTED, AND I CALLED DETECTIVE FIGIEL TO INFORM HIM OF MY CONVERSATION WITH RANDY.

Robert Fague 022702

NOTE

COMPLETED AT ROSELLE P.D.
2-27-02 11:00 AM

MR-104

OFFICE OF THE CIRCUIT COURT CLERK

CHRIS KACHIROUBAS, CIRCUIT CLERK

18th JUDICIAL CIRCUIT COURT - DU PAGE COUNTY

4 / 1 /09

THE PEOPLE OF THE STATE OF ILLINOIS

VS

Randy Liebich # R34940
Defendant

Case No. 02 CF 654

FILED
09 APR -1 PM 4:04
CLERK OF THE 18TH JUDICIAL CIRCUIT DU PAGE COUNTY, ILLINOIS

This letter will acknowledge receipt of the following:
Letter / Supplemental documents

which has been filed in the record in the above captioned case on 4/1. Copies of this filing have been forwarded to the following:

- ☐ The Honorable **Ann B. Jorgenson,** Chief Judge of the 18th Judicial Circuit Court
- ☐ The Honorable **Kathryn E. Creswell,** Presiding Judge, Felony Division
- ☐ ~~The Honorable **Michael J. Burke,** Presiding Judge, Misdemeanor and Traffic Divisions~~
- ☒ The Honorable John Kinsella (4000), Trial Judge
- ☒ The Honorable **Joseph E. Birkett,** State's Attorney of DuPage County
- ☐ The Honorable **Lisa Madigan,** Attorney General of the State of Illinois
- ☒ DuPage County Public Defender
- ☐ Sent to the Second District Appellate Court
- ☐ _____

for their further action pursuant to the laws of the State of Illinois or the Supreme Court Rules. You will be advised of the outcome of those proceedings **if required by law or rule of court.**

Sincerely,

Chris Kachiroubas
Circuit Clerk

Prepared by

02CF654

Dear Clerk of the Court,

My name is Randy Liebich and I am a Prisoner at The Stateville Correctional Center. My case number is 02-CF-654.

On March 3, 2009 My Petition for Post Conviction Relief was Filed. In support of my petition, I have additional exhibits to supplement thereto.

I therefore supplement my petition with the following documents:

1). Supplement to Page-14 of my original Petition for Post conviction relief. (1-Page)

2) Signed Affidavit of Denise Foster (2-Pages)

3) Original signed Affidavit of Dion Liebich (4-Pages)

4) Miranda waivers dated (2-13-02) and (3-1-02) in support of my supplemental Affidavit. (2-Pages)

Respectfully Submitted,

Randy Liebich

Randy Liebich R34940
Stateville Correctional Center
Po Box 112
Joliet, IL 60434

this case become the subject of federal habeas proceedings. I expect to file several expert affidavits or reports, most likely from Professor Patrick Barnes, Professor of Radiology at Stanford University and Director of Pediatric Neuroradiology at Lucile Salter Packard Childrens Hospital; Dr. Peter Stephens, a forensic pathologist; and Dr. George Nicholls, former Medical Examiner for Kentucky.

11. Since I am without any income or assets with which to retain counsel, I ask that counsel be appointed to represent me in this proceeding.

Respectfully submitted,

Randy Liebich

Randy Liebich
I.D. Number 34940
Stateville Correctional Center
P.O. Box 112
Joliet, IL 60434

Subscribed to and sworn to before me this 23rd day of FEBRUARY, 2009.

S K Miles

Notary Public for the State of Illinois

OFFICIAL SEAL
SHERWIN K. MILES
NOTARY PUBLIC, STATE OF ILLINOIS
MY COMMISSION EXPIRES 5-20-2012

I SWEAR THAT THE FACTS STATED IN THIS PETETION ARE TRUE AND CORRECT IN SUBSTANCE AND INFACT.

Randy Liebich

Randy Liebich R34940

STATE OF ILLINOIS)
)
COUNTY OF DUPAGE)

AFFIDAVIT OF DENISE FOSTER

I, Denise Foster, being first duly sworn on oath, depose and say as follows:

1. My name is Denise Foster. My address is 1231 Prairiebrook Drive, Apt. A3, Palatine, IL 60074. I have worked at a daycare center at a health club for twenty years. I am Randy Liebich's sister.

2. I testified at Randy's trial about Kenyatta's treatment of Steven. Kenyatta did not take care of Steven. She paid no attention to him, she hit him regularly, and she did not do the things that a mother would normally do. Since I work in daycare, I know how mothers and children behave, and Kenyatta did not have a mothering or nurturing instinct towards Steven. She expected behavior that was not age appropriate, and she would hit him to make him stop crying. This was all well known at the time of Randy's trial, and I testified to some of it.

3. Before Randy and Kenyatta got an apartment, Randy lived with my mother and Kenyatta would stay with them, sometimes bringing Steven. When I went over to visit one day, Kenyatta and my mother were arguing. I don't remember what they were arguing about, but when I turned my back, I heard a thud. When I turned around, my mother was lying on the floor by the door. When I ran over, she was unconscious. Randy also ran over.

4. I was going to call 911 but my mother came to quickly. My mother said that Kenyatta hit or pushed her, which was obvious, and she asked me not to call 911. I didn't call. From the way my mother had fallen, it looked like she had hit the door.

5. After seeing this, I was very concerned about Randy's relationship with Kenyatta. Randy always wants to see the best in people, but sometimes he is wrong. He does not have a temper, I have never known him to hit anyone, and he generally backs away and stays out of potentially violent situations, as he did with Kenyatta. We have many cousins (11 aunts and uncles on my dad's side, with more on my mother's side), so we had many family gatherings. At family gatherings, children always swarmed around Randy. Steven did the same. Randy is very good with children, but I did not think he would be able to stand up to Kenyatta. Instead, he made excuses for her.

6. The incident with my mother caused me real concern for Steven. We already knew that Kenyatta regularly hit and shoved Steven. My concern was that she would do with Steven as she had done with my mother, that is, push him, or shove him, or slam him into something, with serious consequences. The incident with

my mother, who was not a fighter, showed me that Kenyatta did not have much self-control and that she did not think of the consequences of her actions.

I swear under penalty of perjury that the foregoing is true and correct to the best of my knowledge and belief.

Denise Foster

Subscribed to and sworn before me this 26th day of February 2008.

Notary Public in and for the State of Illinois

My commission expires 9/30/2009

"OFFICIAL SEAL"
GUADALUPE MUNOZ
Notary Public, State of Illinois
My Commission Expires 9/30/2009

STATE OF ILLINOIS)
) **AFFIDAVIT OF DION LIEBICH**
COUNTY OF DUPAGE)

I, Dion Liebich, being first duly sworn on oath, depose and say as follows:

1. My name is Dion Liebich. I work for 6-D Aluminum, which is located in Crest Hill, Illinois (phone 708-487-1855). I am Randy Liebich's cousin.

2. In February 2002, I saw a television report saying or implying that my cousin Randy had murdered his girlfriend's son, Steven. I immediately called Randy's sister, Denise, who told me that she heard that Steven had been beaten when Kenyatta was at work and Randy was looking after him.

3. When I heard this, I was extremely angry. This was completely unlike anything I knew of Randy, and I could not believe he had done this. However, since he was the only one home, he had to know what happened.

4. Denise, Randy's sister, told me that Randy was at his mother's, and I drove there immediately and grabbed Randy by the back of his neck. Before we talked about what happened, we decided to go to see our cousin, Robert Liebich, who is a police officer in Rosselle, Illinois. I don't remember whose idea this was but I thought this would let us figure out what was going on. I gave Randy a ride to the Roselle police station. It was a very quiet ride. Since Randy generally did not trust the police and wasn't close to Robert, I thought that Randy was going to confess or at least tell Robert what really happened.

5. Robert took us in an interrogation room. Robert was very angry, and there were a lot of raised voices. There were no Miranda warnings or anything like that. This was a full interrogation, with a lot of anger directed at Randy. Robert and I wanted answers on how Steven died, and Randy wasn't giving us answers. Robert and I had kids, and we were angry with Randy for letting this happen to a child. We thought he had to know what happened since he was there.

6. I let Robert do the questioning since he was a police officer and experienced at doing this. Randy said that Steven choked on a hot dog and that Randy put his finger in Steven's mouth to see if something was stuck in his throat. When he did this, Steven clamped down on the index finger of his right hand. Randy said that when Steven wouldn't let go, he hit Steven on the side of the head to get him to let go. Randy showed us a small cut by his fingernail where he said Steven had bitten him.

7. Robert questioned Randy on how he struck Steven, and Randy illustrated. He showed a tapping from a few inches away that should not have hurt anyone. It

was almost like a push, more pressing than hitting. Robert went over this with Randy several times. Randy's description seemed like a natural instinctive reaction, exactly what I think most people would have done if someone was biting their finger. It did not seem like anything that would have hurt Steven.

8. The atmosphere in the room was very tense and filled with anger, almost hate, since nothing Randy described explained the serious injuries that we heard about. Robert kept telling Randy this but Randy kept telling the same story, over and over again. Robert and I were very angry that Randy wouldn't explain how Steven ended up dead.

9. Finally, Robert asked Randy if he would swear on their fathers' graves that he was telling the truth and the whole truth. Randy was very close to his father, who died when he was a teenager. Robert's father had also died fairly recently. In our family, this type of oath means a lot. Randy said that he swore on their fathers' graves that he was telling the truth.

10. At first, Randy looked at the floor when he said this. I didn't put a lot of importance on this since Randy always looked at the floor when he was upset. When his father died unexpectedly, almost everyone was crying. However, Randy, who was very close to his father and the most upset, sat by himself with his head down. That is how he looked at the police station. Robert, who doesn't know Randy as well as I did, asked the same question again, maybe in different words, and Randy looked Robert straight in the eye and repeated that he swore on their father's graves that he was telling the truth and that he had not hurt Steven.

11. Randy talked about Steven not feeling well the week before he collapsed. He also said that Steven didn't seem that sick after he choked on the hot dog but that after Kenyatta came home they noticed that he was making odd moaning noises and they took him to the hospital. Randy said he took the dog out first. Robert and I didn't pay much attention to any of this since it didn't explain how Steven died.

12. Robert was angry and frustrated because Randy kept saying the same things over and over and wasn't giving any explanation for Steven's condition. Since Robert's father died of a cocaine overdose, Robert despises drugs and looks down on people who use them, including Randy. He was very harsh, therefore, in interrogating Randy. He wanted a confession, and he wasn't getting it.

13. I did not know what to believe. I knew Randy much better than Robert did since I spent a lot of time with him after his father died. Randy could be easily persuaded to do things, but he was never violent and he was really good with kids.

14. Robert kept asking me things like, "what do you think?" I didn't know what to think. Robert said he was trained to know when people were lying and that he knew for a fact that Randy was lying. He seemed to think Randy was lying because he hung his head, but I knew that Randy did this when he was depressed.

15. Robert asked Randy over and over if he could have hit Steven hard enough to hurt him or kill him. Randy always said no. I asked the same questions, and Randy gave the same answers. When Robert asked important questions like this, Randy would look him straight in the eye and say that he didn't do anything that could have hurt Steven and that the only unusual thing that day was when Steven choked on the hotdog. He kept saying that he didn't know what happened to Steven. Robert and I were angry that a three year old had been killed and no one could give an explanation.

16. When Randy was going with Kenyatta, I helped him get an apartment and a job. When I picked him up for work, he talked about problems with Kenyatta. I didn't like the situation with Kenyatta, and I didn't want to hear about the problems.

17. When I visited Randy ~~after Angelique was born~~ DSK, it was over ninety degrees in their apartment, and Kenyatta was frying chicken in the kitchen. It was much too hot for the children, so I went out and bought an air conditioning unit and put it in the window. When Steven went over and started to play with the knobs, Kenyatta grabbed him by the left arm, opened the door to the bedroom and chucked him in, like she was throwing a baseball.

18. I saw this from the kitchen table, and I jumped up and started going after Kenyatta, saying, "how could you do this?" Randy got in the middle, and I yelled at him too, saying, "how can you let this happen?" I told both of them I would call the department of child services if I ever saw or heard about anything like this again. I told my wife about this after I got home.

19. Earlier, Randy told me that Kenyatta would use broken coat hangers to hit Steven. I didn't know why Randy didn't stop it but I don't think he thought he could. Kenyatta was a strong personality, and I don't think she would have listened to him. I was mad with Randy because I felt he was condoning it by staying with her. However, it may have been worse if he left.

20. In all the years I have known Randy, I have never seen him harm or hurt a child. He has been around many children, including my own son, and he never showed a mean streak and rarely showed impatience. When he and Kenyatta lived with us, Randy and Steven got along well, and Randy seemed to like to have him around.

21. When we heard that Steven had been beaten, it occurred to me that if Steven had been beaten, it was more likely to be Kenyatta than Randy. We all knew that Kenyatta hit Steven, but no one had ever seen Randy hit Steven or any other child. I don't think that Robert knew any of this when he interrogated Randy.

22. Months after Steven died, detectives came to my home. They told me that Randy had hit Steven on the head so hard that his brain swelled and there was bleeding on his brain, and that this was the only explanation for his injuries. Since I was

present when Robert interrogated Randy and heard what Randy had to say, I thought that Randy's attorneys would also want to talk to me, but they never did.

23. Randy and Kenyatta lived with my wife and me for awhile, and they had some fights. I never knew who started them or what happened. One time, when my wife and I were home, Randy and Kenyatta had a fight in the bedroom. Randy left the apartment, and Kenyatta came out crying and saying that he hit her. I gave her a hug because she was crying. She immediately came on to me sexually, and I walked out. She also had men come by in Cadillacs. I told Randy over and over that he needed to break off the relationship, but he wouldn't. This created a bit of problem in our relationship.

24. In my gut, I always believed, and will always believe, that Randy is innocent. I couldn't see Randy hurting a child or not telling what he had done, even when interrogated. The only problem was that he couldn't explain why the child died. Since none of this made sense, I always wanted to know what really happened, and I never felt that anyone had the right answers. .

I swear under penalty of perjury that the foregoing is true and correct to the best of my knowledge and belief.

Dion Liebich

Subscribed to and sworn before me this 19 day of FEB, 2009.

Notary Public in and for the State of Illinois

My commission expires April 3rd 2011

OFFICIAL SEAL
MARLENE FRANCES SZAFRANSKI
NOTARY PUBLIC STATE OF ILLINOIS
MY COMMISSION EXPIRES:04/03/11

DU PAGE COUNTY SHERIFF
FIELD INTERROGATION WAIVER

A. You have a right to remain silent and do not have to say anything at all.

B. Anything you say can and will be used against you in Court.

C. You have a right to talk to a lawyer of your own choice before we ask you any questions, and also to have a lawyer here with you while we ask questions.

D. If you cannot afford to hire a lawyer, and you want one, we will see that you have a lawyer provided to you, before we ask you any questions.

E. If you are willing to give us a statement, you have a right to stop any time you wish.

F. Do you agree to answer our questions here and now?

YES ...X... NO .....

G. ...Randy Leibsle...
Signature

Witness ...[signature]...

Witness ...Raymond V. Bradford...

Time and Date ...11:20A — 2-13-02...

SO-00251

MR 824

JOHN E. ZARUBA
SHERIFF

501 N. COUNTY FARM ROAD
WHEATON, ILLINOIS 60187
ADMINISTRATION (630) 682-7269
CIVIL DIVISION (630) 682-7250

OFFICE OF THE SHERIFF
COUNTY OF DUPAGE

DUPAGE COUNTY SHERIFF'S OFFICE
RIGHTS ADVISEMENT WAIVER FORM

RL 1. You have the right to remain silent.

RL 2. Anything you say can be used against you in a court of law.

RL 3. You have the right to talk to a lawyer and have them present with you during questioning.

RL 4. If you cannot afford to hire a lawyer, one will be appointed to represent you without cost before any questioning.

RL 5. You can exercise these rights at any time.

RL 6. A videotape providing a video and audio record of this interview is being made and I voluntarily consent to this recording.

I understand all of the above rights and voluntarily waive those rights.

Signature: _____

Name: _____
(print)

Witness: _____ **Date:** 3-14-02

Witness: _____ **Time:** 1:00 A.M.

Case Number: _____

M 775

SO-00267-B

Randy Liebich R34940
After 5 Days Return to
P.O. Box 112
Joliet, IL 60434-0112

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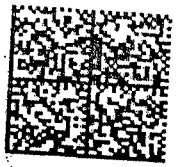
OF THE ILLINOIS
DEPT OF CORRECTIONS



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CHRIS KACHIROUBAS, CIRCUIT CLERK

18th JUDICIAL CIRCUIT COURT - DU PAGE COUNTY

4 / 1 /09

THE PEOPLE OF THE STATE OF ILLINOIS

VS

Randy Liebich # R34940
Defendant

Case No. 02 CF 654

FILED
09 APR -1 PM 4:34
CLERK OF THE 18th JUDICIAL CIRCUIT DU PAGE COUNTY, ILLINOIS

This letter will acknowledge receipt of the following:
Letter / Supplemental documents

which has been filed in the record in the above captioned case on 4/1. Copies of this filing have been forwarded to the following:

- ☐ The Honorable **Ann B. Jorgenson,** Chief Judge of the 18th Judicial Circuit Court
- ☐ The Honorable **Kathryn E. Creswell,** Presiding Judge, Felony Division
- ☐ ~~The Honorable **Michael J. Burke,** Presiding Judge, Misdemeanor and Traffic Divisions~~
- ☒ The Honorable John Kinsella (4000), Trial Judge
- ☒ The Honorable **Joseph E. Birkett,** State's Attorney of DuPage County
- ☐ The Honorable **Lisa Madigan,** Attorney General of the State of Illinois
- ☒ DuPage County Public Defender
- ☐ Sent to the Second District Appellate Court
- ☐ _____

for their further action pursuant to the laws of the State of Illinois or the Supreme Court Rules. You will be advised of the outcome of those proceedings **if required by law or rule of court.**

Sincerely,

Chris Kachiroubas
Circuit Clerk

Prepared by _____

02CF654

Dear Clerk of the Court,

My name is Randy Liebich and I am a Prisoner at The Stateville Correctional Center. My case number is 02-CF-654.

On March 3, 2009 My Petition for Post Conviction Relief was Filed. In support of my petition, I have additional exhibits to supplement thereto.

I therefore supplement my petition with the following documents!

1). Supplement to Page-14 of my original Petition for Post conviction relief (1-Page)

2) Signed Affidavit of Denise Foster (2-Pages)

3) Original signed Affidavit of Dion Liebich (4-Pages)

4) Miranda waivers dated (2-13-02) and (3-1-02) in support of my supplemental Affidavit. (2-Pages)

Respectfully Submitted,

Randy Liebich

Randy Liebich R34940
Stateville Correctional Center
Po Box 112
Joliet, IL 60434

this case become the subject of federal habeas proceedings. I expect to file several expert affidavits or reports, most likely from Professor Patrick Barnes, Professor of Radiology at Stanford University and Director of Pediatric Neuroradiology at Lucile Salter Packard Childrens Hospital; Dr. Peter Stephens, a forensic pathologist; and Dr. George Nicholls, former Medical Examiner for Kentucky.

11. Since I am without any income or assets with which to retain counsel, I ask that counsel be appointed to represent me in this proceeding.

Respectfully submitted,

Randy Liebich

Randy Liebich
I.D. Number 34940
Stateville Correctional Center
P.O. Box 112
Joliet, IL 60434

Subscribed to and sworn to before me this 23rd day of February, 2009.

S K Miles

Notary Public for the State of Illinois

OFFICIAL SEAL
SHERWIN K. MILES
NOTARY PUBLIC, STATE OF ILLINOIS
MY COMMISSION EXPIRES 5-20-2012

I SWEAR THAT THE FACTS STATED IN THIS PETETION ARE TRUE AND CORRECT IN SUBSTANCE AND INFACT.

Randy Liebich

Randy Liebich R34940

STATE OF ILLINOIS)
)
COUNTY OF DUPAGE)

AFFIDAVIT OF DENISE FOSTER

I, Denise Foster, being first duly sworn on oath, depose and say as follows:

1. My name is Denise Foster. My address is 1231 Prairiebrook Drive, Apt. A3, Palatine, IL 60074. I have worked at a daycare center at a health club for twenty years. I am Randy Liebich's sister.

2. I testified at Randy's trial about Kenyatta's treatment of Steven. Kenyatta did not take care of Steven. She paid no attention to him, she hit him regularly, and she did not do the things that a mother would normally do. Since I work in daycare, I know how mothers and children behave, and Kenyatta did not have a mothering or nurturing instinct towards Steven. She expected behavior that was not age appropriate, and she would hit him to make him stop crying. This was all well known at the time of Randy's trial, and I testified to some of it.

3. Before Randy and Kenyatta got an apartment, Randy lived with my mother and Kenyatta would stay with them, sometimes bringing Steven. When I went over to visit one day, Kenyatta and my mother were arguing. I don't remember what they were arguing about, but when I turned my back, I heard a thud. When I turned around, my mother was lying on the floor by the door. When I ran over, she was unconscious. Randy also ran over.

4. I was going to call 911 but my mother came to quickly. My mother said that Kenyatta hit or pushed her, which was obvious, and she asked me not to call 911. I didn't call. From the way my mother had fallen, it looked like she had hit the door.

5. After seeing this, I was very concerned about Randy's relationship with Kenyatta. Randy always wants to see the best in people, but sometimes he is wrong. He does not have a temper, I have never known him to hit anyone, and he generally backs away and stays out of potentially violent situations, as he did with Kenyatta. We have many cousins (11 aunts and uncles on my dad's side, with more on my mother's side), so we had many family gatherings. At family gatherings, children always swarmed around Randy. Steven did the same. Randy is very good with children, but I did not think he would be able to stand up to Kenyatta. Instead, he made excuses for her.

6. The incident with my mother caused me real concern for Steven. We already knew that Kenyatta regularly hit and shoved Steven. My concern was that she would do with Steven as she had done with my mother, that is, push him, or shove him, or slam him into something, with serious consequences. The incident with

my mother, who was not a fighter, showed me that Kenyatta did not have much self-control and that she did not think of the consequences of her actions.

I swear under penalty of perjury that the foregoing is true and correct to the best of my knowledge and belief.

Denise Foster

Subscribed to and sworn before me this 26th day of February 2008.

Guadalupe Munoz
Notary Public in and for the State of Illinois

My commission expires 9/30/2009

"OFFICIAL SEAL"
GUADALUPE MUNOZ
Notary Public, State of Illinois
My Commission Expires 9/30/2009

STATE OF ILLINOIS)
) **AFFIDAVIT OF DION LIEBICH**
COUNTY OF DUPAGE)

I, Dion Liebich, being first duly sworn on oath, depose and say as follows:

1. My name is Dion Liebich. I work for 6-D Aluminum, which is located in Crest Hill, Illinois (phone 708-487-1855). I am Randy Liebich's cousin.

2. In February 2002, I saw a television report saying or implying that my cousin Randy had murdered his girlfriend's son, Steven. I immediately called Randy's sister, Denise, who told me that she heard that Steven had been beaten when Kenyatta was at work and Randy was looking after him.

3. When I heard this, I was extremely angry. This was completely unlike anything I knew of Randy, and I could not believe he had done this. However, since he was the only one home, he had to know what happened.

4. Denise, Randy's sister, told me that Randy was at his mother's, and I drove there immediately and grabbed Randy by the back of his neck. Before we talked about what happened, we decided to go to see our cousin, Robert Liebich, who is a police officer in Rosselle, Illinois. I don't remember whose idea this was but I thought this would let us figure out what was going on. I gave Randy a ride to the Roselle police station. It was a very quiet ride. Since Randy generally did not trust the police and wasn't close to Robert, I thought that Randy was going to confess or at least tell Robert what really happened.

5. Robert took us in an interrogation room. Robert was very angry, and there were a lot of raised voices. There were no Miranda warnings or anything like that. This was a full interrogation, with a lot of anger directed at Randy. Robert and I wanted answers on how Steven died, and Randy wasn't giving us answers. Robert and I had kids, and we were angry with Randy for letting this happen to a child. We thought he had to know what happened since he was there.

6. I let Robert do the questioning since he was a police officer and experienced at doing this. Randy said that Steven choked on a hot dog and that Randy put his finger in Steven's mouth to see if something was stuck in his throat. When he did this, Steven clamped down on the index finger of his right hand. Randy said that when Steven wouldn't let go, he hit Steven on the side of the head to get him to let go. Randy showed us a small cut by his fingernail where he said Steven had bitten him.

7. Robert questioned Randy on how he struck Steven, and Randy illustrated. He showed a tapping from a few inches away that should not have hurt anyone. It

was almost like a push, more pressing than hitting. Robert went over this with Randy several times. Randy's description seemed like a natural instinctive reaction, exactly what I think most people would have done if someone was biting their finger. It did not seem like anything that would have hurt Steven.

8. The atmosphere in the room was very tense and filled with anger, almost hate, since nothing Randy described explained the serious injuries that we heard about. Robert kept telling Randy this but Randy kept telling the same story, over and over again. Robert and I were very angry that Randy wouldn't explain how Steven ended up dead.

9. Finally, Robert asked Randy if he would swear on their fathers' graves that he was telling the truth and the whole truth. Randy was very close to his father, who died when he was a teenager. Robert's father had also died fairly recently. In our family, this type of oath means a lot. Randy said that he swore on their fathers' graves that he was telling the truth.

10. At first, Randy looked at the floor when he said this. I didn't put a lot of importance on this since Randy always looked at the floor when he was upset. When his father died unexpectedly, almost everyone was crying. However, Randy, who was very close to his father and the most upset, sat by himself with his head down. That is how he looked at the police station. Robert, who doesn't know Randy as well as I did, asked the same question again, maybe in different words, and Randy looked Robert straight in the eye and repeated that he swore on their father's graves that he was telling the truth and that he had not hurt Steven.

11. Randy talked about Steven not feeling well the week before he collapsed. He also said that Steven didn't seem that sick after he choked on the hot dog but that after Kenyatta came home they noticed that he was making odd moaning noises and they took him to the hospital. Randy said he took the dog out first. Robert and I didn't pay much attention to any of this since it didn't explain how Steven died.

12. Robert was angry and frustrated because Randy kept saying the same things over and over and wasn't giving any explanation for Steven's condition. Since Robert's father died of a cocaine overdose, Robert despises drugs and looks down on people who use them, including Randy. He was very harsh, therefore, in interrogating Randy. He wanted a confession, and he wasn't getting it.

13. I did not know what to believe. I knew Randy much better than Robert did since I spent a lot of time with him after his father died. Randy could be easily persuaded to do things, but he was never violent and he was really good with kids.

14. Robert kept asking me things like, "what do you think?" I didn't know what to think. Robert said he was trained to know when people were lying and that he knew for a fact that Randy was lying. He seemed to think Randy was lying because he hung his head, but I knew that Randy did this when he was depressed.

15. Robert asked Randy over and over if he could have hit Steven hard enough to hurt him or kill him. Randy always said no. I asked the same questions, and Randy gave the same answers. When Robert asked important questions like this, Randy would look him straight in the eye and say that he didn't do anything that could have hurt Steven and that the only unusual thing that day was when Steven choked ~~on the hotdog. He kept saying that he didn't know what happened to~~ Steven. Robert and I were angry that a three year old had been killed and no one could give an explanation.

16. When Randy was going with Kenyatta, I helped him get an apartment and a job. When I picked him up for work, he talked about problems with Kenyatta. I didn't like the situation with Kenyatta, and I didn't want to hear about the problems.

17. When I visited Randy ~~after Angelique was born~~ DSK, it was over ninety degrees in their apartment, and Kenyatta was frying chicken in the kitchen. It was much too hot for the children, so I went out and bought an air conditioning unit and put it in the window. When Steven went over and started to play with the knobs, Kenyatta grabbed him by the left arm, opened the door to the bedroom and chucked him in, like she was throwing a baseball.

18. I saw this from the kitchen table, and I jumped up and started going after Kenyatta, saying, "how could you do this?" Randy got in the middle, and I yelled at him too, saying, "how can you let this happen?" I told both of them I would call the department of child services if I ever saw or heard about anything like this again. I told my wife about this after I got home.

19. Earlier, Randy told me that Kenyatta would use broken coat hangers to hit Steven. I didn't know why Randy didn't stop it but I don't think he thought he could. Kenyatta was a strong personality, and I don't think she would have listened to him. I was mad with Randy because I felt he was condoning it by staying with her. However, it may have been worse if he left.

20. In all the years I have known Randy, I have never seen him harm or hurt a child. He has been around many children, including my own son, and he never showed a mean streak and rarely showed impatience. When he and Kenyatta lived with us, Randy and Steven got along well, and Randy seemed to like to have him around.

21. When we heard that Steven had been beaten, it occurred to me that if Steven had been beaten, it was more likely to be Kenyatta than Randy. We all knew that Kenyatta hit Steven, but no one had ever seen Randy hit Steven or any other child. I don't think that Robert knew any of this when he interrogated Randy.

22. Months after Steven died, detectives came to my home. They told me that Randy had hit Steven on the head so hard that his brain swelled and there was bleeding on his brain, and that this was the only explanation for his injuries. Since I was

present when Robert interrogated Randy and heard what Randy had to say, I thought that Randy's attorneys would also want to talk to me, but they never did.

23. Randy and Kenyatta lived with my wife and me for awhile, and they had some fights. I never knew who started them or what happened. One time, when my wife and I were home, Randy and Kenyatta had a fight in the bedroom. Randy left the apartment, and Kenyatta came out crying and saying that he hit her. I gave her a hug because she was crying. She immediately came on to me sexually, and I walked out. She also had men come by in Cadillacs. I told Randy over and over that he needed to break off the relationship, but he wouldn't. This created a bit of problem in our relationship.

24. In my gut, I always believed, and will always believe, that Randy is innocent. I couldn't see Randy hurting a child or not telling what he had done, even when interrogated. The only problem was that he couldn't explain why the child died. Since none of this made sense, I always wanted to know what really happened, and I never felt that anyone had the right answers. .

I swear under penalty of perjury that the foregoing is true and correct to the best of my knowledge and belief.

Dion Liebich

Subscribed to and sworn before me this 19 day of FEB, 2009.

Notary Public in and for the State of Illinois

My commission expires April 3rd 2011

OFFICIAL SEAL
MARLENE FRANCES SZAFRANSKI
NOTARY PUBLIC STATE OF ILLINOIS
MY COMMISSION EXPIRES 04/03/11

DU PAGE COUNTY SHERIFF
FIELD INTERROGATION WAIVER

A. You have a right to remain silent and do not have to say anything at all.

B. Anything you say can and will be used against you in Court.

C. You have a right to talk to a lawyer of your own choice before we ask you any questions, and also to have a lawyer here with you while we ask questions.

D. If you cannot afford to hire a lawyer, and you want one, we will see that you have a lawyer provided to you, before we ask you any questions.

E. If you are willing to give us a statement, you have a right to stop any time you wish.

F. Do you agree to answer our questions here and now?

YES ....X..... NO .....

G. ....Randy Leibert.....
Signature

Witness .....

Witness ..Raymond V. Bradford.....

Time and Date 11:20A — 2-13-02

SO-00251

JVR 824

JOHN E. ZARUBA
SHERIFF

501 N. COUNTY FARM ROAD
WHEATON, ILLINOIS 60187
ADMINISTRATION (630) 682-7269
CIVIL DIVISION (630) 682-7250

OFFICE OF THE SHERIFF
COUNTY OF DUPAGE

DUPAGE COUNTY SHERIFF'S OFFICE
RIGHTS ADVISEMENT WAIVER FORM

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RL 5. You can exercise these rights at any time.

RL 6. A videotape providing a video and audio record of this interview is being made and I voluntarily consent to this recording.

I understand all of the above rights and voluntarily waive those rights.

Signature: _____

Name: _____
(print)

Witness: _____ **Date:** 3-14-02

Witness: _____ **Time:** 1:00 A.M.

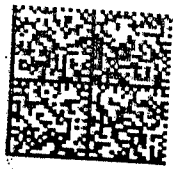
Case Number: _____

MR 775

SO-00267-B

Randy Liebich R34940
After 5 Days Return to
P.O. Box 112
Joliet, IL 60434-0112

THIS CORRESPONDENCE
IS FROM AN INMATE
OF THE ILLINOIS
DEPT OF CORRECTIONS



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STATE OF ILLINOIS)
) SS
COUNTY OF DU PAGE)

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CLERK OF THE CIRCUIT COURT
DU PAGE COUNTY, ILLINOIS

IN THE CIRCUIT COURT OF THE EIGHTEENTH JUDICIAL CIRCUIT
DU PAGE COUNTY, ILLINOIS

PEOPLE OF THE STATE OF ILLINOIS,)
Plaintiff,)
)
-vs-) No. 02 CF 654
)
RANDY LIEBICH,)
Defendant)

AMENDED PETITION FOR POST-CONVICTION RELIEF

NOW COMES the Defendant, RANDY LIEBICH, by his attorney, JEFFREY R. YORK, DuPage County Public Defender, through his assistant, Senior Assistant Public Defender JOAN L. PANTSIOS, and requests that this Honorable Court to grant him relief under 725 ILCS 5/122-1, et seq, and in support thereof, he states as follows:

INTRODUCTION AND PROCEDURAL HISTORY.

1. The Defendant was charged in the above-entitled cause with First Degree Murder, was convicted of that offense on July 16, 2004, following a bench trial, and on September 9, 2004, was sentenced to 65 years in the Illinois Department of Corrections. (See Exhibit 1, attached.)

2. The defendant filed a timely Notice of Appeal. His conviction and sentence were affirmed on December 12, 2007, and a petition for rehearing was denied on January 17, 2008. The Defendant then filed a Petition for Leave to Appeal to the Illinois Supreme Court on February 29, 2008; that petition was denied on May 29, 2008. (See Exhibit 2, attached.)

3. The defendant filed his *pro se* Petition for Post-Conviction Relief on February 23, 2009, which he adopts and incorporates herein. On March 9, 2009, the defendant sent certain exhibits to the Clerk of the Circuit Court for inclusion in his *pro se* Petition for Post-Conviction Relief, which he adopts and incorporates herein. On April 22, 2009, the defendant filed a Motion to file supplemental exhibit, and an affidavit of Dr. Peter J. Stephens, which he adopts and incorporates herein. (See Exhibits 3, 4 and 5, attached.)

FACTS:

1. This case arises from the death of two-year-old Steven Quinn, the son, by a previous relationship, of the Petitioner's girlfriend, Kenyatta Brown. Although Steven did not reside with the couple on a permanent basis, he stayed there from time to time.

2. On February 8, 2002, Petitioner and Brown took Steven to Mt. Sinai Hospital in Chicago. From there, he was transferred to Rush Presbyterian Hospital, where he died on February 11, 2002, after being removed from a ventilator.

3. The prosecution presented expert testimony regarding the cause and manner of Steven's death. The defense presented expert testimony which accepted the prosecution's experts as to injuries to Steven, but disagreed as to the timing of those injuries.

CLAIMS OF ERROR:

A. THE PETITIONER WAS DENIED THE EFFECTIVE ASSISTANCE OF COUNSEL WHERE HIS ATTORNEY FAILED TO INVESTIGATE AND LITIGATE A MOTION TO SUPPRESS STATEMENTS WHERE THE PETITIONER WAS INTERROGATED DESPITE HIS REQUEST FOR COUNSEL AND INVOCATION OF HIS RIGHT TO REMAIN SILENT.

1. The Amended Post-Conviction Petition adopts, as the facts supporting this claim, the matters set forth in the Petitioner's Supplemental Affidavit RE: Interrogation, attached to his *Pro Se* Petition for Post-Conviction Relief.

2. The Petitioner made these facts known to his trial counsel. Despite the fact that the events as presented by the Petitioner supported to a Motion to Suppress Statements, his trial counsel failed to file any such motion.

B. THE PETITIONER WAS DENIED THE EFFECTIVE ASSISTANCE OF COUNSEL WHERE HIS ATTORNEY PREVENTED HIM FROM TESTIFYING AT TRIAL.

1. Following the State's case, the Petitioner informed his attorney that he wanted to testify to correct false information that had been presented to the court. However, trial counsel said that he would not put the Petitioner on the stand. He never told the Petitioner that he had a constitutional right to testify or that this was the Petitioner's decision to make.

2. A defendant in a criminal case has an absolute right to testify in his own defense, under Article I, section 8, of the Constitution of the State of Illinois, and the Fifth, Sixth and Fourteenth Amendments to the Constitution of the United States. It is his own decision, not his lawyer's. The refusal of trial counsel to allow the Petitioner to testify thus violated his constitutional rights and denied him the effective assistance of counsel.

C. THE PETITIONER WAS DENIED THE EFFECTIVE ASSISTANCE OF COUNSEL, WHERE COUSEL FAILED TO INVESTIGATE AND PRESENT AVAILABLE DEFENSES.

1. Counsel failed to present evidence that would have contradicted the testimony of the State's experts regarding the cause of the death of Stephen Quinn, and the timing of onset of the abdominal injuries/infection. (See Exhibit 5, Affidavit of Dr. Peter J. Stephens)

WHEREFORE, Defendant prays this Honorable Court to grant him an evidentiary hearing on his Petition for Post-Conviction Relief, vacate the judgment herein, and set this matter for trial.

Respectfully submitted,

JOAN L. PANTSIOS
Senior Assistant Public Defender
Attorney for Randy Liebich

Joan L. Pantsios
#100077
Office of the Public Defender for DuPage County
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(630) 407-8300

PETITIONER'S EXHIBIT #1:

Sentencing Order, September 9, 2004

CRIMINAL SENTENCE FORM 2152 (Rev. 11/99)

STATE OF ILLINOIS UNITED STATES OF AMERICA COUNTY OF DU PAGE

IN THE CIRCUIT COURT OF THE EIGHTEENTH JUDICIAL CIRCUIT

PEOPLE OF THE STATE OF ILLINOIS

-VS-

RANDY R. LIEBICH

Defendant

CASE NUMBER 02 CF 654

DCN NUMBER -03

- [X] Indictment
- [ ] Information
- [ ] Complaint
- [ ] Resentence

FILED 04 SEP -9 PM 4:34 JOEL A. KAGANN CLERK OF THE 18TH JUDICIAL CIRCUIT DU PAGE CO. ILLINOIS

File Stamp Here

PLEA: [X] NOT GUILTY [ ] GUILTY *FINDING OF GUILTY BY:* [X] COURT [ ] JURY

It is hereby ordered that the defendant is sentenced as follows:

TYPE OF SENTENCE

- [ ] FINED TOTAL AMOUNT $ _____ which includes court costs, penalties and fees as provided by statute.
- [X] FINED $ 0 plus statutory court costs and fees and the following penalties:
  - [X] Criminal Surcharge [ ] Court Sentence Monitoring Program
  - [ ] Driver's Education Fund [X] Violent Crime Victim Assistance
- [ ] COURT SUPERVISION - END DATE _____
- [ ] PROBATION _____ months END DATE _____
- [ ] CONDITIONAL DISCHARGE END DATE _____
- [ ] COUNTY JAIL _____ DAYS TO BEGIN _____
- [ ] PERIODIC IMPRISONMENT
  - [ ] Work Release Program [ ] Weekend [ ] YAWC
- [X] ILLINOIS DEPARTMENT OF CORRECTIONS 65 years w/credit of time served since 3.1.02
  - [ ] Boot Camp
- [ ] CUSTODY OF THE U.S. ATTORNEY GENERAL
- [ ] $100 SECOND OFFENDER DUI FEE (55 ILCS 5/5-1101)
- [ ] $ _____ CANNABIS ADDITIONAL ASSESSMENT (720 ILCS 550/10.3)
- [ ] $ _____ CONTROLLED SUBSTANCE ASSESSMENT (720 ILCS 570/411.2)
- [ ] $100 ADDITIONAL FINE FOR DOMESTIC VIOLENCE (730 ILCS 5/5-9-1.5)
- [ ] $10 ADDITIONAL FINE FOR DOMESTIC BATTERY (730 ILCS 5/5-9-1.6)
- [ ] $100 ADDITIONAL FINE FOR SEXUAL ASSAULT (730 ILCS 5/5-9-1.7)
- [ ] $100 ADDITIONAL FINE FOR TRAUMA CENTER FUNDS EMS ACT (730 ILCS 5/5-9-1.10)
- [ ] $100 ADDITIONAL FINE FOR DUI TECHNOLOGY (625 ILCS 5/11-501(J))
- [X] STATES ATTY ALLOWED 10 DAYS PER DIEM FEE
- [ ] AMENDED CHARGE _____
- [X] NO CREDIT FOR GOOD TIME
- [ ] NO CREDIT FOR TIME SERVED (Credit will be given unless this box is checked)

SENTENCE: FIRST DEGREE MURDER 720 ILCS 5/9-1(a)(1) and 730 ILCS 5/5-5-3 2(b)(4)(i)
UPON A PLEA OF NOT GUILTY COURT FINDS GUILTY AFTER A BENCH TRIAL AND AFTER SENTENCING HEARING: SIXTY-FIVE (65) YEARS IL. Dept. of Corrections w/ credit of Time served from the date of custody 3/1/02
DNA INDEXING + DNA FEE 730 ILCS 5/5-4-3

Disposition of companion cases (not sentenced) _____

State's Attorney MR/JR
Defense Attorney HULMAN/CASEY
Deputy Clerk FRAN
Reporter _____
Bailiff BAILS

JUDGE Doryan
DATE 9/9/04
- [ ] Sentence Stayed Until _____
- [ ] Defendant released from custody _____
- [ ] Appearance on Return Date Required _____

JOEL A. KAGANN, CLERK OF THE 18TH JUDICIAL CIRCUIT COURT © SEP 10 2004
WHEATON, ILLINOIS 60189-0707

ORIGINAL - CIRCUIT COURT CLERK COPIES - STATE'S ATTORNEY - DEFENDANTS ATTORNEY - BAILIFF/PROBATION - DEFENDANT

PETITIONER'S EXHIBIT #2:

Rule 23 Order

No. 2--04--1238

IN THE

APPELLATE COURT OF ILLINOIS

SECOND DISTRICT

THE PEOPLE OF THE STATE OF ILLINOIS,	)	Appeal from the Circuit Court of Du Page County.
Plaintiff-Appellee,	)	4000 S.A.D.
v.	)	No. 02--CF--654
RANDY R. LIEBICH,	)	Honorable Ann B. Jorgensen,
Defendant-Appellant.	)	Judge, Presiding.

Affirmed

RULE 23 ORDER

Defendant, Randy R. Liebich was indicted on four counts of first-degree murder (see 720 ILCS 5/9--1 (West 2002)), one of which was later nolle prossed, arising out of the death of Steven Quinn, who was two years old at the time. Following a bench trial, defendant was convicted and sentenced to 65 years' imprisonment. He now appeals, alleging the following errors: First, he contends that the trial court failed to conduct an adequate inquiry into his pro se claims of ineffective assistance of counsel. Second, he argues that he was not proven guilty beyond a reasonable doubt because the trial court relied on medical evidence that was incompetent, the trial court also made inconsistent findings, and the State failed to satisfy the mental-state element of the offense. Third, he alleges error in the admission of certain opinion testimony of two physicians who treated Steven. Fourth, he asserts that he was denied the effective assistance of counsel. For the reasons that follow, we affirm.

I. BACKGROUND

At the time of the incident that led to the death of Steven Quinn, defendant resided in an apartment in Willowbrook. Also residing in the apartment were Kenyatta Brown, defendant's girlfriend, and Angelique Liebich, the eleven-day-old child of defendant and Kenyatta. From time to time, Steven would also stay at the apartment. Steven was Kenyatta's child from a previous relationship. On February 8, 2004, Steven was left in the care of defendant while his mother was at work. Defendant and Kenyatta took Steven to Mount Sinai Hospital in Chicago later that day because Steven was exhibiting signs of certain medical problems, which we will discuss later. Subsequently, he was transferred to Rush Presbyterian Hospital due to the severity of a head injury he had suffered. Steven was eventually taken off a ventilator, and he died on February 11, 2002. The balance of what follows is taken from the evidence adduced at trial.

The first witness to testify for the State was Karen Clark, Kenyatta's mother. When Kenyatta was 15 years old, Kenyatta gave birth to Steven. He was born at Mount Sinai. Following Steven's birth, he and Kenyatta resided with Karen for about two or three months. Karen never observed Kenyatta exhibit any violence toward Steven. When Steven was about four months old, Karen took over his primary care, as Kenyatta was too young and immature to raise a child. Dorothy Herron, Steven's great aunt, also provided care. Steven would, however, stay with Kenyatta during weekends and sometimes for two weeks at a time. Karen never observed any injuries to Steven when he was returned to her care. On February 8, 2002, Karen was called to Mount Sinai Hospital, where she observed Steven. The child was lying in bed with his clothes off. Karen noted that his right testicle was swollen and red, but did not note any unusual marks on his thighs. Later, after Steven had been moved to Rush Presbyterian Hospital, Karen again observed Steven. She noted marks on his thighs

that were not visible at Mount Sinai and explained "They just started appearing." The marks were "like whip marks, red lines on his thighs, his foot, his ankle, neck, across his stomach and *** on his back and like pressure marks on his neck." During cross examination, Karen acknowledged that she had told a police officer that Kenyatta was an "excellent liar." Further, Karen had never seen defendant hit Steven.

The State next called Sadie Brown. Sadie ran a day-care business out of her home. The center had been in operation since 1996 and is licensed by the Department of Children and Family Services (DCFS). Sadie is Kenyatta's aunt. Sadie provided day care for Steven when he was in Karen Clark's care, as Karen had a job. During the time Sadie watched Steven, he appeared well nourished and she never had any reason to believe he was abused or neglected.

Kenyatta Brown testified that she was Steven's mother. She was 20 years old at the time of the trial. Steven was her child from a prior relationship. She met defendant when she was 16 years old and moved in with him sometime thereafter. Steven would either live with Kenyatta and defendant or stay with her mother or aunt. At the time of Steven's death, they were living in an apartment in Willowbrook. On January 27, 2002, Kenyatta gave birth to a daughter, Angelique. Defendant was the father. Steven stayed with Dorothy Herron while Kenyatta was in the hospital giving birth. Steven returned to the Willowbrook apartment on February 2, 2002. He appeared to be in fine condition. On the way back from picking up Steven, defendant and Kenyatta bought some PCP and stopped at a park to smoke it. The children were present. They then drove to Willowbrook.

At the time of Steven's death, Kenyatta worked for Car-Lene Research. As part of her job, she took surveys at the Yorktown Mall in Lombard. Defendant got Kenyatta the job through his aunt. Sometimes, Kenyatta would bring Steven to work with her; sometimes her aunt or mother

would watch Steven. Steven was never left home with defendant. Kenyatta testified that during the period from February 2, 2002, to February 6, 2002, Steven had a runny nose but did not require medical attention. Further, during that period, she did not discipline or hit Steven.

When at home, Steven would typically follow Kenyatta around the house. Kenyatta referred to him as her shadow. This behavior annoyed defendant. Sometimes, when defendant and Steven were alone in a room together, Kenyatta would hear a "hollow" sound and Steven would cry.

On February 7, 2002, Kenyatta was home with Steven and Angelique. She fed Angelique and prepared pork chops, mashed potatoes, and corn for dinner. Steven ate a few bites of pork and refused to eat anymore, so Kenyatta sent him to his room. Defendant came home at about 8:30 p.m., and Steven came out of the bedroom. Kenyatta testified that she had not struck Steven in any way prior to this time. Defendant had purchased some marijuana before coming home. Defendant sat at the table, rolling the marijuana into a "blunt." Kenyatta noted that defendant's eyes were glazed and his face was "droopy." She believed defendant was under the influence of heroin.

Kenyatta asked Steven if he was ready to finish his food. Steven replied "no," so she sent him back to his room. Steven was crying. Defendant went into Steven's room, and Kenyatta heard a "hollow" sound. When defendant emerged from the bedroom, Kenyatta asked him if he had hit Steven. Defendant stated that he had not. Kenyatta went into the bedroom to talk with and comfort Steven. Shortly thereafter, defendant returned to the bedroom, and he and Kenyatta smoked the "blunt." Steven was crying the whole time. After finishing the "blunt," the two sat on the floor to smoke a cigarette. Defendant said, "Shut the damn kid up." Kenyatta tried to speak to Steven, but he was not paying attention. She "muffed" him to get his attention. Kenyatta explained that to "muff" someone was to shove them with one's fingertips on the side of the head. She told Steven that

she was going to spank him if he did not stop crying. She testified that she was trying to scare him into stopping crying on his own. She asked defendant for his belt. She struck Steven three times on the diaper with the belt, which was folded over at the time. Steven was still crying, so Kenyatta removed Steven's diaper and told him she was going to spank him. She then slapped him on the butt with an open hand. She put the diaper back on and suggested that she and defendant just leave the room, which they did. Steven stopped crying.

Sometime later, Steven came out of the room and stated that he was ready to eat his food. Steven ate about half of his dinner. She let him watch television for a short time and then put him to bed. Steven was able to walk normally at this time, and he slept through the night.

The next morning, Kenyatta woke up at about 9:45 a.m. because she had to go to work. She changed and fed Angelique. At 10 a.m., she entered Steven's room, and he said, "Mom." He appeared fine. Kenyatta got dressed. Before leaving for work, she made a bowl of cereal for Steven. Kenyatta worked until 3:30 p.m, and then she ran some errands.

Kenyatta returned to the apartment at about 4:30. She set down some bags that she was carrying and asked defendant where Steven was. Defendant indicated that Steven was lying on the floor in the living room. Steven was, in fact, covered with a blanket and facing the wall. Kenyatta picked up Angelique, who had wet herself. Defendant started to get ready to go to work. Kenyatta approached Steven and noted that his breathing was not normal. When she rolled him over, she observed that his eyes were "just like a cold stare" and that he had vomit in his mouth. She asked Steven what was wrong, but he did not answer. She asked defendant how long Steven had been in this condition. Defendant said, "About an hour." Kenyatta saw four small red marks, "[a]bout the

size of fingers," on Steven's neck. Kenyatta asked defendant what had happened to Steven, and defendant said that he did not know.

Kenyatta stated that she needed to use the car to take Steven to the hospital and that she might be late picking defendant up from work. Defendant stated he would accompany her to the hospital. Kenyatta told defendant that he needed to go to work, but defendant was insistent. When Kenyatta picked up Steven, she was surprised that defendant had changed his diaper. Kenyatta, defendant, Steven, and Angelique embarked for the hospital. On the way to the hospital, they stopped by defendant's place of employment so defendant could tell his boss that he was not coming in to work. When defendant went inside to do so, he brought Steven with him, so his boss could see how sick Steven was. They then proceeded to Mount Sinai Hospital in Chicago. Mount Sinai was the hospital where Steven was born, and Kenyatta felt familiar with it. During the trip, Kenyatta again asked defendant what happened. Defendant stated that he had fed Steven and that Steven had choked on a hot dog. Defendant stuck his fingers into Steven's mouth. Steven bit defendant's finger. Defendant stated that he was going to hit Steven at this point, but Steven let go of his finger.

When they arrived at the emergency room, medical personnel immediately started to treat Steven. Subsequently, a doctor asked about a lump on Steven's head. There were also bruises on Steven's back. She noted the marks that she had observed earlier on Steven's neck had gotten bigger. Kenyatta asked defendant about the lump on Steven's head, and defendant stated he did not know how Steven got it. When a nurse took off Steven's diaper, Kenyatta noticed that one of his testicles was swollen. Steven was transferred to Rush Presbyterian Hospital for surgery. Additional bruise marks continued to appear after the transfer. Kenyatta spoke with law enforcement personnel from Du Page county during this time. She consented to a search of the apartment.

Kenyatta acknowledged that she would sometimes physically discipline Steven. This would involve striking him on the hand or butt. Steven never sustained an injury or required medical attention as a result of such discipline.

During cross-examination, Kenyatta agreed that Steven spent most of his life staying with relatives. She reiterated that February 8, 2002, was the only time Steven had been left alone with defendant. She acknowledged that there were times in the past when she had hit Steven with her hand on the butt, but denied ever hitting him on the head, striking him in the face, throwing him, picking him up by one arm or shaking him. Kenyatta also clarified that a "muff" was really just a touch with no real force behind it. Kenyatta acknowledged having pleaded guilty to a charge of prostitution in Winnebago County. She agreed that she had never seen defendant strike Steven.

John Georgopolous, a manager at the Patio Restaurant in Darien, also testified for the State. Georgopolous was working during the evening of February 8, 2002. Defendant had been working at the Patio for about two weeks at the time. Shortly after 5 p.m., defendant entered the restaurant holding a child. Defendant explained that his girlfriend's child was sick, and he had to take it to the hospital. Georgopolous stated that that was fine. The child appeared sick to Georgopolous, but he did not observe any marks on the child. Nicolas Brinias, another manager, later testified that on February 5, 2002, defendant had to leave work early because of a sick child. Also, on February 6, 2002, defendant missed work without explanation.

The State next called Dr. Paula Green. Green testified that she is a physician who specializes in emergency medicine. She was working in the emergency room at Mount Sinai on the day that Steven was brought there. A nurse asked Green to examine Steven. Green initially thought Steven was experiencing a febrile seizure, which can result from a high fever. Defendant and Kenyatta were

present. Green had a conversation with defendant regarding what had happened to Steven. Defendant stated that Steven had choked on a hot dog and had been in the state in which they brought him to the emergency room ever since. Defendant said that he did not pat Steven on the back when he choked and that Steven had not fallen or bumped his head. Green stated that during her first examination of Steven, she did not note any signs of trauma. A short time later, Green performed what she termed a "secondary survey." At this point, she noticed some marks. This indicated that Steven's condition was acute, that is, "something [that] has just occurred." Green called Kenyatta over and asked her if she had ever seen a mark that was on Steven's head before. Kenyatta turned to defendant and said, "[W]hat did you do to my baby." Steven was gazing to the left, which is indicative of a head injury, and he had a bruise on his lip. There was a red bruise on Steven's abdomen. The color signified an injury occurring within hours. There were also red marks on Steven's buttocks and back. Green examined a photograph of Steven that was taken after Steven left Mount Sinai. It showed bruising, particularly lateral marks on Steven's legs and feet, that had appeared subsequent to the time Green examined him. Green also noted "posturing" in Steven. "Posturing" is an involuntary flexion of the extremities and is a sign of severe brain injury. Dr. Boykin began assisting Green. A CAT scan revealed a "bleed," so a neurosurgeon, Dr. Munoz, was called. Munoz was working at Rush Presbyterian Hospital that night, so Steven was transported there.

During cross-examination, Green agreed that a mark on Steven's back could have been caused by a belt buckle. Similarly, the marks on Steven's buttocks could have been caused by someone striking him forcefully in that area. Also, the trauma team, which was called in after the results of the CAT scan were known, did note abdominal bruising while Steven was still at Mount Sinai. Green further agreed that lethargy, slurred speech, irritability, nausea, lack of appetite, and finicky eating

could be symptoms of a head injury (though during redirect, she stated that she had never seen a person with a head injury of the severity of Steven's walking, talking, eating, or playing). Further, a person who has suffered a head injury can remain conscious. Green found defendant's hot-dog story incredulous. She did, however, acknowledge that a layperson could confuse a choking incident with a seizure. Green testified that at the time Kenyatta asked defendant what he had done to Steven, Green had not yet indicated to Kenyatta that she suspected child abuse was involved. Green described defendant's demeanor as calm and respectful during the time he was at the emergency room, despite his being interrogated by doctors and nurses.

Dr. Tracy Boykin, the other emergency room physician who treated Steven at Mount Sinai, testified next for the State. Boykin stated that Mount Sinai was a level one trauma center, the "highest definition of a trauma center." She is board certified in emergency medicine. On the evening of February 8, 2002, Boykin was working at the Mount Sinai emergency room. She observed defendant carrying Steven into the hospital. Steven was limp. Boykin assumed Steven had had a febrile seizure. About three to five minutes later, someone came and told Boykin that Green needed her assistance. She went to Steven's bedside and immediately observed that he was posturing. Boykin was called to the CAT scan machine in the radiology department. The person that summoned her stated that the results of the CAT scan were really bad and that Steven's head was full of blood. Boykin then ran back to the emergency department and informed Green of Steven's condition.

Boykin then went to find Kenyatta and defendant. Boykin stated that she was angry at this time. She explained that she was angry because "Stevie had obviously suffered a severe brain injury secondary to trauma," and that "[i]t wasn't secondary to the hot dog that they said he choked on earlier." Defendant was "very calm, nonchalant," which further angered Boykin. Boykin told

defendant, "[I]t doesn't look like Stevie choked on a hot dog at all, it looks like [you] had been sitting at home beating him all day." Defendant did not say anything and just shrugged. Boykin told defendant that she was going to contact the police and DCFS. At this point, Steven was brought back to the emergency department, where he was intubated. The Chicago police arrived at about this time.

During cross-examination, Boykin testified that Steven could not have possibly eaten a hot dog after sustaining the injuries that she witnessed. When asked by defense counsel whether she would have wanted to know of events transpiring the night before Steven came into the emergency room, Boykin replied no because, had he sustained the injuries at that time, Steven would have been dead by the evening that she treated him.

The next witness called by the State was Marlene Szafranski. Szafranski was an office manager at Car-Lene Research, where Kenyatta worked. She is defendant's aunt. Kenyatta also worked for Car-Lene research. Szafranski testified that Kenyatta would sometimes bring Steven to work. On February 8, 2002, Kenyatta arrived to work at 10:40 a.m and worked until 3:40 p.m. She did not bring Steven with her on that day.

Letitia Beasley, was working as a nurse in the emergency room at the time Steven was brought in. She testified that she evaluated Steven when he first arrived there. Beasley noted that Steven was not breathing normally and that he was posturing. His eyes deviated to the left, and there was a bruise on the right side of his head. She rated Steven a four on the Glasgow Coma Scale. The Glasgow Coma Scale is a scale for recording a persons conscious state, which ranges from 3 (most likely dead) to 15 (normal and healthy). Steven's temperature was 94.7 degrees. There were bruises to Steven's scrotum. However, linear marks that were photographed after Steven had been moved

to Rush Presbyterian Hospital were not visible at the time Beasley performed her examination. Beasley observed Kenyatta speaking to defendant. Kenyatta was tearful and wanted to call her mother. Defendant told her not to do so. Defendant had a "very flat affect" and was not tearful.

On cross-examination, Beasley testified that defendant's demeanor was not paranoid, nervous, or fidgety. She also testified that she administered Ativan to Steven. Ativan is a medication used to treat seizures and to "break out *** [of] a dystonic reaction, a tightened, rigid reaction."

The next witness called by the State was Sergeant Michael Price. Price was involved in the investigation into the death of Steven beginning in the early morning of February 9, 2002. Price was dispatched to Rush Presbyterian to photograph Steven. He arrived about 2 a.m. After photographing Steven, Price went to defendant's apartment and took several photographs of it as well.

Lieutenant Edward Kunz of the Du Page County sheriff's office then testified that he arrived at Rush Presbyterian Hospital between 11:30 p.m. and midnight on February 8, 2002. Defendant was pacing a lot. At one point, defendant stated that he wanted a cigarette and to see his daughter. Kenyatta gave defendant a cigarette, and Kunz accompanied defendant outside. There, the two had a conversation. Kunz told defendant that he had a son about the same age as Steven. Kunz asked defendant if Steven's crying was aggravating to him. Defendant replied, "[Y]es, very." Kunz asked how defendant dealt with Steven's crying. Defendant stated that he normally does not have to deal with it, since Kenyatta does.

Defendant was granted permission to call Ruben Martinez out of order. Martinez testified that he had known defendant for about seven years. Martinez had witnessed Kenyatta strike Steven on one occasion. According to Martinez, Kenyatta struck Steven one time, back-handed, in the head. Martinez thought that this incident occurred at Kenyatta's aunt's house, but Martinez could not say

when. Cross-examination revealed that Martinez and defendant were good friends and that Martinez had visited defendant several times while defendant was in jail.

The State next called Tammy Smith. Smith is a pediatric and neonatal intensive care nurse who works at Rush Presbyterian. She was working on the evening of February 8, 2002, and was involved in Steven's treatment. She traveled to Mount Sinai by ambulance to transport Steven to Rush Presbyterian. A physician also accompanied her. Smith performed an initial evaluation of Steven. He was unresponsive, his hands and feet were cold, and his body temperature was low. There was a fresh bruise on the side of Steven's head, and there were also red bruises on his abdomen. Smith identified a bruise in a photograph of Steven that was not visible when she first examined him at Mount Sinai. They left Mount Sinai at 8:50 p.m and arrived at Rush Presbyterian at approximately 9 p.m. Steven went into surgery at about 10 p.m. and was in surgery for one and one-half hours.

Smith remained with Steven until 7:30 a.m. She was present when a police officer came and photographed Steven, which was approximately two hours after surgery. Smith noted additional marks that were present in the photographs that were not present when she examined Steven at Mount Sinai, and she told a physician of their existence because they were not there earlier. Also, bruises she had seen at Mount Sinai on Steven's thigh had become more defined in the interim. Similarly, marks on Steven's back had become more defined, and bruises on his abdomen were more pronounced. A mark under Steven's jaw, which Smith did not notice at Mount Sinai, had also appeared by this time. His abdomen became distended. Throughout the night, Steven's bruises darkened in color and were generally more pronounced. On cross-examination, Smith acknowledged that, other than two marks appearing on Steven's foot, she did not make any entries in Steven's chart

regarding bruises changing throughout the night. She explained that she was extremely busy during the latter portion of her shift.

Greg Figiel, who was previously employed with the Du Page County sheriff's office, testified next. Figiel was involved in the investigation of Steven's death. Figiel and Boris Vrbos of the Du Page County Children's Advocacy Center traveled to Rush Presbyterian at about 10:40 p.m. Figiel and Vrbos had a conversation with defendant at 11:20 p.m. in a conference room at the hospital. Tammy Smith brought defendant to the room. Defendant stated that he was watching Steven while Kenyatta was at work. Steven came to the kitchen table that morning and ate his cereal but did not drink his milk. Steven then played with the dog. Defendant took a nap in the afternoon. At about 3 p.m. he got up and made a hot dog for Steven. Defendant stated that Steven ate one-half of the hot dog. Steven also had a glass of water and, as he was drinking from the glass, he started to choke. He then vomited. Steven was breathing funny and wheezing, so, defendant said, he patted Steven on the back. Defendant asked Steven if he was all right. Steven nodded and said, "[Y]eah." Steven then laid down in the living room, moaned a few times, and fell asleep. According to defendant, Kenyatta arrived home at 4:30. She checked Steven and determined that he was not breathing properly, so they decided to take Steven to the hospital. Defendant also told Figiel that Steven was playing that day, but not as hard as he usually did.

Figiel and Vrbos went over defendant's story with defendant. Figiel asked what happened after Steven vomited. Defendant stated that he picked Steven up, and Steven went limp in his arms. Figiel asked what caused Steven to go limp and whether Steven had fallen or hit his head. Defendant answered that Steven had not fallen that day. They went over the story a third time. This time, defendant stated that everyone woke up at 10 a.m. He also stated that he had cut up the hot dog that

he later fed to Steven, that Steven had a glass of orange juice in addition to water, and he did not mention anything about Steven vomiting. This interview terminated at about 12:15 a.m. Figiel then spoke with Smith and was updated about Steven's condition.

Figiel met with defendant a second time at 1:05 a.m. This meeting took place in the same conference room as the previous interview, and defendant, Figiel and Vrbos were present. Figiel testified that he gave defendant Miranda warnings. At this point, someone knocked on the door and said that they had food for defendant. Defendant was given a taco salad, and the officers left him alone for about 10 minutes to eat it. They went over defendant's story again. Figiel told defendant that he did not believe that nothing happened in the apartment that day, given the nature of Steven's injuries. Defendant then told Figiel that after he had patted Steven on the back, Steven had fallen head-first to the floor. Defendant said that it was not a hard fall, however. Figiel stated that this is the first time defendant mentioned a fall.

At about 7 a.m., according to Figiel, he, defendant and Vrbos left the hospital in Figiel's unmarked car. Defendant voluntarily accompanied them. They stopped at McDonald's to get defendant orange juice and a potato cake and then at Burger King, because defendant also wanted a chicken sandwich. They then proceeded to the sheriff's office, arriving at 8 a.m. They placed defendant in an interview room. At about 8:40, defendant vomited in a waste paper basket. Figiel entered the room and asked defendant if he was all right. Defendant stated that he had a $20-a-day heroin habit.

Figiel went to Kenyatta's mother's house on February 19, 2002. The purpose of the visit was to listen in on a telephone conversation between Kenyatta and defendant. Prior to this time, Figiel had never mentioned to defendant that a clothes hanger may have been used as a weapon against

Steven. During the conversation, defendant told Kenyatta that she had hit Steven on the leg with a clothes hangar on February 7. She replied that she did not and that was the occasion on which she asked defendant for a belt to scare Steven. Defendant then stated, "Well, you came out of the closet with a hanger in your hand." This was the first time Figiel heard defendant say anything about a clothes hanger. Defendant called Kenyatta a second time that same day. Defendant asked if anyone was listening in on the call, which only lasted about 10 seconds. Defendant then called a third time a few minutes later. He told Kenyatta that he suspected that someone was listening in on their conversation, so Kenyatta should stay on the line and call him with a different phone at a different number. Kenyatta declined, and the two continued to speak. In all, defendant called Kenyatta nine times that day.

On cross-examination, Figiel agreed that, as he and defendant repeatedly went over the defendant's story, the substance of the story remained, for the most part, consistent. Figiel also acknowledged that defendant never told him that he struck Steven in any way. The court also permitted the State to elicit rebuttal testimony, out of order, from Figiel. The rebuttal testimony concerned the testimony of Martinez. Figiel testified that Martinez told him that Martinez had had a conversation with defendant wherein defendant stated that Kenyatta had hit Steven with a clothes hanger and that defendant had removed the hanger from the apartment as he knew police would be searching the apartment.

Thomas Filipiak, a Chicago police officer, testified that he was dispatched to Mount Sinai Hospital with regard to an injured child on February 8, 2002. Child abuse was suspected. He arrived at the emergency room around 6:30 p.m. Filipiak spoke with defendant. Defendant stated that Steven had choked on a hot dog and that he had slapped Steven in the back in an attempt to dislodge

it. Defendant denied striking Steven in the head. Defendant also replied "no" when asked if there was anyone else who could have harmed Steven. Filipiak described defendant's demeanor as aloof and nonchalant. As the evening progressed and it became clear that Steven's injuries were serious, defendant "became very very [sic] scared." Responding to questioning by defense counsel, Filipiak acknowledged that he did not know how much information defendant possessed regarding Steven's condition at the time he appeared nonchalant and aloof.

The State next called Dr. Paul Severin. Severin is a physician who is board certified in pediatrics and pediatric critical care, and the court recognized him as an expert in those areas. Severin provided treatment to Steven after Steven was transferred to Rush Presbyterian. Severin noted that Steven's Glasgow Coma Scale score was quite low and that Steven was posturing. Due to the interventions provided by the doctors at Mount Sinai, Steven's Glasgow Coma Scale score did improve. Severin explained that one of these procedures was to increase Steven's rate of breathing to relieve pressure in his head. Increasing breathing causes more oxygen and less carbon dioxide to be present in the blood. This, in turn, causes an organ, such as the brain, to shrink its blood vessels which, in turn, decreases blood flow and pressure in the organ. Severin further explained that such "auto-regulation" is not possible where the brain has been injured for a prolonged period of time. Thus, according to Severin, that Steven's brain was able to adjust indicated a more recent injury.

Severin testified that he observed numerous bruises and marks on Steven's back, scrotum, and thigh. The injuries appeared to be of approximately the same age. Steven's right pupil was larger than his left one, indicating that his brain was about to herniate. Severin later learned that the pressure in Steven's head measured 90 millimeters of mercury. The normal range is between 15 and 20 millimeters of mercury. Steven's abdomen was soft and there were bowel sounds which indicated

that no abdominal injury was likely. Due to elevated enzyme levels, Severin ordered additional testing. Steven's pancreatic enzyme level was in the thousands, while the normal level for a child of his age is 200.

Severin continued to provide care to Steven on February 9, 2002. He examined Steven that morning. Steven's abdomen appeared distended and he had "defuse redness around his belly button." That, coupled with elevated pancreatic enzyme, constitutes a "Cullen's sign." A "Cullen's sign" is an indication of hemorrhagic pancreatitis. At this point, Severin could no longer detect bowel sounds. Severin testified that a child that had sustained the type of injuries Steven had received would not be able to eat. At best, the child would throw up anything he ate. Severin opined that Steven's injuries resulted from nonaccidental, external trauma. Additionally, Severin stated that they occurred within four to six hours prior to Steven's admission to Mount Sinai. Further, Severin opined that Steven's injuries could not have been caused the night before, as there was "no way anybody would be able to survive a whole day later to get to the hospital." Additionally, choking on a hot dog was not consistent with Steven's condition.

Defense counsel then cross examined Severin. Severin acknowledged that he is not typically called upon to determine the timing of an injury. Further, Severin agreed that the history received from the family is a "critical factor" in determining the timing of an injury. The bruises Severin observed on February 9 could have occurred within the last 48 hours; however, bruising is difficult to time. Also, even if Steven had suffered no head injury, the injuries to his abdomen were potentially fatal. Severin further stated that the discovery by the pathologist, Dr. Mileusnic, of signs of healing would not change his opinion regarding the timing of the injuries due to his findings during his physical examination of Steven and the acuteness of Steven's injuries.

Janet David then testified that defendant came to the McDonald's where she worked between 2:30 p.m. and 3:30 p.m. on February 8, 2002, and "bummed" a cigarette from her. The McDonald's was across the street from defendant's apartment, and he only stayed a few minutes. The State then called Thomas Szalinski.

Szalinski testified that he had been a law enforcement officer for 19 1/2 years. He spoke with defendant in an interview room at the Du Page County sheriff's office. Defendant told Szalinski that, on the night of February 7, he and Kenyatta were in Steven's bedroom and Kenyatta disciplined Steven because he was crying. Defendant stated that Steven was wearing a diaper and Kenyatta struck him a few times on the buttocks with a belt. Kenyatta did not strike Steven on any other part of his body. Defendant stated that, in his opinion, Kenyatta was not hitting Steven hard enough, as the discipline was not having its intended effect. Defendant stated that he did not think Steven could feel it. Kenyatta then pulled the diaper down and struck Steven with her bare hand. At about midnight, defendant told Szalinski, Steven was crying and he and Kenyatta went into Steven's room. Kenyatta "popped" Steven in the head a few times. By "pop," defendant meant a sort of slap with the palm of the hand. Defendant denied striking Steven.

Defendant related that the next morning he and Kenyatta woke about 9:30 a.m. Neither disciplined Steven. Defendant fed Steven breakfast (cereal, juice, and water) after Kenyatta left for work at 10 a.m. Steven ate all of his cereal and drank his orange juice, but did not touch his water. Defendant then took Steven out of his high chair and placed him on the floor. Steven later played with the dog under the kitchen table. Szalinski asked if Steven could have been injured while playing with the dog, and defendant stated that he did not think that that was possible. Defendant changed Steven's diaper, and he did not observe any injuries while doing so. Later, defendant, Steven, and

Angelique watched the television together. Steven appeared dazed. Defendant stepped out to get a cigarette at about 1 p.m. while the children were asleep. Szalinski asked if anyone could have been in the apartment while defendant was gone. Defendant stated that he doubted so because he was only gone five minutes and the children were still sleeping when he returned. Szalinski testified that defendant told him that at about 3 p.m., he gave Steven a hot dog and some water. Steven choked on the water. Detective Raymond Bradford, who the State called next, then entered the room and took over the interview.

Bradford testified that he was an investigator for the Du Page State's Attorney's office. Bradford told defendant that he had just received information from the hospital that Steven likely would not survive his injuries. Bradford also stated that they were sure that Steven's injuries occurred while Steven had been in his care and that defendant was responsible for them. Defendant stated that he had not hurt Steven. Defendant was crying. Bradford stated that he understood that things like this could happen by accident and that defendant did not mean to hurt Steven. Defendant, resting his head in his hands, appeared to be listening very intently and nodded in agreement. Defendant then asked to use the telephone and to see Kenyatta. He was allowed to do so.

The State then called Robert Liebich. Robert is a police officer with the Roselle police department and is the cousin of defendant. At about 4:30 p.m. on February 14, he was on patrol and he received a dispatch that he had a family member waiting for him in the lobby of the police department. When he arrived there, he saw defendant and another cousin named Dion. The three went into a private room. Defendant asked if it was safe to talk in the room, meaning was the room "bugged." Robert said that it was not. Robert asked defendant about a LEADS message that had been sent out throughout the State that defendant was missing and suicidal. Defendant stated that

he had not been running and that he had in fact been speaking with the detectives investigating the case. Defendant showed Robert a small cut on the right index finger and told Robert that Steven had bitten him and drawn blood. Robert said, "So what?" Defendant explained that he was worried about his DNA being inside Steven's stomach. Defendant also told Robert that he saw Kenyatta hit Steven in the head four times because Steven would not stay in his room. The next morning Steven was lying in his bed awake, which is unusual because Steven usually gets right up. Later, when defendant was feeding Steven a hot dog, Steven started to choke. Defendant hit Steven on the back in an attempt to dislodge the food. When that did not work, defendant inserted his finger into Steven's mouth. It was at this point that Steven bit defendant. Defendant "smacked" Steven twice on the right side of his head so that he would let go. As defendant removed his fingers from Steven's mouth, Steven vomited. Steven was "kind of dizzy, wasn't walking right." Defendant asked Robert what Robert thought he should do. Robert advised him to tell the truth. During cross-examination, Robert acknowledged that he asked defendant if he would "swear on his father's grave" and then defendant looked him right in the eye and stated that he did not hit Steven that hard.

Dr. Darinka Mileusnic Polchan, a forensic pathologist, testified next for the State. Mileusnic explained that there was a difference of opinion in her field as to whether children sometimes experience a lucid interval following a traumatic head injury prior to becoming symptomatic. Studies that extrapolate from car accidents are inherently flawed, as the mechanism of injury is quite different from child abuse. Some studies purporting to demonstrate that no lucid interval can occur suffer from this or similar flaws. She has twice been hired to testify for defendants in criminal cases. Mileusnic performed a postmortem examination of Steven on February 12, 2002.

Her external examination revealed that Steven appeared to be a well-developed, well-nourished child. She noted a healing contusion on the right side of Steven's forehead. Dating the injury based on appearance was not possible. There was also a contusion on the nose and two on the left side of the head as well as one on the inner aspect of the ear. A healing abrasion was present on the back of Steven's head. A sixth injury--a bruise--existed on the right temple, and there was a seventh near the right ear. There were also bruises under Steven's chin. Mileusnic noted a superficial abrasion on Steven's neck that appeared to have been caused by his medical treatment, since it was much fresher than his other injuries. His right wrist was bruised. Mileusnic counted five bruises upon Steven's lower back. A photograph taken at Rush Presbyterian showed a cluster of bruises on Steven's back that were no longer present at the time of the autopsy. In fact, Mileusnic testified that "many of the injuries already healed and was [sic] obscured by the time that [she] saw Steven on the 12th." His scrotum was swollen.

Mileusnic documented three injuries to the right leg. One was a cluster of ten bruises, the second was a cluster of two larger bruises, and the third was another cluster of smaller bruises. She also found four injuries to the left leg. On the thigh was a cluster of four bruises, which Mileusnic listed as the 14th injury she found. Also, she found a bruise on the inner side of the left ankle, seven linear bruises on the foot, and a bruise on the top of the foot.

Mileusnic was shown a blue plastic clothes hanger and asked whether being whipped by it could have made the linear bruises found upon Steven (such as were found on his back, thigh and foot). She was of the opinion that it could. Further, the injuries would not be consistent with being struck with a belt. She did, however, note that there was one mark on Steven's buttock which could have been made by a belt. Outside of some preexisting marks on Steven's back and the abrasion

caused by medical treatment, the rest of the injuries appear to have occurred at approximately the same time.

Mileusnic also performed an internal examination of Steven. She noted a three inch hemorrhage into the soft tissue under the skin of the head. This is indicative of some sort of blunt force trauma. She also noted some residual subdural blood in the cranial cavity. On the left side of the head, which was not touched by the surgeon, she found a significant subdural hemorrhage. Additionally, a subarachnoid hemorrhage enveloped the brain (this comprised a thin layer). The right cerebral hemisphere showed a contusion, hemorrhage, and necrosis;[1] however, she stated she could not comment on the relevance of these findings due to the surgery performed on the right side of Steven's brain. She found blood descending "passively along the dura[2] downwards," which is consistent with blunt force trauma to the head and a severe brain injury. An examination of the eyes revealed an accumulation of blood, which is another sign of severe head trauma.

An internal examination of Steven's body cavity revealed peritonitis, a form of inflamation. Fibrin deposits, which are a sign of healing, were present over the proximal bowel or jejunum. The first segment of the jejunum was perforated and appeared hemorrhagic and necrotic. There was blood in the bowel, which was in the process of dying. There was a hemorrhage around the head of the pancreas. Mileusnic opined that these injuries were also the result of blunt force trauma. Also,

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[1] "Necrosis" is defined as the "[p]athological death of one or more cells, or of a portion of tissue or organ, resulting from irreversible damage." Stedman's Medical Dictionary 1185 (27th ed. 2000).

[2] "Dura," short for "dura mater," refers to "a tough, fibrous membrane forming the outer covering of the cental nervous system." Stedman's Medical Dictionary 548 (27th ed. 2000).

"there was a collection of blood under the capsul [sic] of the liver that corresponded to the same level of injury." The retroperitoneum (which is the soft tissue behind the bowel that "sort of interfaces between the abdomen and the back") had blood tracking down it. In fact, what had been perceived as bruising to Steven's testicle was actually from blood tracking all the way down to his scrotum from this area. Mileusnic examined the testes and found no blunt injury to them during the autopsy; however, she later stated that a photograph of Steven nearer to the time of his admission showed "swelling, redness and a linear abrasion on the scrotum." Furthermore, that there was hemorrhaging on both the right and left side of Steven's body indicated blunt force trauma to both sides. Typically, the bowel is "pink/tan and glistening." Steven's bowel was dark red in areas indicating hemorrhaging, and yellow, which shows necrosis.

In cases of suspected child abuse, the usual practice is to make incisions through the skin of the upper and lower extremities to look for bruises that are deep and not visible superficially. Such an examination revealed three areas of hemorrhaging in the arms and three in the legs and feet. Mileusnic also found evidence of hemorrhaging in the back and buttocks through a similar examination. These injuries are also indicative of blunt force trauma. Mileusnic agreed that injuries like these would not be caused by "normal corporal punishment," but would require "something much more forceful." Again, Mileusnic was of the opinion that all of these injuries occurred at about the same time and that none were related to medical treatment Steven received. Moreover, these injuries were consistent with child abuse and with Steven being beaten to death. Finally, Mileusnic opined that Steven's death was caused by blunt force trauma.

On cross-examination, defense counsel asked Mileusnic about an article upon which she was working. One of the premises of the article concerned lucid intervals in cases of child abuse. Head

trauma does not have to manifest immediately. In the case of child abuse, a child may not become significantly symptomatic for up to 48 hours after the injury. The doctor also authored another case study where a lucid interval occurred where a child died a "couple of days" following head trauma. Mileusnic also explained that there was a difference of opinion in her field regarding the existence of lucid intervals. One school holds that such intervals never occur; the other, less well defined, questions the first school's tenet. The first school normally holds that the last person with a child when symptoms of a head injury manifest is the perpetrator of abuse. Mileusnic finds this proposition problematic and believes that knowing a history from a nonmedical source is important. She expressly was not giving an opinion regarding whether Steven experienced a lucid interval in this case.

Mileusnic agreed that timing is an issue she frequently confronts as a forensic pathologist. Generally, she gives times in terms of days and would never give an interval for the timing of an injury such as four to six hours. Moreover, giving an interval requires looking at microscopic evidence.

Mileusnic did perform a microscopic examination during her autopsy of Steven. Establishing when the abdominal injuries occurred was problematic due to the degree of necrosis. However, she testified that she was "more comfortable" with regard to the head injury as that area of the body is "kind of sequestered" and "not exposed to a lot of decay." Mileusnic opined that Steven's head injuries were inflicted approximately five days, plus or minus a day, from the time of his death. She later clarified that the day of Steven's death would count as a day, so, February 7, plus or minus a day, was, in her opinion, when the injuries occurred. Mileusnic believed the injuries "could have occurred on the 8th, but they could have easily occurred before that," even as early as the 5th. She further explained that abdominal injuries often were slow to manifest. The symptoms are not instantaneous

and, though there would be pain, a two year old would likely have difficulty expressing what he was going through. Necrosis, bleeding, and pancreatitis would follow "hours after the injury." Mileusnic saw signs of healing in Steven. One would usually see a fibrin layer[3] within the first day. However, the peritoneum ("a thin layer of irregular connective tissue[] that lines the abdominal cavity" Stedman's Medical Dictionary 1353 (27th ed. 2000)) responds to an injury differently. In Steven's head, Mileusnic found mononuclear cell fibrin, which is a "third kind of level of defense that happens in the body." Typically, these occur five to seven days after an injury.

Mileusnic agreed that finicky eating, lack of appetite, inconsolable crying for no apparent reason, lethargy, and excessive sleeping could all be symptoms of the injuries Steven sustained. Head injuries could result in seizures, and clenching one's jaw is a sign of a seizure. A seizure could be mistaken for choking. However, during redirect, she explained that it would be difficult for a person to force a finger between the teeth of a child clenching his or her jaw during a seizure. Furthermore, such a child would be experiencing severe pain and would be vomiting within hours. She would not expect a child with these injuries to eat breakfast if he had sustained the injuries the night before.

The final witness to testify for the State was Dr. Lorenzo Munoz. Munoz is board certified in neurosurgery. Munoz was on duty on February 8, 2002, when Steven was transferred to Rush Presbyterian from Mount Sinai. Munoz was informed that Steven was "neurologically very sick", that he had required intubation, and that a CT scan[4] had revealed a lot of blood in his head. Munoz first

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[3] Wikipedia defines "fibrin" as "protein involved in the clotting of blood. It is a fibrillar protein that is polymerised to form a 'mesh' that forms a hemostatic plug or clot (in conjunction with platelets) over a wound site." See http://en.wikipedia.org/wiki/Fibrin.

[4] Munoz explained that " 'CT' stands for computerized tomogram."

saw Steven at around 8 p.m. or 9 p.m. He conducted a gross visual examination of Steven's head and observed bruises. Steven was nonresponsive but not medically paralyzed. Munoz stated that it was obvious that Steven had not been given medication to relax his muscles because he was posturing. Posturing, according to Munoz, "is a very ominous sign that there's something very bad going on with the central nervous system." The CT scan revealed what Munoz believed was a fresh subdural hematoma. It also showed a "rather diffused and very severe subarachnoid hematoma." "Diffused" means not localized, which is significant because it "speaks of a trauma to the whole brain." Indeed, there were other indications of generalized swelling and injury to the entire brain. Munoz stated that Steven's brain was "extremely swollen."

After examining Steven, Munoz decided that surgery was the best course. Munoz testified that because Steven's pupils were reactive, he felt he had to "give this kid a chance." The goal was to decompress a blood clot and relieve the pressure on Steven's brain. The operation commenced at approximately 10 p.m. Munoz opened Steven's skull. The dura (the outermost layer covering the brain) was taught. Upon opening the dura, Munoz observed a subdural hematoma. Steven's brain was "red with a massive amount of subarachnoid blood" and swollen. The brain started to herniate, that is, to come out of the opening in the skull that Munoz had made. Munoz stated that herniation is also an ominous sign "because that tells you the brain is so swollen *** that it's trying to find a way out to decompress itself." He continued, "The problem with that is that as the brain starts coming out there is not much you can do about it at that point anymore." The blood Munoz observed in Steven's head was bright red, as opposed to "currant, jelly color" or "motor oil color." This coloration indicated that the blood constituted a fresh clot. Munoz characterized the amount of blood

as "massive." In Munoz's experience, "the longer removed the injury that brings about the bleeding in your brain is, the darker the blood looks."

Munoz testified that at this point, there was not much he could do except evacuate the subdural blood and close up Steven's skull. Subarachnoid blood is too intimately associated with the brain to be removed. Munoz also installed a "intracranial pressure monitor bolt," which is a device that measures pressure inside the skull. The device gave a reading of over 90, which is incompatible with life. After surgery, Munoz again evaluated Steven. His eyes were fixed and were no longer reactive to light (Munoz later explained that had Steven's eyes been fixed prior to surgery, Munoz likely would have regarded surgery as futile). Subsequently, Steven was declared brain dead by the pediatric staff of the intensive care unit. He was then taken off of his ventilator and died.

In the course of treating Steven, Munoz was provided a history that had been taken at Mount Sinai regarding Steven choking on a hot dog. Munoz testified that that history was "impossible." He also opined that Steven's injuries were not accidental and that they occurred no more than six hours prior to the time Steven arrived at Mount Sinai. Munoz further opined that it would have been impossible for Steven's injuries to have occurred during the evening of the day before he was brought in to Mount Sinai. Additionally, Munoz opined that Steven could not have walked, talked, eaten, or drunk anything after sustaining these injuries. He later testified that it was not possible that Steven had a lucid interval after sustaining his head injury.

When cross examined, Munoz acknowledged that he had only been board certified for nine months and that this was the first criminal case in which he had testified. However, he had given his opinion in approximately six depositions regarding the timing of an injury. Munoz agreed that history played a large role in determining when an injury occurred. By "history," he meant information

gathered by hospital staff from family members and eyewitnesses to the injury. However, Munoz testified that, in the instant case, he really did not have to look at any history because of the appearance of Steven's brain. The history that Munoz did consider was that Steven was "doing okay" before Kenyatta left for work and that he was not when she returned from work. Furthermore, Munoz has contacted DCFS "hundreds" of times regarding suspected child abuse, and on each occasion, DCFS personnel have asked him to give an opinion regarding the timing of the injury. When asked whether he "extrapolate[d] a lot of [his] opinions on what happened to Steven from car accidents," Munoz replied, "No[,] I extrapolate my opinion from having seen hundreds of children like Steve." However, he did disagree when asked whether it was necessarily scientifically invalid to equate head injuries resulting from motor vehicle accidents with head injuries caused by abuse. Later, he added that the majority of cases of pediatric trauma he has seen arise out of automobile accidents rather than child abuse, but both involve similar mechanisms of injury. Finally, Munoz agreed that a seizure could be a symptom of severe head trauma and that nonmedical personnel could mistake a seizure for choking. After Munoz's testimony concluded, the State offered its exhibits and rested.

The first witness presented by defendant (except for those taken out of order for scheduling reasons) was Officer Figiel, who had testified earlier for the State. Figiel sat in on an interview of Kenyatta Brown on February 15, 2002. Three other individuals were also present. Figiel testified, and the State stipulated, to several impeaching statements. Additionally, Figiel testified that he spoke with Dr. Severin at Rush Presbyterian during the late evening of February 8, 2002. Severin told Figiel that Steven's injuries were 24 to 48 hours old. However, Severin also characterized this estimate as a guess.

Defendant then called Joseph DelGiudice, a detective from the Du Page County sheriff's office. DelGiudice interviewed Kenyatta on February 9, 2002, at about 1:10 a.m. DelGiudice also testified regarding several impeaching statements. DelGiudice spoke with Dr. Green. Green pointed out a "knot" on Steven's head, and Kenyatta stated that Steven had had knots on his head since birth. However, after Green showed Kenyatta the "knot," Kenyatta looked at defendant and asked him what he had done. DelGiudice spoke with Nurse Beasley that day as well. Beasley indicated that Kenyatta's behavior was strange and that she believed Kenyatta "may have been in shock and did not realize the magnitude of the situation." She characterized defendant's behavior as, inter alia, "defensive."

Defendant then called Sergeant Michael Price, also of the Du Page County sheriff's office. In the early morning of February 9, 2002, Price went to the apartment of Kenyatta and defendant. He observed a vomit-stained pillow. Price took a picture of a closet in the bedroom. He also visited the apartment on February 10, 2002, and took more photographs, primarily of the closet area. Several items of clothing were missing. There were two additional empty hangers in the closet at this time.

Denise Foster next testified on defendant's behalf. Foster testified that she is defendant's sister and had known defendant for 24 years. She stated that she had seen Kenyatta strike Steven on two occasions. One occasion occurred at Foster's home in the December or January preceding Steven's death. Kenyatta and Steven were in the bathroom. Foster heard Kenyatta spanking Steven and heard Steven crying. According to Foster, Kenyatta then left Steven on the toilet for about 20 minutes. Foster also witnessed another, earlier incident. Kenyatta slapped Steven, back-handed, at least three times because Steven was crying. During cross-examination, she denied that she did not like

Kenyatta. She later clarified that she got along with Kenyatta fine, but did not like the way Kenyatta treated Steven.

Defendant then called Frank Belpedio. Belpedio is defendant's cousin. He testified that he observed Kenyatta strike Steven on three occasions. The first time was in June or July of 2001. Belpedio was driving a car, defendant was in the front seat, and Kenyatta and Steven occupied the back seat. Steven was crying, and Belpedio heard a loud slap. He then looked in his rear-view mirror and saw Kenyatta slap Steven in the cheek and shoulder. He saw Kenyatta strike Steven a second time at Karen Clark's apartment. The second incident occurred a few weeks after the first one. Belpedio was sitting on the couch with Steven and three other children. Steven started to cry. Kenyatta came into the room, told him to be quiet, and slapped him twice. One slap landed on Steven's head and the other on his back. The slaps were delivered with an open hand. Steven fell off of the couch. The third incident happened sometime after Angelique was born, about three or four days before defendant was arrested (Belpedio also stated it occurred in the summer of 2001). Belpedio testified that Steven was crying. Kenyatta walked over to him, shook him causing his head to flail about, and slapped him. Belpedio heard defendant ask Kenyatta, "what are you doing that for?". She slapped Steven "[o]pen hand[ed] right in the face." On cross-examination, Belpedio acknowledged that he and defendant would "hang out" together socially and that they were "tight." Further, Belpedio added that the third incident left a half-inch scratch upon Steven's face.

Defendant next called Karen Clark--Kenyatta's mother. Clark testified that Steven spent half his life living with her and half of it living with his great aunt, Dorothy Herron. He "never really permanently" lived with Kenyatta. Clark stated that she had never seen Kenyatta slap Steven in the face. However, she acknowledged telling a detective that "Kenyatta slapped Steven in the face a few

times in the past due to his whining and crying." She later clarified that she did not actually see Kenyatta do this.

Defendant then recalled Detective DelGiudice. He testified that he spoke with Clark on February 8, 2002 at Rush Presbyterian at about 11:30 p.m. In that conversation, Clark told DelGiudice that "Kenyatta slapped Steven in the face a few times in the past due to his whining and crying." The State also inquired of DelGiudice at this time regarding a statement made by Denise Foster. Foster told him that she did not like Kenyatta and did not think that Kenyatta was right for her brother. When asked to explain herself, Foster declined to discuss the matter further.

Crystal Zeis (formerly Crystal Holdmann) testified next. She had known defendant for five or six years, and she also knew Steven and Kenyatta. Zeis and her boyfriend lived with Kenyatta and defendant for a while. Zeis saw Kenyatta strike Steven on several occasions. One time, Kenyatta told Steven to throw his diaper away. Steven threw it in the kitchen sink. Kenyatta grabbed him by the arm and threw him out of the kitchen. After cleaning up the mess, Kenyatta shoved Steven into the bedroom and told him to take a nap. Zeis stated that she did not note any injuries. Zeis added that, though she would not say that Kenyatta struck Steven on a daily basis, it did happen on a regular basis or "quite often." "Every time he did something wrong," Zeis testified, "he ended up getting hit for it." Often, this involved an open-handed blow to the side or back of the head. Zeis never saw Kenyatta use an object to discipline Steven, but she did observe Kenyatta strike Steven on various parts of his body including his back, butt, arms, legs, and "wherever [she] could reach at the time."

On another occasion, Zeis stated, Kenyatta flung Steven out of the kitchen after he spilled some juice. Zeis described yet another incident where Kenyatta threw a fan which landed about two or three feet from Zeis's daughter. Zeis never saw Randy strike Steven. Steven "always had bruises

on him." Zeis once saw a mark on Steven's back that looked like it was made by a ruler or some similar object. On a few occasions, Zeis saw defendant attempt to intervene to try to stop Kenyatta "when she got out of hand," but Kenyatta would yell at him and tell him that he was not Steven's father so he should stay out of it. In response to questioning by the State, Zeis acknowledged that she had dated defendant for a short time. She further acknowledged that she did not like Kenyatta.

The last witness to testify for defendant was Dr. Shaku Teas. The trial court recognized Teas as an expert in the area of forensic pathology and child abuse. Teas had previously testified for the State "[p]robably hundreds of times" and on behalf of defendants only about 20 times. Teas reviewed Mileusnic's autopsy report, photographs, and histology[5] slides. She also examined the records from Mount Sinai and Rush Presbyterian as well as DCFS records, police reports, and witness's statements. Teas spoke with Mileusnic prior to her testifying. Regarding the cause of Steven's death, Teas agreed with Mileusnic's opinion that he died as a result of multiple blunt-trauma injuries. Further, she opined that a combination of head and abdominal injuries caused Steven's death.

Teas testified that the type of injury Steven sustained to his abdomen was caused by punching, hitting, kicking, or some sort of crushing mechanism. A person sustaining such an injury, she explained, might experience pain for a while and then be fine for a while. Then, as the ulceration and peritonitis "sets up," pain would become more general and the person might become septic and lose consciousness. Further, a person with such an injury would be able to eat, initially. Injuries like these are not necessarily painful; a person might only feel a little discomfort. She later added that it takes time for a perforation to occur and peritonitis to set in. There is usually a delay between the injury

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[5] "Histology *** is the study of tissue sectioned as a thin slice, using a microtome. It can be described as microscopic anatomy." See http://en.wikipedia.org/wiki/Histology.

and the onset of symptoms. Further, Teas felt that abdominal injuries were easier to time than head injuries.

Regarding the head trauma, Teas stated that she reviewed the CT scan from Mount Sinai. There was no mention of "midline shift." "Midline shift" refers to a state where "there is a mass lesion on that side of the brain and so it is shifting the brain to the opposite side," and its absence indicated that there was not "just one localized area." Additionally, histology slides of the testicles revealed no evidence of trauma. Teas explained that the discoloration in the scrotum was likely due to blood trickling down to that area from the abdomen.

Teas testified that, as a pathologist, assessing the timing of injuries is something she is familiar with doing. Timing is "an integral part of pathology." The best way, according to Teas, to address timing is histology; however, she did also state that it is "imprecise." Thus, estimates of the timing of an injury are usually given in days. She testified that she would never give, nor has she ever seen given in any textbook or paper, an estimate in terms of hours. She did believe that all of Steven's injuries occurred within a 24-hour period.

Teas found evidence of acute inflammatory cells in Steven's gastrointestinal tract. These typically appear about 12 to 24 hours after an injury. She did not recall seeing any in the subdural hematoma, however. On about the second day following an injury, mononuclear cells (also called lymphocytes) start to appear and grow in numbers. On the third day, and possibly on the second, spindle-shaped cells called fibroblasts begin to lay down collagen. As time passes, Teas explained, layers of fibroblasts increase; hence, the more that are present, the older the injury. Also, at about the same time, new capillaries form, making the surface of the area very granular. In Steven's case, Teas saw early granulation of tissue and several layers of fibroblasts. After between four and five

days, there are three to five layers of fibroblasts, and at 7 to 10 days, the capillaries become very prominent. "In this case," said Teas, "you could almost see the capillaries."

Teas opined that Steven's injuries were about five days old. She said they could have been six days old and it was possible that they were only four days old. However, in her opinion, due to the amount of healing that had taken place, they could not have been less than four days old. Steven was pronounced dead on February 11, 2002, at about 12:30 p.m. Discounting the fact that Steven was on a respirator for a while, which may have slowed healing, Steven's injuries would have occurred, according to Teas, "on or around February 6." Teas testified that she also found evidence of injuries that could have been a month old. In a histological slide, she found a new blood vessel forming in the dura, which requires four to five days after an injury to occur. Teas also stated that it was more likely that Steven's injuries were seven days old as opposed to three days old. Later, Teas testified that children heal a bit faster than adults, but that would be offset by the respirator.

Teas further testified that Steven's purported choking on a hotdog could be secondary to either his abdominal injuries or his head injuries. She also stated that a seizure resulting from a head injury could result in clenching of the teeth and would involve shaking in other areas of the body. Furthermore, timing an injury based on bruising is problematic, as "each person responds differently." A person who suffered the type of head injury experienced by Steven could remain conscious. Teas stated that she reviewed the postoperative reports of Munoz, and they did not change her opinion that Steven could have remained conscious. That Steven's intercranial pressure was 90 and rapidly progressing (normal is 10 to 20) was consistent with an injury occurring days before, as the brain does not necessarily start to swell at the time of an injury.

Also, having a history of the injury is important in determining timing. Furthermore, extrapolating data and opinions from automobile accidents and applying it to a nonaccidental injury is problematic. Teas explained that she typically sees patterns of injuries that are "a little different" in car accidents and instances of child abuse. When asked again about timing, Teas stated, "The subdural--I can't say anything about the other head injury, but the subdural and abdominal were consistent with five days." These injuries, she added, were the cause of death. Red lines on Steven's foot noted in a Rush Presbyterian record from 3:30 a.m. on February 9, according to Teas, could have been caused by tubes from a blood pressure cuff on Steven's leg.

During cross-examination, Teas explained that calcification (which she observed in the dura) could indicate that the injury is older than the presence of fibroblasts would indicate. It could also be evidence of a second, earlier injury. Teas acknowledged that Mileusnic was of the opinion that the calcification was an artifact of the surgery on the right side of Steven's head. She further acknowledged that her report stated that Steven was transferred to Rush Presbyterian on February 9, 2002, at 11 a.m., which is incorrect as to both date and time. Teas did not differentiate between who reported what injury in her report; that is, the report does not specify that certain injuries that manifested themselves later were not apparent at the time Steven was brought in to Mount Sinai. Later, she stated she "sort of *** tried to separate" what was observed at Rush Presbyterian. She also explained that there were some discrepancies in the records of the anesthesiologist and the surgeon. However, these discrepancies did not affect her opinions. According to Teas, posturing is a type of a seizure. Following Teas testimony, defendant rested.

The trial count found defendant guilty of first-degree murder (see 720 ILCS 5/9--1 (West 2002)). The trial court also found that Steven was under the age of 12, but declined to find that the

murder was accompanied by exceptionally brutal and heinous behavior indicative of wanton cruelty" (see 730 ILCS 5/5--5--3.2 (West 2002)). In so ruling, it made the following findings. Steven was born on April 17, 1999 and was a normal, healthy child. He had been raised primarily by his grandmother, Karen Clark, and his aunts, Dorothy Herron and Sadie Brown. None of these women had ever seen any marks or injuries on Steven indicative of child abuse after Steven had returned from being with Kenyatta. The court found them "extremely credible." It also found that they clearly loved Steven and "would have done everything in their power to keep him from Kenyatta had they thought for a moment that he was in danger."

The cause of Steven's death was blunt trauma to the head and abdomen. The court stated that the sole issue was who caused that trauma. The court explained that the defense's theory was that Kenyatta beat Steven so severely that he was mortally wounded early in the week of February 2, 2002, "perhaps as early as the 4th, 5th, or 6th." Then, Steven experienced a "lucid interval," which lapsed about an hour before Kenyatta returned home on February 8, 2002. Kenyatta's purported history of abusing Steven was presented through the testimony of Zeis, Martinez and Beldepio. However, the trial court expressly found that two of the witnesses (Zeis and Belpedio) were not credible. The court reiterated that Clark, Brown, and Herron never observed an signs of abuse after Steven was with Kenyatta. The pathologists that defendant offered in support of his position, dated Steven's injuries, based on the rate of healing, as occurring between the fourth and eighth or between the fifth and ninth of February. The court observed that these estimates, while "hardly an exact measurement," were further complicated by other factors that affected the rate of healing such as oxygenation, Steven's youth, and the use of a respirator. It is clear that the court did not find the opinions of the pathologists particularly useful and did not attribute great weight to them. In sum,

the trial court found that "[t]he evidence *** does not support any allegation that Kenyatta Brown chronically abused Steven Quinn."

The State's theory, on the other hand, was that Steven was in the sole care of defendant starting at about 10 a.m. on February 8, 2002. When Kenyatta left for work at that time, Steven was healthy; when she returned at 4 p.m., Steven was nonresponsive, " 'breathing funny,' " eyes locked in a cold stare, with vomit in his mouth.

The trial court found the testimony of the medical personnel who treated Steven most compelling. By 6:18 p.m. on February 8, 2002, Steven was posturing. At this time, a treating nurse did not see any marks on Steven's legs or ankles. Nurse Smith at Rush Presbyterian later observed fresh red bruises and marks on Steven's legs. At about 11:30 p.m., welts were noted on his left foot. Moreover, the marks were changing and becoming more defined. Dr. Green observed red marks on his head. That they were red, she testified, indicated that they were fresh. Dr. Boykin testified that Steven could not have eaten a hot dog, as described by defendant, after he sustained these injuries. Because Steven could not have eaten after sustaining such injuries, he had to have sustained them after he last ate. Further, he could not have sustained these severe injuries on February 7, for he would not have survived until the time he was brought to Mount Sinai. At Rush Presbyterian, injuries that were not visible at Mount Sinai continued to appear. Steven's abdomen was not distended until the following morning. Dr. Severin observed that Steven's autoregulation system was intact during the evening of February 8, indicating that the brain injury was recent rather than days old. Amylase and lipase levels had doubled between the time blood tests were taken at Mount Sinai and later at Rush Presbyterian. The only explanation for such an increase, found the trial court, was the recency

of the injury. A Cullen's sign appeared on the morning of February 9. Severin stated that these appear relatively quickly after a severe injury to the pancreas.

Dr. Munoz observed bright red blood, which also indicated a recent injury. He added that it was impossible for the injury to have been sustained on the previous evening, for Steven would have been unable to walk, talk, eat, or drink. A head injury of this magnitude would preclude a lucid interval.

The court noted that the sudden appearance of all of Steven's injuries on the evening of February 8 was inconsistent with a chronic pattern of abuse by Kenyatta. One would expect to see injuries of a different age. The court also noted that while defendant had failed to change Angelique's diaper, he did change Steven's. It also relied upon Robert Liebich's testimony that defendant told him that he "didn't hit the kid that hard," noting that this was an admission that he did, in fact, hit Steven. The court further noted defendant's changing demeanor as the night progressed and the extent of Steven's injuries became clear.

On August 27, 2004, the trial court heard defendant's motion for a new trial. At the beginning of that proceeding, defense counsel informed the trial court that defendant was in the process of attempting to hire private counsel because he had raised issues pertaining to the ineffective assistance of counsel. Defense counsel also told the trial court that the attorneys defendant contacted would not proceed unless they were either retained by defendant or appointed by the court. The trial court, after asking the State for input, simply stated:

"The Court has appointed the public defender to represent you, and if the public defender--If you chose to to [sic]dismiss the public defender, you have the right to hire your

own attorney, but the Court will not give you an additional attorney because you do not wish to proceed today."

Defendant responded, "Okay." The court then turned to the motion for a new trial, which it ultimately denied. In the course of arguing the motion, defense counsel, at defendant's request, raised the following issues regarding ineffective assistance of counsel: (1) that counsel should have brought in additional witnesses to impeach Kenyatta regarding her purported abuse of Steven; (2) that counsel should have had a witness testify that defendant had changed Steven's diapers in the past; (3) that counsel should have brought in additional witnesses to testify regarding other inconsistencies in Kenyatta's testimony; (4) that other detectives should have been called to testify--in addition to Figiel--regarding a "hollow noise" Kenyatta stated she heard while defendant was in the bedroom with Steven; (4) that counsel should have conducted more thorough cross-examinations; (5) that counsel failed to present evidence that no clothes hangers were found in the garbage on February 9, 2002. Counsel then stated, "I believe that covers everything that Mr Liebich wanted us to bring up for ineffective assistance." Later during the hearing, when defense counsel was discussing the testimony of Dr. Severin, defendant interjected, "When they said the bruising was zero to 24 to 48, which was not said. That was never cleared up."

Also, on August 27, 2004, the State called to the trial court's attention the fact that Dr. Teas had sent the court a letter. Copies were also sent to the State and defense counsel. The State asked that the trial court not view it, as it was an "improper ex parte communication." Defense counsel agreed. The court impounded the letter and made it part of the record. On September 9, 2004, the trial court sentenced defendant to 65 years' imprisonment.

Defendant--by counsel--filed a motion to reconsider sentence. He also filed a pro se motion alleging the ineffective assistance of counsel. The latter motion was filed on November 8, 2004. On November 10, the court asked defendant what he was asking for in his motion. Defendant replied that he wanted new counsel and "hopefully" a new trial. In a hearing on November 29, the trial court stated to defendant, "[Y]ou've indicated to your attorney that you wish to make your argument in writing, and you have a copy?" Defendant replied that the copy was illegible. The trial court then made arrangements for defense counsel to make and file a legible copy.

On December 7, 2004, the final hearing in this cause was held. The trial court asked defendant about the papers he had filed and whether they constituted separate motions. Defendant explained that one was a motion, and two packets were arguments in support of that motion. The following colloquy then ensued between defendant and the trial judge:

"THE COURT: What is your position then

MR. LIEBICH: I just feel that I didn't receive a fair trial because my attorneys weren't fully prepared to try this case. And I pretty much stand on my written argument.

THE COURT: A lot of what you say in there are conclusions. Do you have anything to back up those conclusions?

MR. LIEBICH: No.

THE COURT: If you say somebody wasn't prepared, what do you mean?

MR. LIEBICH: Well, there's no way he could have been prepared by failing to present most of this evidence that was critical to my defense. Mr. Holman wasn't even aware of a lot of the stuff that I brought to his attention during the trial. So I don't feel there's any way he could have been fully prepared not knowing about this information.

THE COURT: Again, do you have any examples? I read your, the longer presentation.

MR. LIEBICH: Basically a lot of it's just evidence that he didn't bring forward that was in discovery.

THE COURT: For example?

MR. LIEBICH: Let's see. There were other possible witnesses that should have been brought forward that he didn't call.

THE COURT: For example, who would that be and what would they have testified to?"

Defendant then listed the following purported deficiencies: (1) counsel failed to call Richard O'Brien, a polygrapher, to testify that Kenyatta admitted striking Steven with a belt and comb; (2) counsel failed to call Dion Liebich to impeach Robert Liebich by testifying that defendant never asked Robert whether the room in the Roselle police station was "bugged" during their conversation; (3) counsel failed to bring forth evidence from Kenyatta's diary that Lee Clark (Karen's husband) beats the children in the Clark home and Karen does not interfere, to undermine the notion, cited by the trial court in its adjudication of guilt, that Karen would have done anything to protect Steven; (4) counsel failed to bring forth evidence that Steven had Tylenol and aspirin in his system to undermine the proposition that Steven was feeling fine prior to February 8, 2002; (5) counsel failed to bring forth evidence that Steven lost five pounds between November 6, 2001, and February 8, 2002; (6) counsel failed to bring forth evidence that Steven had been given drugs to sedate him prior to the time he was first examined at Rush Presbyterian, which caused his stomach to be soft and not distended; (7) counsel failed call Dorothy Herron to testify that February 8, 2002 was not the first time Steven was

left home alone with defendant; and (8) counsel failed to bring forth evidence or investigate information regarding a child in Rockford, whose mother's name was Kenyatta Brown, "mysteriously end[ing] up with a broken leg."

After defendant set forth these complaints, the trial judge asked, "Anything else?" Defendant replied, "That's pretty much it, your Honor." The trial court then inquired of one of defendant's attorney--John Casey--regarding whether he wished to address any of these issues. Casey stated that the facts defendant set forth were correct; however, he maintained that the decision regarding whether to present them was a matter of trial strategy. The court then requested that defendant's other attorney, Ricky Holman, be brought to the court room.

The court then asked Holman whether he wished to offer any explanations regarding the issues raised by defendant. Holman explained that, as the trial court had previously observed, it would be unreasonable to expect him to call defense witnesses to offer additional impeachment after the court assessed testimony of certain state witnesses, particularly where those state witnesses were in fact impeached. Holman stated that he reviewed reports from additional witnesses and made a strategic decision not to present them. Evidence of aspirin and Tylenol was contained in Teas's ex parte letter, and thus, though he was now aware of it, he could no longer use this evidence. Investigators from both the public defender's office and the State determined that the Kenyatta Brown in Rockford was not the same person as the Kenyatta Brown involved in this trial.

The trial court then asked Holman whether he failed to communicate a plea agreement to defendant. Holman stated that he did communicate one posttrial offer to defendant, which they agreed to reject. Prior to trial, said Holman, no offers were made, and defendant and Holman agreed to maintain defendant's innocence. Holman did tell defendant that the State had made overtures

regarding a plea, even though no firm offer was ever made. The court also inquired of Holman regarding defendant wishing to testify, which Holman denied. The court then ruled, finding that defendant's allegations pertained to evidence that was cumulative, collateral, or not of any particular relevance. The court also found that no offer was made before trial. It then ruled that it was not necessary to appoint new counsel, and it denied defendant's pro se motion. It further ordered that defendant's pro se motion be incorporated into defendant's original posttrial motion for the purpose of preserving these issues for appellate review. The trial court specifically asked whether either party objected to this final ruling, and neither side did. It also denied defendant's motion to reconsider the sentence it had imposed. Defendant now appeals.

II. ANALYSIS

Defendant raises a number of issues on appeal. First, he asserts that the trial court did not conduct an adequate inquiry into his pro se allegations of ineffective assistance of counsel and that this cause should be remanded for such an inquiry. Next, he contends that he was not proven guilty beyond a reasonable doubt because (1) the trial court considered evidence that was inadmissible under Frye v. United States, 293 F. 1013 (D.C. Cir. 1923); (2) the State failed to prove the essential elements of first-degree murder and instead only proved involuntary manslaughter; and (3) the trial court made inconsistent findings regarding defendant's mental state. Third, defendant argues that the testimony of Dr. Munoz and Dr. Severin should not have been admitted under Frye. Fourth, defendant claims trial counsel was ineffective. We will address these arguments as defendant presents them in his brief.

A. ADEQUACY OF THE INQUIRY INTO DEFENDANT'S PRO SE ALLEGATIONS OF INEFFECTIVE ASSISTANCE OF COUNSEL

Defendant first argues that the trial court did not conduct an adequate inquiry into his pro se claims of ineffective assistance of counsel. See People v. Moore, 207 Ill. 2d 68, 81 (2003). Defendant point out that, on August 27, 2004, he made an oral motion for a new trial alleging trial counsel's ineffectiveness. The trial court, after asking only the State for input, stated:

> "The Court has appointed the public defender to represent you, and if the public defender--If you chose to to [sic]dismiss the public defender, you have the right to hire your own attorney, but the Court will not give you an additional attorney because you do not wish to proceed today."

The trial court made no inquiry of defendant, or, for that matter, defense counsel.

When a defendant makes a pro se charge of ineffective assistance of counsel, the appointment of new counsel is not always necessary. Moore, 207 Ill. 2d at 77. Instead, the trial court must conduct an inquiry into the factual basis of the defendant's claim. Moore, 207 Ill. 2d at 77-78. Where a claim lacks merit or only touches upon trial strategy, new counsel need not be appointed. People v. Williams, 147 Ill. 2d 173, 251 (1991). Only where the claim shows possible neglect of the case is new counsel necessary. Williams, 147 Ill. 2d at 251.

On review, the inquiry focuses upon the adequacy of the trial court's inquiry into a defendant's claims. People v. Johnson, 159 Ill. 2d 97, 125 (1994). The inquiry may take three forms. Typically, some discussion between the trial court and trial counsel, including counsel simply answering questions and explaining the circumstances surrounding defendant's allegations, is usually necessary. Moore, 207 Ill. 2d at 78. Additionally, the trial court may discuss the allegations with the defendant. Moore, 207 Ill. 2d at 78. Further, the trial court may rely on its own recollection of defense counsel's performance. Moore, 207 Ill. 2d at 79. A defendant's allegations may also be insufficient on their

face. Moore, 207 Ill. 2d at 79. Whether the trial court made an adequate inquiry into a defendant's pro se claims of ineffective assistance of counsel presents a question of law subject to de novo review. People v. Strickland, 363 Ill. App. 3d 598, 606 (2006).

In this case, the trial court did not discuss defendant's allegations with either defense counsel or defendant during the hearing on August 27, 2004. There is also no indication in the record that it was relying on its own recollection of defense counsel's performance. The record does not reveal, and the State does not suggest, that the claims were facially insufficient. Indeed, defense counsel informed the court that defendant was raising the issue of ineffectiveness, but counsel did not relay to the court the nature of those claims and the court did not make any inquiry into what, precisely, they were, much less their factual basis.[6] The trial court simply dismissed the claim, stating defendant

[6] In this section of his brief, defendant states that "many or most of [his] pro se claims dealt with counsel's failure to do things at trial, such as call witnesses, so it is unclear how the trial court's recollection would shed any light on this issue." Later during the hearing, after the trial court denied defendant's motion, defense counsel set forth defendant's claims with some specificity. These issues were (1) that counsel should have brought in additional witnesses to impeach Kenyatta regarding her purported abuse of Steven; (2) that counsel should have had a witness testify that defendant had changed Steven's diapers in the past; (3) that counsel should have brought in additional witnesses to testify regarding other inconsistencies in Kenyatta's testimony; (4) that other detectives should have been called to testify regarding a "hollow noise" Kenyatta stated she heard while defendant was in the bedroom with Steven; (4) that counsel should have conducted more thorough cross-examinations; (5) that counsel failed to present evidence that no clothes hangers were found in the garbage on February 9, 2002. The trial court expressly directed that these issues, though presented orally, had

had an attorney, had the right to hire private counsel, but would not be appointed an additional attorney. The State agrees that the trial court did not make an adequate inquiry into defendant's claims during the August 27, 2004, hearing.

However, the State contends that, under the unique facts and circumstances of this case, a remand is not necessary. The State points out that defendant filed a written motion alleging ineffective assistance of counsel on November 10, 2004. On that date, the State reminded the court of its obligation to examine the factual basis of defendant's allegations. A hearing was ultimately held on defendant's motion on December 7, 2004. During this hearing, the trial court conducted an extensive inquiry into defendant's allegations, asking defendant to clarify and exemplify them. Defendant, in fact, articulated eight distinct complaints about defense counsel's conduct. After defendant specified his complaints, the trial judge asked, "Anything else?" Defendant answered, "That's pretty much it, your Honor." The trial court also inquired of both of defendant's trial attorneys. The State asserts that this inquiry cured any deficiency in the trial court's earlier handling of defendant's claims. We agree.

Essentially, defendant is asking that we remand this cause so the trial court can ask "Anything else?" yet one more time. The remedy where a trial court fails to make an adequate inquiry into a claim of ineffectiveness is a remand to allow the court to make the proper inquiry. Moore, 207 Ill. 2d at 81. Neither a full evidentiary hearing on the question of trial counsel's purported incompetence nor the appointment of new counsel to aid in the inquiry is required. Moore, 207 Ill. 2d at 81-82. On December 7, 2004, the trial court inquired, defendant explained the factual basis for his claims, defendant stated that there were no additional issues he wished to raise, and the court ruled. Insofar

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been sufficiently raised and preserved.

as ineffectiveness is concerned, it is not apparent to us how a hearing on remand would differ in any appreciable way from the hearing conducted on December 7. Defendant raised all the issues he wanted to at that time, as he indicated when he stated, "That's pretty much it." A remand so that this issue could be rehashed one more time would be both a meaningless gesture and a waste of judicial resources. The trial court already ruled on the issues defendant raised, and defendant provides no basis for supposing that a different result would obtain on remand. See People v. Blair, 215 Ill. 2d 427, 446-47 (2005) ("To hold otherwise, we would be forcing courts to waste judicial resources by merely delaying the dismissal of a petition which the judge knows could never bear fruit for the petitioner").

In short, we will not engage in such a meaningless gesture. As defendant was given an adequate opportunity to present his claims to the trial court and indicated that he was satisfied that he had presented them all, and further because the trial court made an extensive inquiry of defendant and both his trial attorneys, we conclude that any error resulting from the trial court's summary dismissal in August was cured in the December hearing. No remand is necessary because the trial court already made the inquiry we would be directing it to make on remand. Finally, we find no error regarding the trial court's determination that new counsel was not necessary. Following a proper inquiry, such a decision will be disturbed only if it is manifestly erroneous (People v. Young, 341 Ill. App. 3d 379, 382 (2003). Here, the trial court's point-by-point explanation of its reasons for not appointing new counsel are clearly sufficient under that standard.

B. PROOF OF GUILT BEYOND A REASONABLE DOUBT

Defendant makes three arguments as to why he was not proven guilty beyond a reasonable doubt. He argues that the trial court considered inadmissible evidence, that the State failed to prove

the essential elements of the crime, and that the trial court made inconsistent findings. We will address these contentions in turn.

1. Inadmissible Evidence under Frye

While defendant characterizes this argument a Frye issue (see Frye v. United States, 293 F. 1013 (D.C. Cir. 1923)), in actuality, he is simply asking that we reweigh the evidence and accept the testimony of two expert witnesses that opined favorably to him rather than the treating physicians upon whom the trial court relied. Frye is, in fact, only cited once, in the course of a three-sentence discussion, and its tenets are not discussed or applied in a sustained or coherent form.

Defendant begins this argument by setting forth the strengths of the two favorable experts--Dr. Mileusnic Polchan and Dr. Teas. Both are forensic pathologists with impressive credentials and are certainly qualified to opine as to the timing of the injuries inflicted upon Steven. Further, both relied upon histological analysis, a well-accepted methodology, in coming to their conclusions.

Before proceeding further, we note that Mileusnic stated that the injuries "could have occurred on the 8th," which is consistent with the State's position and the testimony of Dr. Munoz and Dr. Severin. Thus, even if we were to accept defendant's invitation to reassess the evidence, it is unclear to whom we would deem Mileusnic's testimony favorable. In addition to setting forth the credentials and testimony of Mileusnic and Teas, defendant also points out that no witness saw him strike Steven and no physical evidence links him to Steven's death (we cannot help but note that such observations have nothing to do with the propriety of the testimony of Munoz and Severin under Frye, which reinforces the notion that defendant is actually asking us to reweigh this evidence). We also note the Mileusnic stated that she would defer to a treating physician on the issue of timing and

that there was testimony that Steven was fine on the morning of February 8 before being left alone with defendant for over six hours and injured after that.

Defendant attacks Munoz's qualifications to determine the timing of an injury, incorrectly stating that Munoz "offered no expert experience in determining the timing of injuries." To the contrary, Munoz testified DCFS had consulted with him "hundreds" of times regarding suspected child abuse, and on each occasion, he was asked to give an opinion regarding the timing of the injury. Defendant also disingenuously states that neither Munoz nor Severin "had ever testified in court about the timing of injuries before." (Emphasis omitted and added.) Munoz, in fact, testified that he had given his opinion in six depositions previously. While, strictly speaking, these opinions were not given "in court," they were obviously given in the course of legal proceedings.

Defendant does complain of Munoz's testimony regarding the timing of Steven's injuries based on the color of the blood Munoz found when he opened Steven's head. The color of the blood was bright red, which Munoz took as a sign of a recent injury. The State asserts that this deduction is not subject to Frye. Scientific evidence is that which derives from the "application of scientific principles, rather than on skill or experienced-based observations, for the basis of his opinion." Jackson v. Seib, 372 Ill. App. 3d 1061, 1073 (2007). The Sixth Circuit Federal Court of Appeals illustrated the distinction between scientific and nonscientific opinion evidence thusly:

> "The distinction between scientific and non-scientific expert testimony is a critical one. By way of illustration, if one wanted to explain to a jury how a bumblebee is able to fly, an aeronautical engineer might be a helpful witness. Since flight principles have some universality, the expert could apply general principles to the case of the bumblebee. Conceivably, even if he had never seen a bumblebee, he still would be qualified to testify, as

long as he was familiar with its component parts.

On the other hand, if one wanted to prove that bumblebees always take off into the wind, a beekeeper with no scientific training at all would be an acceptable expert witness if a proper foundation were laid for his conclusions. The foundation would not relate to his formal training, but to his firsthand observations. In other words, the beekeeper does not know any more about flight principles than the jurors, but he has seen a lot more bumblebees than they have." Berry v. City of Detroit, 25 F.3d 1342, 1349-50 (6th Cir. 1994).

Munoz is more like the beekeeper. His expertise is not derived from the abstract application of scientific principles; rather, it is based upon what he has observed in his years as a doctor. Hence, his observation that bright red blood is indicative of a recent injury is not subject to Frye because it is grounded in his own experience. Defendant was, of course, free to attack Munoz's experience regarding whether it was a sufficient basis to render this opinion, but such arguments go only to the weight to which the opinion is entitled, not its admissibility. People v. Swart, 368 Ill. App. 3d 614, 633 (2006).

Defendant attacks Severin to the extent that he relied upon Munoz's conclusions. As we have determined that Munoz's testimony was proper, defendant's derivative attack upon Severin must also fail. Accordingly, we find defendant's assertion that the trial court relied on testimony that was not admissible under Frye ill founded. Moreover, to the extent that the opinions of Munoz and Severin conflicted with those of Mileusnic and Teas, it was for the trial court, in the first instance, to resolve that conflict, (People v. Harrison, 366 Ill.App. 3d 210, 219 (2006)), and its resolution of this issue is amply supported by the evidence.

2. Elements of First-Degree Murder

Defendant next asserts that the State failed to prove him guilty of first-degree murder. In reviewing the sufficiency of the evidence to sustain a verdict, we must construe the record in the light most favorable to the State. People v. Tabb, 374 Ill. App. 3d 680, 691 (2007). At issue is whether "any rational trier of fact could have found the essential elements of the crime beyond a reasonable doubt." People v. Bush, 214 Ill. 2d 318, 326 (2005). A reviewing court will not set aside a conviction unless the evidence is so unsatisfactory or the possibility of a defendant's guilt so improbable as to raise a reasonable doubt regarding that guilt. People v. McGee, 373 Ill. App. 3d 824, 832 (2007).

Defendant focuses his argument upon the State's evidence concerning mens rea. The first-degree murder statute provides:

> "A person who kills an individual without lawful justification commits first-degree murder if, in performing the acts which cause the death:
>
> (1) he either intends to kill or do great bodily harm to that individual or another, or knows that such acts will cause death to that individual or another; or
>
> (2) he knows that such acts create a strong probability of death or great bodily harm to that individual or another; or
>
> (3) he is attempting or committing a forcible felony other than second degree murder." 720 ILCS 5/9--1 (West 2002).

Defendant was in fact indicted on four counts of first-degree murder. The first count alleged defendant acted "knowing said act would cause the death of Steven Quinn." The second count alleged that defendant intended to do great bodily harm to Steven. The third count alleged that defendant knew his actions created a strong probability of death. Finally, the fourth count alleged

defendant knew his actions created a strong probability of great bodily harm. The State apparently nolle prossed the first count, and the trial court convicted defendant on the remaining counts but merged the last two into the second for purposes of sentencing. Relevant here, "intent" is defined as "when [a person's] conscious objective or purpose is to accomplish that result or engage in that conduct." 720 ILCS 5/4--4 (West 2002). "Knowledge" with respect to the result of conduct exists when a person is "consciously aware that such result is practically certain to be caused by his conduct." 720 ILCS 5/4--5(b) (West 2002).

> Defendant contends that the State only proved recklessness, which is defined as follows:
>
> "A person is reckless or acts recklessly, when he consciously disregards a substantial and unjustifiable risk that circumstances exist or that a result will follow, described by the statute defining the offense; and such disregard constitutes a gross deviation from the standard of care which a reasonable person would exercise in the situation." 720 ILCS 5/4--6 (West 2002).

Defendant contends that the trial court has an independent duty to ascertain whether a defendant is guilty of a lesser-included offense, even if defense counsel did not advance such an argument. Indeed, the law is clear that, in a bench trial, "the judge determines from the evidence whether the defendant is guilty of murder or of some lesser included offense, and the defendant has no 'right' to restrict the judge's determination to the question of his guilt or innocence of murder." People v. Garcia, 188 Ill. 2d 265, 273 (1999), quoting People v. Taylor, 36 Ill. 2d 483, 488-89 (1967); see also People v Turner, 337 Ill. App. 3d 80, 90 (2003).

In support of this argument, defendant points to the trial court's findings that he was calm, nonchalant, aloof and relaxed, rather than tearful or nervous during the initial period after arriving

at Mount Sinai. It further stated that he had a "flat affect." At this time, the court observed, the seriousness of Steven's injuries had not been communicated to defendant. As defendant learned of Steven's condition, he became scared and his demeanor changed. The court also noted defendant's statement to his cousin, Officer Robert Liebich, that he "didn't hit the kid that hard." This statement, the court found, was consistent with defendant's lack of concern when they first arrived at the hospital. As the trial court put it, "It also explains why he was not concerned, originally, being at Mount Sinai because it is his belief he didn't hit the kid that hard, what could be wrong." These findings, according to defendant, show only recklessness.

The State counters that it is sufficient to prove that defendant "voluntarily and willfully committed an act, the natural tendency of which is to destroy another person's life, with the intent being implied from the character of the act, and the disparity in size and strength between the defendant and the victim." See People v. Reeves, 228 Ill. App. 3d 788, 798 (1992). Further, knowledge is often proven by circumstantial evidence rather than direct proof. People v. Brogan, 352 Ill. App. 3d 477, 493 (2004). The State points to the quantity and severity of the injuries Steven sustained, and we agree that, from this alone, it is possible to infer an intent to kill or do great bodily harm as well as knowledge on defendant's part that his acts created a strong probability of great bodily harm. See People v Tye, 141 Ill. 2d 1, 16 (1990) ("In the present case, the defendant was an adult male, and the victim was a three-year-old child. In beating the child, the defendant used first a belt and then an extension cord. According to the defendant's own statement, the beating lasted about an hour. The injuries sustained by the child were severe and too numerous to count. Considering the disparity in size between the defendant and the victim, the brutality and duration of

the beating, and the severity of the victim's injuries, we conclude that the trial judge could infer that the defendant acted with the necessary mental state in bringing about the child's death").

In People v. Rodriguez, 275 Ill. App. 3d 274, (1994), the First District of this appellate court confronted a situation similar to the one present here. Rodriguez involved the beating death of a three-year old child perpetrated by an adult male. The medical examiner testified that during the autopsy, he observed numerous bruises on the child's face, ears, back, left buttock, arms, and legs. The victim's stomach was distended, and the medical examiner discovered a large amount of blood in her abdominal cavity. There were three lacerations to the child's intestines. It would have required significant force to inflict such injuries, opined the medical examiner. Additionally, multiple areas of bleeding were discovered in the soft tissue over the victim's skull. There was evidence that defendant struck the victim twice in the abdomen with an open hand. Unlike this case, there was also evidence of previous abuse by the defendant; however, the Rodriguez court expressly noted that it would have come to the same conclusion even without such evidence. Rodriguez, 275 Ill. App. 3d at 286. Similarly, there was evidence that the defendant had struck the victim out of anger, but the court found this to be additional, supporting evidence. Rodriguez, 275 Ill. App. 3d at 285.

Relevant for our purposes is the Rodriguez court's analysis of the victim's injuries as it bore upon the defendant's mental state. The court began by observing, "With respect to whether a defendant intends to kill or knows that his actions are probable to cause death or great bodily harm, an accused's intent or knowledge can be reasonably inferred from, and are often proved by, the circumstances surrounding the incident including the nature and severity of the victim's injuries." Rodriguez, 275 Ill. App. 3d at 284. An inference that a defendant intended or had knowledge that his actions created a strong probability of death "arises when a defendant strikes a blow with a bare

hand when a great disparity in size and strength exists between him and the victim even though a bare hand is not ordinarily regarded as a deadly weapon." Rodriguez, 275 Ill. App. 3d at 284. The court further reasoned:

> "[T]he admissible evidence in this case overwhelmingly proved that the defendant possessed the requisite mental state for first-degree murder at the time of the killing. A great disparity in size and strength obviously existed between the three year old, three foot tall, thirty pound victim and the defendant, who admitted to striking two blows to her abdomen. [Citation.] [The medical examiner] testified that 'significant force' would have been necessary to produce the injuries to the mesentery which resulted in the victim's death and Detective Winistorfer described the defendant's demonstration of the manner in which he struck her as delivering 'two forceful blows' with the palm of his open hand which 'reverberated in the room and on the wall.' " Rodriguez, 275 Ill. App. 3d at 285.

It then held: "This evidence showing the great disparity in size between the victim and the defendant, the vital part of the victim's body to which the fatal blows were struck, and the testimony suggesting that they were in fact forceful, together create a strong inference that the defendant either knew that his acts created a substantial probability of, or that he intended to cause, death or great bodily harm." Rodriguez, 275 Ill. App. 3d at 285.

Rodriguez provides sound guidance for the resolution of this issue. Like Rodriguez, in the present case, defendant is an adult and Steven was a child, so there was a great disparity in size between defendant and Steven. Further, a number of the blows suffered by Steven were directed to his head, and, as in Rodriguez, his abdomen. These are vital parts of the respective victims' bodies. In Rodriguez, the medical examiner testified that significant force was required to inflict the injuries.

In ths case, Mileusnic stated that the injuries inflicted upon Steven would not be caused by "normal corporal punishment," but would require "something much more forceful." In sum, knowledge and intent can both be inferred from the severity and quantity of the injuries defendant inflicted upon Steven. As we stated previously, "a conviction will not be set aside on grounds of insufficient evidence unless the proof is so improbable or unsatisfactory that there remains a reasonable doubt as to the defendant's guilt." People v. Doll, 371 Ill. App. 3d 1131, 1135 (2007). Such is not the case here.

3. Inconsistent Findings

Defendant next asserts that the trial court made inconsistent findings in that it found he did not appreciate the gravity of the situation when he first arrived at Mount Sinai. The trial court noted that defendant's demeanor changed as the night progressed, and, as he became aware of Steven's condition, defendant became scared. The court pointed to Robert Liebich's testimony that defendant told him he "didn't hit the kid that hard." This, according to the trial court, explained his nonchalant demeanor when he first arrived at the hospital: "it is his belief that he didn't hit the kid that hard, what could be wrong." This finding, according to defendant, is inconsistent with the trial court's finding that he acted knowingly and intentionally when he killed Steven. In support, defendant relies primarily on cases involving inconsistent verdicts (see e.g., People v. Hoffer, 106 Ill. 2d 186 (1985); People v. Spears, 112 Ill. 2d 396 (1986)), which are not directly on point here. Unlike these cases, the trial court never reached verdicts that were not consistent. Instead, what defendant is complaining about is an apparent discrepancy between one of the trial court's findings and its ultimate verdicts.

Initially, we note the finding of which defendant complains is not entirely inconsistent with all of the verdicts in this case. Defendant points out that "it is legally impermissible to find that a defendant's mental state is both less-than-knowing and knowing." In Hoffer, 106 Ill. 2d at 195, for example, the court determined that the mental state necessary to convict a person of involuntary manslaughter precluded the existence of the mental state necessary for a murder conviction. This is because involuntary manslaughter is defined as the unintentional killing of a human being caused by acts that are performed recklessly. Thus, it expressly excludes an intentional killing from its scope. Further, since recklessness is defined as the conscious disregard of a substantial risk (People v. Barham, 337 Ill. App. 3d 1121, 1130 (2003)), and "knowledge" for the purpose of the murder statute requires awareness of a "strong probability" of death or great bodily harm (see 720 ILCS 5/9--1 (West 2002)), these mental states are mutually exclusive in that they require awareness of different levels of risk. However, in this case, the trial court did not find defendant guilty of involuntary manslaughter, it simply found he was unaware of the gravity of the situation when he arrived at Mount Sinai. In other words, the finding at issue in cases like Hoffer--that the defendant was guilty of involuntary manslaughter--entails a finding that the killing was unintentional, as per the statutory definition of the crime. In this case, the trial court made no finding regarding defendant's intent, and, more importantly, its finding that defendant did not comprehend the seriousness of Steven's condition does not preclude a finding that defendant intended to cause Steven great bodily harm at the time he acted.

Indeed, defendant's argument, in a sense, compares proverbial apples and oranges. Defendant's mental state at Mount Sinai concerned things that had already happened, that is, the past consequences of past actions. The relevant mental state for determining guilt is that mental state that

accompanied the actions that constituted the crime. People v. Grever, 353 Ill. App. 3d 736, 757 (2004), rev'd on other grounds, 222 Ill. 2d 321 (2006) ("Criminal liability requires the conjunction of a culpable mental state (at common law, the mens rea) and a punishable act or omission (at common law, the actus reus)"). Thus, defendant's nonchalance at Mount Sinai may have been because he did not believe he did anything sufficient to fulfil his intent to kill Steven. Similarly, his statement to Robert Liebich that "he didn't hit the kid that hard," could be based on his assessment of his actions made at the time he took them or it could be based on his perception of the results of his actions between the time he beat Steven and the time Steven's injuries began to manifest themselves. Thus, the trial court's finding is not inconsistent with a finding of intent.

Lest defendant charge that we are doing nothing more here than engaging in much hypothetical speculation, we point out the following. Defendant is relying on cases about inconsistent verdicts. Such cases typically involve a certain amount of speculation regarding possible bases for the verdicts, and, if there is some plausible basis that does not require the finding of the existence and nonexistence of some element of the crimes, the verdicts are allowed to stand. See People v. Foley, 152 Ill. App. 3d 354, 357 (1987) ("As in Munday, the verdicts could have been based on compromise or an exercise of lenity. In addition, defendant presented some evidence which tended to indicate that the breathalyzer reading was inaccurate. Thus, the jury might have disregarded it entirely, yet still found sufficient other evidence to find defendant guilty of driving under the influence of alcohol" (emphasis added)). In People v. Munday, 134 Ill. App. 3d 971, 976-77 (1985), the court, after considering a number of possible reasons for two seemingly conflicting verdicts, including lenity, compromise, and confusion, held:

"We do not know, and need not decide, which of these possible explanations is correct; the fact remains that defendant has not carried his burden to demonstrate the acquittal was necessarily based on a finding he had no intent to commit rape. As defendant has thus not demonstrated a legal inconsistency of the verdicts, collateral estoppel cannot be applied and his retrial for residential burglary was proper."

The Munday court resolved the issue based entirely on possible scenarios that would explain the apparent conflict between the two verdicts at issue. Thus, we can do the same here. Since, as explained above, the trial court's factual finding based on defendant's demeanor at Mount Sinai is not inconsistent with a finding of intent, case law regarding inconsistent verdicts is of no help to defendant. Furthermore, as the trial court's findings did not preclude convictions based on intent, and because all counts were merged, any complaint regarding the knowledge-based counts are moot. See People v. Hemphill, 230 Ill. App. 3d 453, 468 (1992) ("Next, McIntyre argues that his due process rights were violated because he was not proved guilty of conspiracy of armed robbery beyond a reasonable doubt. McIntyre was found guilty of armed robbery against Jackson. The trial court stated that the conspiracy conviction merged with the armed robbery conviction and only sentenced McIntyre for the armed robbery conviction; thus, this issue is moot").

Accordingly, the trial court's findings regarding defendant's behavior at the hospital do not preclude a finding that defendant intended to kill or do great bodily harm to Steven. Finally, to the extent that evidence regarding defendant's behavior at Mount Sinai weighs in favor of an acquittal, it merely created an evidentiary conflict for the trial court to resolve. People v. Roberts, 374 Ill. App. 3d 490, 497-98 (2007). Substantial evidence militated for a finding of guilt, not the least of which was Kenyatta's testimony that Steven was fine when she left for work, Steven spent the day alone

with defendant, and when Kenyatta returned, Steven was severely injured. None of defendant's arguments persuade us that he was not proven guilty beyond a reasonable doubt.

C. ADMISSIBILITY OF EXPERT OPINION TESTIMONY

Defendant next alleges error in the trial court's admission of the opinion testimony regarding the timing of Steven's injuries from Dr. Munoz and Dr. Severin. Defendant argues that they had no training and possessed no expertise regarding timing. He also contends that their opinions lacked a "scientifically-recognized foundation." The admissibility of an expert opinion is a matter committed to the discretion of the trial court, and a court of review will not interfere with an exercise of that discretion so long as it is not abused. Volpe v. IKO Industries, Ltd., 327 Ill. App. 3d 567, 576 (2002).

Defendant first baldly and incorrectly asserts that, "Since neither Munoz nor Severin had any prior experience with respect to establishing the time of an injury, their opinions on that issue should not have been admitted." This statement is simply false. Munoz testified that he has been involved in "hundreds" of cases of suspected child abuse, and in each case, DCFS personnel have asked him to give an opinion regarding the timing of the injury. Severin, on the other hand, testified that sometimes, authorities, DCFS personnel, other health care workers, or individuals from Child Protective Services (CHP) would sometimes ask him to "correlate their exam with findings of progression of the disease." However, he did acknowledge that he had never been asked for such an estimate in any of the 20 child-abuse cases in which he has been involved. However, to state that neither of these doctors had any experience is pure hyperbole. Whether they had enough experience to render such opinions is a different question. Defendant cites no case law regarding the quantum of experience necessary to render an opinion. In fact, defendant cites no case law whatsoever in

support of this argument, which waives the issue. See People v. Acevedo, 191 Ill. App. 3d 364, 366 (1989).

Defendant next asserts Munoz and Severin "relied on faulty information" in forming their opinions. Defendant points out that Munoz, upon reviewing the CAT scan from Mount Sinai, believed there was a large subdural hematoma in the right side of Steven's head. Munoz did not find such an injury when he subsequently operated upon Steven. Defendant states, "So Munoz's conclusion about the fact that there was a significant subdural hematoma on the right side of the brain proved to be incorrect." We fail to see the relevance of these observations. Munoz presumably did not formulate his ultimate opinion until after he performed surgery. It is a dubious suggestion indeed, and one we find completely unpersuasive, that Munoz relied on his interpretation of the CAT scan rather than what he learned during surgery in coming to the conclusions to which he testified at trial. Cf. Jeffers v. Weinger, 132 Ill. App. 3d 877, 882 (1985) ("Therefore, any possibility that Dr. Fossier based his opinion on an incorrect assumption was nullified when the defendants [subsequently] clarified the situation on cross-examination"). Defendant also argues that Munoz believed that, prior to surgery, Steven was not paralyzed in any way. Dr. Teas's report indicated that records from Mount Sinai show that Steven had been given medicines to paralyze his abdomen. Defendant does not explain how this gap in Munoz's knowledge affected his opinion, or, for that matter, whether he was still unaware of the use of these medications at the time he rendered his opinion. Moreover, our supreme court has held that "the basis for a witness' opinion generally does not affect his standing as an expert; such matters go only to the weight of the evidence, not its sufficiency." Snelson v. Kamm, 204 Ill. 2d 1, 26 (2003). That principle controls here, and it was for the trial court to determine how any such omissions affected the weight to which Munoz's testimony was entitled (People v. Harrison,

366 Ill. App. 3d 210, 219 (2006)). Defendant attacks Severin's opinion on this same basis, and we find the attack no more persuasive here than when it was directed against Munoz.

Defendant cites People v. Wilhoite, 228 Ill. App. 3d 12 (1991), for the proposition that an expert's opinion must be disregarded where it is without proper foundation. Indeed, the exact language from Wilhoite is: "If the expert's opinion is without proper foundation, particularly where he fails to take into consideration an essential factor, that opinion 'is of no weight and must be disregarded.' " (Emphasis added.) Wilhoite, 228 Ill. App. 3d at 21, quoting 32 C.J.S., Evidence, § 569(1) at 609. The Wilhoite court spoke of "an essential factor." Defendant, aside from pointing out the alleged flaws in the bases of Munoz's and Severin's opinions, makes no attempt to show why these purported defects were so critical as to constitute a matter of admissibility rather than weight. In any event, we hold that they were the latter.

Finally, defendant contends that Munoz and Severin offered no scientific bases for their opinions that would comport with the Frye standard. See Frye, 293 F. 1013. Defendant again complains that Munoz based his opinion on the color of the blood in Steven's head. Defendant contends that this is not an accepted scientific methodology. We agree. In fact, as we explained earlier, this was not a scientific methodology at all. It is not derived from the "application of scientific principles" but from Munoz's own skill and experience based upon his observations made in the course of his career. Jackson, 372 Ill. App. 3d at 1073. Moreover, "[s]imply because scientific principles relate to aspects of an opinion witness's testimony does not transform that testimony into 'scientific' testimony." Jackson, 372 Ill. App. 3d at 1073. Defendant also points to a number of alleged defects in Munoz's opinions here, such as the fact that much of Munoz's experience was derived from car accidents rather than child abuse and his failure to consider the effect of Steven's

abdominal injury on his head injury. Such things go to weight, not admissibility. Harrison, 366 Ill. App. 3d at 219.

Regarding Severin, defendant states, "Severin likewise offered no scientifically-based explanation regarding how his estimate of the timing of the bruises on Steven's body comported with his later estimate of the timing of the injuries." The absence of a "scientifically-based explanation" is not a basis for objecting to Severin's opinion. Severin is board certified in pediatric critical care. Based on his training and experience, he was certainly entitled to opine regarding Steven's injuries. Scientific testing may have provided additional support for Severin's opinion, but such additional support was not necessary for the trial court to consider the opinion. Indeed, defendant presented countervailing scientific evidence (the histology slides), and it was for the trial court to resolve the conflict between Severin's testimony and that scientific evidence just as it would have been for the trial court to attribute additional weight to Severin's opinion had it been supported by corroborating scientific evidence.

In sum, Munoz's and Severin's testimony was clearly admissible. The sorts of things of which defendant complains typically are matters of weight. Therefore, we reject his arguments on this point.

D. INEFFECTIVE ASSISTANCE OF COUNSEL

Defendant makes five distinct arguments as to why his counsel was ineffective. First, he points to counsels' failure to interpose a Frye objection to the testimony of Munoz and Severin. Based on our discussion of this issue above, any such objection would have been futile, and we will not consider the issue further. See In re Ottinger, 333 Ill. App. 3d 114, 118 (2002) ("The failure of defendant's counsel to make a futile objection does not constitute fundamentally deficient performance"). Second, defendant complains of his trial attorneys' failure to argue that a letter from

Dr. Teas to the court that was received after the trial had concluded should be considered. Defense counsel actually agreed with the State that the letter should be impounded. Third, defendant asserts that trial counsel should have argued that he was guilty of a lesser-included offense, namely involuntary manslaughter. Fourth, defendant claims that "[d]efense counsel neglected to effectively cross-examine and impeach several witnesses." Defendant's entire argument on this point consists of the following: "The Record [sic] contains multiple instances of defense counsel failing to effectively impeach prosecution witnesses." This statement is followed by a string citation to five places in the record, but contains no reference to legal authority. As has oft been stated, "A reviewing court is entitled to have the issues before it clearly defined and is not simply a repository in which appellants may dump the burden of argument and research; an appellant's failure to properly present his own arguments can amount to waiver of those claims on appeal. People v. Chatman, 357 Ill. App. 3d 695, 703 (2005). We deem that to be the case here, and we will not give this issue further consideration. Fifth, defendant contends that a Rule 604(d) (188 Ill. 2d R. 604(d)) certificate filed by trial counsel contained material inaccuracies.

To succeed on a claim of ineffective assistance of counsel, a defendant must show that his attorney's performance fell below an objective level or reasonableness in light of prevailing professional norms and that this deficient performance prejudiced the defendant. People v. Ramirez, 371 Ill. App. 3d 738, 744 (2007). In order to establish deficient performance, a defendant "must overcome the strong presumption that the challenged action or inaction might have been the product of sound trial strategy." People v. Jackson, 205 Ill. 2d 247, 259 (2001). To show prejudice, a defendant must show a reasonable probability that, but for counsel's errors, the outcome of the proceeding would have been different. Jackson, 205 Ill. 2d at 259. A "reasonable probability" is one

sufficient to undermine confidence in the result of the proceeding. People v. Harris, 206 Ill. 2d 293, 304 (2002). Sometimes, it is easier to dispose of a claim on the prejudice prong of the test, and, in such cases, "counsel's performance need not be evaluated." People v. Brooks, 187 Ill. 2d 91, 137 (1999). With these standards in mind, we will now turn to the balance of defendant's arguments.

1. Dr. Teas's Letter

After the trial had concluded, Teas sent a letter to the trial court. The letter set forth a number of purported exculpatory facts that were supposedly not brought out by defense counsel during the trial. She characterized these facts as "significant findings in the medical records." She sent copies of the letter to the State, defense counsel, and Dr. Mileusnic. Defense counsel agreed with the State that the letter constituted an improper ex parte communication and that it should be impounded in the record.

Defendant now claims trial counsel was ineffective for not seeking to have the letter admitted into evidence. Defendant, citing Black's Law Dictionary, 597 (7th ed. 1999), contends that the letter is not an ex parte communication, since it was sent to all parties. Accepting this assertion as true, defendant does not explain how the letter is otherwise admissible. For example, the statements in the letter appear to be hearsay. See People v. Douglas, 362 Ill. App. 3d 65, 70 (2005) ("Hearsay is an out-of-court statement offered to prove the truth of the matter asserted"). A related problem is that, even though the State received a copy of the letter, it was unable to cross-examine Teas regarding its contents. Moreover, the letter was received well after the trial had concluded. Whether to reopen proofs is a matter committed to the discretion of the trial court. People v. Allen, 344 Ill. App. 3d 949, 953 (2003). Defendant makes no attempt to show that the trial court would have granted such a motion, particularly given the hearsay character of the letter.

To the extent the letter might serve as evidence of ineffectiveness, the relevant question is whether defense counsel's failure to bring out this evidence at trial constituted ineffectiveness. In this regard, defendant has not established that he was prejudiced by these purported failures. We see no reasonable probability that the outcome of the trial would have been different had this evidence been presented. Teas's letter addresses four main points: (1) Teas states that all autopsy findings indicate that Steven's injuries were at least five days old, particularly "[t]he specific characteristics of Steven's tissue reaction take at least several days to develop;" "[t]hey do not occur in 3 or even 4 days;" (2) Steven had aspirin and acetaminophen in his system, which undercuts the notion that he was feeling fine prior to the morning of February 8; (3) Steven lost four pounds between November 6, 2001, and February 8, 2002; (4) Steven was sedated before his abdomen was examined at Rush Presbyterian, which would have initially masked signs of abdominal distress and made the injury appear to be evolving to someone (i.e., Severin) not aware of the sedation.

The first point appears to be a recapitulation of Teas's trial testimony, which was, obviously, presented at trial. The remaining three points were called to the attention of the trial court during posttrial proceedings. Specifically, defendant raised them in his pro se motion alleging ineffective assistance of counsel, and the trial court expressly addressed two of them in its ruling on the motion. The trial court stated that the weight loss was not relevant in that Steven was in the custody of Dorothy Herron for most of the time between November 6, 2001, and February 8, 2002. As for the medication that may have affected observations of Steven's abdomen at Rush Presbyterian, the trial court stated "I think far more critical were the initial observations of the abdomen at Mount Sinai Hospital when Steven was initially brought in." Furthermore, we note that this evidence would have served only to impeach Severin regrading timing, and there was considerable other evidence on this

issue. Most notably, the trial court placed great weight on the testimony and observations of all of the treating medical personnel, including Munoz's opinion. Nurse Smith and Dr. Green both testified that they observed fresh bruises. Dr. Boykin testified to the impossibility of Steven eating a hotdog after sustaining these injuries. They must have occurred, therefore, after Steven last ate. According to defendant's statements to officers Figiel and Szalinski, Steven last ate at about 3 p.m., long after Kenyatta went to work. In fact, defendant told Kenyatta that Steven had been exhibiting signs of injuries for about an hour prior to the time Kenyatta returned from work on February 8. Kenyatta testified that Steven was fine when she left in the morning, but not when she returned after he was left in the exclusive care of defendant. Munoz confirmed that Steven could not walk, talk, eat, or drink after sustaining such injuries and that a head injury of this magnitude would preclude a lucid interval. Steven's amylase and lipase levels doubled following his admission to Mount Sinai. Numerous witnesses testified regarding injuries and bruises that continued to appear throughout the night of February 8. Severin observed that Steven's autoregulation system was still functioning during the evening of February 8. Mileusnic stated that she, as a pathologist, would defer to a treating physician on the issue of timing. Teas is not a treating physician.

In light of all of this evidence, we see no reasonable probability that undermining a portion of the basis for Severin's opinion would have led to a different result at trial. Similarly, that Steven had taken a pain killer at some point (we do not know when) certainly does not support an inference that Steven had sustained these massive injuries at an earlier time than is indicated by the weight of the evidence. As soon as Kenyatta observed the state that Steven was in upon her return from work, she wanted to take him to an emergency room, not give him an over-the-counter pain killer. Furthermore, two doctors testified that, after receiving such injuries, Steven would not have been able

to eat. If he could not eat, he could not swallow aspirin or acetaminophen. Thus, he must have taken these drugs prior to sustaining these injuries, and their presence in his system likely had nothing to do with the injuries that manifested themselves on February 8 at Mount Sinai and Rush Presbyterian. We further note that Kenyatta testified that Steven had a runny nose in the days leading up to his death, which could explain their presence. Again, defendant has not shown a reasonable probability that had this evidence been presented, a different outcome would have followed.

Finally, we note that even if we were to conclude that defendant had satisfied the prejudice prong of the ineffective-assistance-of-counsel test, we are not convinced that any purported error regarding the presentation of the material in Teas's letter would fit within the definition of ineffectiveness contained in the first prong of the test. See Ramirez, 371 Ill. App. 3d at 744. Except in extreme cases, decisions regarding what witnesses to call and what evidence to present are typically matters of trial strategy. People v. Enis, 194 Ill. 2d 361, 378 (2000); People v. Ward, 371 Ill. App. 3d 382, 433 (2007). Matters of trial strategy are "outside the scope of review for purposes of establishing incompetency of counsel." People v. Medrano, 271 Ill. App. 3d 97, 101 (1995).

Accordingly, we hold that trial counsel's failure to present the material in Teas's letter does not constitute ineffective assistance of counsel.

2. Lesser-Included Offense

Defendant next argues that trial counsel was ineffective for failing to argue that he was guilty of the lesser-included offense of involuntary manslaughter (720 ILCS 5/9--3 (West 2002)). Indeed, involuntary manslaughter is a lesser-included offense of first-degree murder with a less culpable mental state. People v. Robinson, 374 Ill. App. 3d 949, ___, 872 N.E.2d 73, 77 (2007). Though this case involved a bench trial, we believe that the best guidance for the resolution of this issue comes

from cases involving the decision to tender a jury instruction on a lesser included offense. Jury instructions place an issue before a jury and ask the jury to resolve it. See Drinkard v. Johnson, 97 F. 3d 751, 761 (5th Cir. 1996) ("The challenged instruction itself asks the jury to consider whether the defendant was temporarily insane (or, more specifically, 'did not know his conduct was wrong') as a result of intoxication 'at the time of the commission of the offense' "). In a bench trial, counsel places an issue before the court by arguing the issue. Insofar as jury instructions are concerned, it is well established that the decision to tender a jury instruction on a lesser-included offense is one of trial strategy. People v. Evans, 369 Ill. App. 3d 366, 383 (2006); People v. McIntosh, 305 Ill. App. 3d 462 471 (1999); People v. Balle, 256 Ill. App. 3d 963 971 (1993) ("Whether to tender an instruction on a lesser-included offense is almost always a question of trial strategy"). Similarly, in this case, counsel's decision not to argue defendant was only guilty of involuntary manslaughter was also a matter of trial strategy. As such, this purported failing by defendant's trial attorneys cannot support a claim of ineffective assistance of counsel. Medrano, 271 Ill. App. 3d at 101.

3. The Rule 604(d) Certificate

Defendant's final contention is that defense counsel was ineffective for filing a certificate pursuant to Rule 604(d) (188 Ill. 2d R. 604(d)) that contained "material misrepresentations." Defendant charges that 70 pages of the transcripts were not included in the record. The "material misrepresentation" defendant refers to is counsel's certification that he had reviewed the record, as set forth in the rule. See 188 Ill. 2d R. 604(d). The chief problem with defendant's argument is that Rule 604(d) does not apply in this case. The rule applies to appeals following a judgment entered on a guilty plea. People v. Willis, 313 Ill. App. 3d 553, 556 (2000). The instant appeal follows a trial. Thus, it is hard to see how defendant could be prejudiced by counsel's noncompliance with Rule

604(d), when he was not entitled to the protections of the rule in the first place. In any event, defendant does not attempt to explain how he was prejudiced despite the fact that the State called his attention to this point in its brief.

IV. CONCLUSION

In light of the foregoing, the judgment of the circuit court of Du Page County is affirmed.

Affirmed.

GROMETER, J., with CALLUM, J., concurring.

O'MALLEY, J., dissenting,

As the majority correctly notes, when a defendant makes a pro se claim of ineffective assistance of trial counsel, a trial court must conduct a threshold inquiry into the defendant's claim to determine if the claim lacks merit or touches upon trial strategy. If so, the claim should be dismissed; and, if not, the trial court should appoint counsel to argue defendant's claim. See slip op. at 44. I disagree with the majority's conclusion that the trial court did not err in its decision not to appoint new counsel to present defendant's claim of ineffective assistance of trial counsel.

First, though I do not dispute the majority's correct citation of the rule that a trial judge's decision not to appoint counsel will be disturbed "only if it is manifestly erroneous," (slip op. at 47, citing Young, 341 Ill. App. 3d at 382), I question the appropriateness of that standard. I trace the "manifestly erroneous" standard to the decision in People v. Jackson, 131 Ill. App. 3d 128, 140 (1985), in which the court, after summarizing the posttrial contention of ineffectiveness raised by the defendant, concluded by saying that the trial court's decision was not "manifestly erroneous." Nowhere in the case law do I find a rationale for applying this standard on appeal. While it is true that a trial court sits in a superior position to answer the above-described threshold questions, I am

not convinced that the trial court's answer is entitled to the high level of deference the "manifest error" rule implies. This type of posttrial motion and a petition under the Postconviction Hearing Act (725 ILCS 5/122--1 et seq. (West 2006)) are quite similar (with the admittedly important difference that the trial judge here, unlike a typical postconviction judge, had recently presided over the trial), and it is well-established that a reviewing court will determine de novo whether a trial court correctly dismissed a postconviction petition without appointing counsel to argue a defendant's claim (eg., People v. Coleman, 183 Ill. 2d 366, 388-89 (1998)). In my view, this similarity warrants more searching review than review for "manifest error."

However, even under the "manifest error" standard, I disagree with the trial court's decision here. After defendant's conviction, Dr. Teas sent a letter detailing what she viewed as relevant exculpatory medical evidence that was not adduced at trial: she wondered why Steven had aspirin in his blood if he had been feeling well; she wondered why Steven had lost four pounds in the four months prior to his death, she noted that some of the drugs doctors administered to Steven may have caused his abdomen to be soft, and she noted that it was not uncommon for symptoms of children's abdominal injuries to be delayed as much as 2-3 days after the injury. To me, defendant's allegation that counsel ignored these facts presents a potentially meritorious claim of ineffective assistance sufficient to withstand the trial court's threshold inquiry. Though counsel generally asserted that any challenged decisions were matters of trial strategy, counsel admitted being unaware of at least one of these medical facts despite Dr. Teas's assertion that it was in the medical records. See slip op. at 42 ("Evidence of aspirin and Tylenol was contained in Teas's ex parte letter, and thus, though he was now aware of it, he could no longer use the evidence"). As for the remaining facts, I believe they raise

enough doubt that defendant should have an opportunity to argue their exclusion was not a matter of reasonable trial strategy.

I would remand this cause for the trial court to appoint counsel to argue defendant's claims.

PETITIONER'S EXHIBIT #3:

Pro se Petition for Post-Conviction Relief