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Attorney for Plaintiffs

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[REDACTED]  
31 W. Liberty Street  
Lancaster, PA 17603

[REDACTED]  
31 W. Liberty Street  
Lancaster, PA 17603

Individually, and as the natural parents  
and next friend of L.S., a minor  
31 W. Liberty Street  
Lancaster, PA 17603

Plaintiffs

v.

Penn State Hershey Medical Center  
500 University Drive  
Hershey, PA 17033

Kathryn R. Crowell, M.D.  
500 University Drive  
Hershey, PA 17033

Andi C. Taroli, M.D.  
500 University Drive  
Hershey, PA 17033

Dorothy V. Rocourt, M.D.  
500 University Drive  
Hershey, PA 17033

Joel M. Weinstein, M.D.  
500 University Drive  
Hershey, PA 17033

Jonas M. Sheehan, M.D.  
500 University Drive  
Hershey, PA 17033

UNITED STATES  
DISTRICT COURT FOR THE  
EASTERN DISTRICT OF  
PENNSYLVANIA

AMENDED COMPLAINT

JURY TRIAL DEMANDED

Mark S. Dias, M.D.  
500 University Drive  
Hershey, PA 17033

Lancaster County  
150 N. Queen Street  
Lancaster, PA 17603

Karen Garber  
900 E. King Street  
Lancaster, PA 17602

Amber Redcay  
900 E. King Street  
Lancaster, PA 17602

Sarah Hasselback  
900 E. King Street  
Lancaster, PA 17602

Susan Murray  
900 E. King Street  
Lancaster, PA 17602

Emily Heugel  
900 E. King Street  
Lancaster, PA 17602

Robin Boyer  
900 E. King Street  
Lancaster, PA 17602

Defendants

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Plaintiffs, [REDACTED] and L.S. place all Defendants on notice of claims as follows:

### **JURISDICTION**

1. This action is brought pursuant to 42 U.S.C. § 1981, 42 U.S.C. § 1983 and 42 U.S.C. § 1985; the First, Fourth, Fifth, Sixth, Seventh and Fourteenth Amendment of the Constitution of the United States; Article 1 of the Pennsylvania Constitution and Pennsylvania law.

2. The jurisdiction of the Court is predicated on 28 U.S.C. § 1343(a), (1), (2), (3) and (4) and 28 U.S.C. § 1331.

### **ALLEGATIONS-PARTIES**

3. Plaintiff, [REDACTED] [REDACTED] at all relevant times to this complaint, was a resident of Lancaster County, Pennsylvania. Mr. [REDACTED] is the father of L.S. who had a rare, unusual birth presentation and a medical condition known as Retino-Dural Hemorrhage of Infancy (RDHI). Mr. [REDACTED] was falsely accused of abusing L.S. when Dr. Andi Taroli of the Penn State Hershey Medical Center's Child Safety Team misdiagnosed L.S.'s RDHI as shaken baby syndrome. Based on Dr. Taroli's misdiagnosis that L.S.'s RDHI was caused by abuse, Mr. [REDACTED] was coerced to "agree" to a safety plan in which he gave up his fundamental liberty interest to the care, custody and control of, and familial association with, his daughter, L.S. under the threat of placement of his daughter in foster care for over eight months without any due process. Furthermore, he was falsely indicated as a perpetrator of child abuse. After an administrative proceeding before the Department of Public Welfare's Bureau of Hearings and Appeals, the administrative indicated report was expunged.

4. Plaintiff, [REDACTED] [REDACTED] at all relevant times to this complaint, was a resident of Lancaster County, Pennsylvania. Ms. [REDACTED] is the mother of L.S. and was also falsely accused of abusing L.S. when L.S.'s RDHI was misdiagnosed as shaken baby. Based on Dr. Taroli's misdiagnosis that L.S.'s RDHI was caused by abuse, Ms. [REDACTED] was also coerced to "agree" to a safety plan in which

she gave up her fundamental liberty interest to the care, custody and control of, and familial association with, her daughter, L.S. under the threat of placement of her daughter in foster care for over eight months without any due process. Furthermore, she was also falsely indicated as a perpetrator of child abuse. After an administrative proceeding before the Department of Public Welfare's Bureau of Hearings and Appeals, the administrative indicated report against Ms. [REDACTED] was also expunged.

5. Plaintiffs [REDACTED] [REDACTED] and [REDACTED] [REDACTED] bring suit as the natural parents and next friend of L.S., a minor. L.S. is the natural child of Mr. [REDACTED] and Ms. [REDACTED] who experienced a rare compound presentation birth and had a medical condition called RDHI. Though there was no indication that L.S. had any bone problems, L.S. was subjected medically unnecessary radiation in the form of skeletal surveys that exposed every bone in L.S.'s body to harmful, medically unnecessary x-rays. Medically unnecessary retinal photo were taken of L.S.'s eyes. L.S.'s parents only consented to medical treatment of L.S. and did not authorize Penn State Hershey Medical Center to conduct medical procedures for the purpose of a child abuse investigation or authorize Penn State Hershey Medical Center doctors to act in bad faith and render opinions about the cause of L.S.'s injuries that dismissed the medical history provided by Mr. [REDACTED] and Ms. [REDACTED]. Mr. [REDACTED] and Ms. [REDACTED] were never notified by the doctors or by Lancaster County employees that the doctors were performing a child abuse investigation or cooperating with and sharing confidential information with Lancaster County employees and law enforcement. Mr. [REDACTED] and Ms. [REDACTED] were never notified that the doctors elected to initiate, and elected to cooperate with, the child abuse investigation and were immunized for breaches of the doctors' fiduciary duty, professional standards and contractual duty of good faith and fair dealing as a result of the doctors' choice to cooperate with the child abuse investigation. Based on this unauthorized child abuse investigation and the unauthorized opinions about the cause of L.S.'s injuries that contradicted the medical history provided, L.S.'s parents, and the failure

to be notified, the Plaintiffs were coerced into a safety plan in which L.S. was deprived of her familial association with her parents and the care, custody and control of her parents for over eight months.

6. Defendant Penn State Milton S. Hershey Medical Center, hereinafter “PSHMC”, is a Pennsylvania non-profit corporation wholly owned by the Pennsylvania State University who operates a hospital, medical school and children’s hospital. Penn State receives Federal and State funding for various activities related to child abuse. For purposes of 42 U.S.C. §1983, Penn State and its employees are state actors. Penn State created a Child Safety Team on September 1, 2009 for the express purpose of, *inter alia*, to actually initiate and conduct an investigation into whether injuries reported as suspicious for child abuse were, in fact, caused by child abuse. Through the establishment of a Child Safety Team, PSHMC instituted a policy of going well beyond the legal mandate to report suspected abuse by encouraging/requiring its doctors to initiate a Child Safety Team investigation and electing to authorize and order medical procedures for the purpose of conducting a child abuse investigation, rather than medical treatment, all of which exceeded the scope of Plaintiffs’ consent to medical treatment. Defendant PSHMC and its employees failed to obtain the informed consent of, or notify, Mr. [REDACTED] and Ms. [REDACTED] that PSHMC was conducting interviews with the Plaintiffs and ordering medical procedures on L.S. for the purpose of investigating child abuse rather than for the purpose of medical treatment. PSHMC and its employees failed to obtain the informed consent of, or notify, Mr. [REDACTED] and Ms. [REDACTED] that, by initiating and cooperating in a child abuse investigation, the PSHMC employees were immunized from civil claims against the PSHMC doctors. PSHMC also had a policy of authorizing unnecessary medical procedures and conducting a child abuse investigation that could result in an indicated report of child abuse and the coercion of a safety plan that infringed on the Plaintiffs’ right to familial association with, and the care custody and control of their daughter, L.S. Defendant PSHMC has a vested interest in reporting and investigating cases of RDHI as child abuse and

perpetuating the hypothesis of shaken baby syndrome in the form of a 2.8 million dollar federal Center for Disease Control grant, and State government funding to educate about shaken baby syndrome.

Defendant PSHMC violated Plaintiffs' rights pursuant to the United States Constitution, the Pennsylvania Constitution and Pennsylvania law.

7. Defendant Kathryn R. Crowell, M.D. at all times relevant to this complaint, was employed by PSHMC as a pediatrician. Defendant Crowell is a state actor for purposes of 42 U.S.C. §1983. Defendant Crowell was a medical provider who had a physician-patient fiduciary and contractual duty to care for the medical needs of L.S. Defendant Crowell failed to notify Plaintiffs that she elected to participate in a child abuse investigation and, as a result, that she was immune for breach of her fiduciary duty by sharing confidential information and immune from civil liability for the breach of her contractual duty of good faith and fair dealing when she failed to believe Plaintiffs' history of L.S.'s illness and ordered medical procedures for the purpose of a child abuse investigation rather than medical treatment. Defendant Crowell has served on the PSHMC Child Safety Team since it was established by PSHMC in September of 2009 and in that capacity issued a consult report on December 2, 2011 concerning L.S. Defendant Crowell's report focused heavily on her interview with the Plaintiffs and L.S.'s medical history provided by Plaintiffs during that interview. It is clear that Defendant Crowell, in bad faith, did not believe the history provided by the Plaintiffs that L.S. had not experienced any inflicted or accidental trauma to her head. Defendant Crowell recommended a skeletal survey and an ophthalmology exam, not for the purposes of medical treatment, but for purposes of a child abuse investigation. Defendant Crowell failed to notify Plaintiffs that Defendant Crowell elected to participate in a child abuse investigation, nor did she disclose that her recommendations were based on the highly controversial hypothesis of shaken baby syndrome, nor did Defendant Crowell notify

Plaintiffs she was immune from civil suit. Defendant Crowell violated the Plaintiffs' rights pursuant to the United States Constitution, the Pennsylvania Constitution and Pennsylvania law.

8. Defendant Joel M. Weinstein, M.D., at all times relevant to this complaint, was employed by Penn State University Hershey Medical Center as an ophthalmologist. Defendant Weinstein is a state actor for purposes of 42 U.S.C. §1983. Defendant Weinstein was a medical provider who owed Plaintiffs a fiduciary and contractual duty to care for the medical needs of L.S. Defendant Weinstein failed to notify Plaintiffs that he had elected to participate in a child abuse investigation and that, as a result, he was immune to civil suit for breaching his fiduciary duty and breaching his contractual duty of good faith and fair dealing by failing to believe Plaintiffs' history of L.S.'s illness and ordering digital retinal photos for the purpose of a child abuse investigation rather than medical treatment. Defendant Weinstein exceeded the scope of consent provided by the Plaintiffs to provide medical treatment and in a grossly negligent manner, misrepresented that retinal hemorrhages and macular retinoschisis are "highly suggestive of repetitive shaking" and failed to disclose that his opinion was based on the highly controversial vitreoretinal traction hypothesis. Defendant Weinstein violated Plaintiffs' rights pursuant to the United States Constitution, the Pennsylvania Constitution and Pennsylvania law.

9. Defendant Andi C. Taroli, M.D. at all times relevant to this complaint, was employed by Penn State University Hershey Medical Center as a pediatrician and Director of PSHMC's child safety team. Defendant Taroli is a state actor for purposes of 42 U.S.C. §1983. Defendant Taroli was a medical provider who had a fiduciary and contractual duty to care for the medical needs of L.S. Defendant Taroli failed to notify Plaintiffs that she elected to perform a child abuse investigation and was sharing confidential information with government employees. Defendant Taroli failed to notify Plaintiffs that she was immune from civil liability for her breach of fiduciary duty for disclosing

confidential information to government employees, immune from civil liability for breach of her contractual duty of good faith and fair dealing by failing to believe Plaintiffs' history of L.S.'s illness and immune from professional liability for rendering a grossly negligent report and opinion. Defendant Taroli fabricated evidence in her report that retinoschisis is "tearing of the retina off its attachment", fabricated evidence in her report that L.S.'s "head circumference grew steadily along the 75<sup>th</sup> %ile until 1 month of age", during her investigation, Defendant Taroli presumed that L.S. was abused because she had RDHI, and she failed to disclose that the basis of her opinion, the shaken baby syndrome, was "simply [a] hypotheses, not proven medical or scientific facts". Defendant Taroli's actions violate the Plaintiffs' right pursuant to the United States Constitution, the Pennsylvania Constitution and Pennsylvania law.

10. Defendant Dorothy V. Rocourt, M.D., at all times relevant to this complaint, was employed by Penn State University Hershey Medical Center as a pediatric surgeon. Defendant Rocourt is a state actor for purposes of 42 U.S.C. §1983. Defendant Rocourt was a medical provider who owed Plaintiffs a fiduciary and contractual duty to care for the medical needs of L.S. Defendant Rocourt failed to notify Plaintiffs that she had elected to participate in a child abuse investigation and that she was immunized from civil liability for breaching her fiduciary duty and contractual duty of good faith and fair dealing when she failed to believe Plaintiffs history of L.S.'s illness and recommended the initiation of a PSHMC child safety team investigation into suspected abuse, an investigation that was not mandated by law, was not medically necessary and was not related to the treatment of L.S.'s RDHI. Defendant Rocourt's actions violate the Plaintiffs' rights pursuant to the United States Constitution, the Pennsylvania Constitution and Pennsylvania law.

11. Defendant Jonas M. Sheehan, M.D., at all times relevant to this complaint, was employed by Penn State University Hershey Medical Center as a neurosurgeon. Defendant Sheehan is a

state actor for purposes of 42 U.S.C. §1983. Defendant Sheehan was a medical provider who owed the Plaintiffs a fiduciary and contractual duty to care for the medical needs of L.S. Defendant Sheehan failed to notify the Plaintiffs that he had elected to participate in a child abuse investigation and that he was immune from civil liability for his breach of fiduciary duty and contractual duty of good faith and fair dealing when he failed to believe Plaintiffs history of L.S.'s illness and recommended the initiation of a child safety team investigation into allegations that L.S. was abused, an investigation that was not mandated by law, was not medically necessary and was not related to the treatment of L.S.'s RDHI. Defendant Sheehan violated the Plaintiffs' rights pursuant to the United States Constitution, the Pennsylvania Constitution and Pennsylvania law.

12. Defendant Mark S. Dias, M.D., at all times relevant to this complaint, was employed by Penn State University Hershey Medical Center as a neurosurgeon. Defendant Dias is a state actor for purposes of 42 U.S.C. §1983. Defendant Dias was a medical provider who owed the Plaintiffs a fiduciary and contractual duty to care for the medical needs of L.S. Defendant Dias failed to notify the Plaintiffs that he had elected to participate in a child abuse investigation and that he was immune from civil liability for his breach of fiduciary duty and contractual duty of good faith and fair dealing when he referred Plaintiffs to the "services" of Lancaster County Children and Youth Services case worker Karen Garber and ordered a follow-up skeletal survey of L.S. in his discharge instructions. Services with Lancaster County Children and Youth Services and a follow-up skeletal survey were not medically necessary and not related to the treatment of L.S.'s RDHI. Defendant Dias' discharge instructions were faxed to Defendant Garber on December 13, 2011, the same day L.S. was discharged from PSHMC. Defendant Dias violated the Plaintiffs' rights pursuant to the United States Constitution, the Pennsylvania Constitution and Pennsylvania law.

13. Defendant Lancaster County is a county of the 3rd class political subdivision of the Commonwealth of Pennsylvania governed by a board of three commissioners elected to four year terms, Commissioner chairman Scott Martin, Commissioner vice chairman Dennis Stuckey and Commissioner Craig Lehman. Defendant Lancaster County is licensed by the Pennsylvania Department of Public Welfare to operate a county child protective services agency. Defendant Lancaster County had a policy of coercing parents into “agreeing” to safety plans without affording parents due process. Defendant Lancaster County had a policy of failing to train employees, and failing require employees, to notify parents that physicians who participated in child abuse investigations with Lancaster County employees were immune from civil suit for breaches of the fiduciary duty of confidentiality and breaches of the contractual duty of good faith and fair dealing. Defendant Lancaster County violated the Plaintiffs’ rights pursuant to the United States Constitution, the Pennsylvania Constitution and Pennsylvania law.

14. Defendant Karen Garber, at all times relevant to this action, was employed by Defendant Lancaster County in the Lancaster County Children and Youth Services Agency as an intake case worker. Defendant Garber threatened to place L.S. in foster care unless Mr. [REDACTED] and Ms. [REDACTED] “agreed” to a safety plan that required L.S. to live with another family member and required Plaintiffs to have only limited supervised contact. After coercing “agreement” to the safety plan, Defendant Garber failed to afford Mr. [REDACTED] and Ms. [REDACTED] due process of law. Defendant Garber failed to notify Plaintiffs that physicians who participated in the child abuse investigation and shared confidential information with her were immune from civil suit for breaches of the fiduciary duty of confidentiality and breaches of the contractual duty of good faith and fair dealing. Defendant Garber violated the Plaintiffs’ rights pursuant to the United States Constitution, the Pennsylvania Constitution and Pennsylvania law.

15. Defendant Amber Redcay, at all times relevant to this action, was employed by Defendant Lancaster County in the Lancaster County Children and Youth Services Agency as an intake supervisor. Defendant Redcay approved the safety plan in which Defendant Garber threatened to place L.S. in foster care unless Mr. [REDACTED] and Ms. [REDACTED] “agreed” to a safety plan that required L.S. to live with another family member and that Plaintiffs could only have limited supervised contact. After approving the coerced safety plan, Defendant Redcay failed to afford Mr. [REDACTED] and Ms. [REDACTED] due process of law. Defendant Redcay failed to notify Plaintiffs that physicians who participated in the child abuse investigation and shared confidential information with her were immune from civil suit for breaches of the fiduciary duty of confidentiality and breaches of the contractual duty of good faith and fair dealing. Defendant Redcay violated the Plaintiffs’ rights pursuant to the United States Constitution, the Pennsylvania Constitution and Pennsylvania law.

16. Defendant Sarah Hasselback, at all times relevant to this action, was employed by Defendant Lancaster County in the Lancaster County Children and Youth Services Agency as an intake case worker. Defendant Hasselback threatened to place L.S. in foster care unless Mr. [REDACTED] and Ms. [REDACTED] “agreed” to a safety plan that required L.S. lived with another family member and that required Plaintiffs to have only limited supervised contact. After coercing “agreement” to the safety plan, Defendant Hasselback failed to afford Mr. [REDACTED] and Ms. [REDACTED] due process of law.

17. Defendant Susan Murray, at all times relevant to this action, was employed by Defendant Lancaster County in the Lancaster County Children and Youth Services Agency as an intake supervisor. Defendant Murray approved the safety plan(s) in which Defendant Lancaster County case workers threatened to place L.S. in foster care unless Mr. [REDACTED] and Ms. [REDACTED] “agreed” to a safety plan that required L.S. to live with another family member and that Plaintiffs could have only

limited supervised contact. After approving the coerced safety plan, Defendant Murray failed to afford Mr. [REDACTED] and Ms. [REDACTED] due process of law.

18. Defendant Emily Heugel, at all times relevant to this action, was employed by Defendant Lancaster County in the Lancaster County Children and Youth Services Agency as an intake supervisor. Defendant Heugel approved the safety plan(s) in which Defendant Lancaster County case workers threatened to place L.S. in foster care unless Mr. [REDACTED] and Ms. [REDACTED] “agreed” to a safety plan where L.S. lived with another family member and that Plaintiffs could only have limited supervised contact. After approving the coerced safety plan, Defendant Heugel failed to afford Mr. [REDACTED] and Ms. [REDACTED] due process of law.

19. Defendant Robin Boyer, at all times relevant to this action, was employed by Defendant Lancaster County in the Lancaster County Children and Youth Services Agency as the director of the intake department. Defendant Boyer approved the safety plans that in which Defendant Lancaster County case workers threatened to place L.S. in foster care unless Mr. [REDACTED] and Ms. [REDACTED] “agreed” to a safety plan that required L.S. to live with another family member and that Plaintiffs could have only limited supervised contact. After approving the coerced safety plan, Defendant Boyer failed to afford Mr. [REDACTED] and Ms. [REDACTED] due process of law.

**ALLEGATIONS - FACTUAL**

20. Plaintiff, [REDACTED] [REDACTED] gave birth to L.S. in September of 2011. During delivery, Ms. [REDACTED] was administered oxytocin to augment contractions.

21. In her medical records, a rare complication of L.S.’s birth was noted as “compound presentation R hand @ neck”.

22. A further “skin condition intact annotation:” in L.S.’s medical records noted, “peeling skin noted on R wrist, infant was a compound R hand presentation delivery”.

23. Compound birth presentations are considered rare complications of birth, occurring in approximately 1 in 2,000 births, and the administration of contraction strength augmenting medication such as oxytocin is contraindicated with such rare compound presentations.

24. Ms. [REDACTED] was administered oxytocin during her labor with L.S.

25. Neither Mr. [REDACTED] nor Ms. [REDACTED] were aware of L.S.'s compound right hand at neck presentation or that oxytocin was contraindicated for a compound presentation.

26. On September 26, 2011, Plaintiffs took L.S. to the pediatrician for a newborn weight check checkup. No evidence of abuse was observed and L.S.'s head circumference was measured as 35.6 cm. According to the United States' Center for Disease Control's (hereinafter "CDC") head circumference chart and her pediatrician, L.S.'s 35.6 cm. head circumference placed her on the 90<sup>th</sup> to 95<sup>th</sup> percentile compared to other children her age. See Exhibit 1. The pediatrician did not examine L.S.'s eyes for the presence of retinal hemorrhages.

27. On October 3, 2011, Plaintiffs took L.S. to the pediatrician for a newborn check. No evidence of abuse was observed and L.S.'s head circumference was not measured. The pediatrician did not examine L.S.'s eyes for the presence of retinal hemorrhages.

28. On October 25, 2011, Plaintiffs took L.S. to the pediatrician for her one-month well check up. No evidence of abuse was observed and L.S.'s head circumference was measured as 38.5 cm. According to the CDC head circumference chart and her pediatrician, L.S.'s 38.5 cm. head circumference placed her on the 98<sup>th</sup> percentile compared to other children her age. See Exhibit 1. The pediatrician did not examine L.S.'s eyes for the presence of retinal hemorrhages.

29. On November 29, 2011, Plaintiffs took L.S. to the pediatrician for a sick visit. L.S. was noted to be "sneezing, not eating, vomiting". The pediatrician diagnosed L.S. with "overfeeding" and "counseled the parents about feeding". No evidence of abuse was observed and

L.S.'s head circumference was not measured. The pediatrician did not examine L.S.'s eyes for the presence of retinal hemorrhages.

30. On December 2, 2011, Plaintiffs took L.S. to the pediatrician for a well check 2-month visit. L.S. was noted to have "multiple spit ups & vomiting, staring into space and less interactive". No evidence of abuse was observed, "no bruises or rash" was noted and L.S.'s head circumference was measured as 43.4 cm. According to the CDC head circumference chart and her pediatrician, L.S.'s 43.4 cm. head circumference placed her well above the 98<sup>th</sup> percentile compared to other children her age. See Exhibit 1. L.S.'s eye's were noted to be "Bilateral pupils equal & reactive to lite[sic]", L.S.'s anterior fontanel was noted to be "Soft open non-tense slightly bulging +".

31. L.S.'s pediatrician testified that she was not aware that L.S. had a compound right hand presentation or peeling skin on the right wrist at birth.

32. Upon the advice of the pediatrician, on December 2, 2011, Ms. [REDACTED] immediately took L.S. to Ephrata Community Hospital where a stat CT scan reported at 2:43 p.m., "Bilateral extra axial fluid collections that are not the same density as CSF suggesting chronic subdural hematomas or hygromas with a somewhat unusual appearance. The more normal CSF density is noted medial to these rather than layering of fluid more typically seen." No skull fracture was observed or noted.

33. Two chest x-rays performed at Ephrata Community Hospital revealed no skeletal abnormalities or fractures.

34. On December 2, 2013, Defendant Garber threatened to place L.S. into foster care if Mr. [REDACTED] and Ms. [REDACTED] did not "agree" to an "Immediate Preliminary Safety Plan" that provided a "safety action" that, "neither parent, Alicia [sic] or [REDACTED] will have unsupervised contact with [L.S.]".

35. The “Immediate Preliminary Safety Plan” identified the safety threat as “AP” meaning that on December 2, 2011 Mr. [REDACTED] and Ms. [REDACTED] were deemed alleged perpetrators (A.P.) by Defendant Garber.

36. No due process was ever afforded to Plaintiffs regarding the December 2, 2013 safety plan.

37. On December 2, 2011, L.S. was transferred to PSHMC.

38. On December 2, 2011, Defendant Garber consulted with a Manheim Township police detective and requested that Mr. [REDACTED] submit to an interview with the detective and Defendant Garber at PSHMC. Mr. [REDACTED] declined to be interviewed by Defendant Garber and the detective.

39. At 9:07 p.m., pursuant to PSHMC policy, Defendant Sheehan, issued a neurosurgery inpatient consult report with Recommendations of “agree with NAI [non-accidental injury] w/u [work up]” unrelated to L.S.’s presenting symptoms or the medical history provided in breach of his fiduciary and contractual duty to act in good faith and fair dealing with Mr. [REDACTED] and Ms. [REDACTED]

40. Defendant Sheehan failed to notify Mr. [REDACTED] and Ms. [REDACTED] that he had elected to participate in the child abuse investigation and, as a result, he was immune from civil liability for breaching his fiduciary duty to keep information confidential and was immune from civil liability for breaching his contractual duty of good faith and fair dealing, was immune from liability for his negligence for failing to review birth records or to recommend that birth records be obtained and reviewed and was immune from liability for exceeding the scope of Plaintiffs’ contractual consent to medical treatment of L.S.

41. At 9:43 p.m., pursuant to PSHMC policy, Defendant Rocourt, issued a pediatric surgery inpatient consult report with Recommendations of a “Child Safety consult” and “would recommend skeletal survey” unrelated to L.S.’s presenting symptoms or the medical history provided in breach of her fiduciary and contractual duty to act in good faith and fair dealing with Mr. [REDACTED] and Ms. [REDACTED].

42. Defendant Rocourt failed to notify Mr. [REDACTED] and Ms. [REDACTED] that she had elected to participate in the child abuse investigation and, as a result, she was immune from civil liability for breaching her fiduciary duty to keep information confidential and was immune from civil liability for breaching her contractual duty of good faith and fair dealing, was immune from liability for her negligence for failing to review birth records or to recommend that birth records be obtained and reviewed and was immune from liability for exceeding the scope of Plaintiffs’ contractual consent to medical treatment of L.S.

43. Sometime before 10:31 p.m. on December 2, 2011, pursuant to PSHMC policy, Defendant Crowell interviewed Mr. [REDACTED] and Ms. [REDACTED] as part of her child safety team consultation.

44. After interviewing Mr. [REDACTED] and Ms. [REDACTED] on December 2, 2011, at 10:31 p.m., Defendant Crowell issued her child safety team inpatient consult “Recommendations” that included a medically unnecessary “Skeletal survey to screen for additional bony trauma”.

45. The chest x-rays at Ephrata Community Hospital were negative for any fractures and the history provided by Mr. [REDACTED] and Ms. [REDACTED] and L.S.’s presenting symptoms did not include any evidence of “trauma” or “bony trauma” and a skeletal survey was not medically warranted. The skeletal survey was strictly related to the PSHMC Safety Team child abuse investigation. Mr. [REDACTED] and Ms. [REDACTED] did not consent to any child abuse investigation.

46. Defendant Crowell failed to notify the Plaintiffs that her interview with them and recommendations were for the purpose of a child abuse investigation rather than medical treatment. Defendant Crowell's interview with Mr. [REDACTED] and Ms. [REDACTED] in which she selectively believed the history provided of L.S.'s presenting symptoms while not accepting representations that L.S. had not been abused, breached Defendant Crowell's contractual duty to act in good faith and fair dealing with Plaintiffs L.S., Mr. [REDACTED] and Ms. [REDACTED]

47. Defendant Crowell negligently failed to review, or recommend a review, of L.S.'s birth records and as a result was unaware of the fact that L.S. had a right hand compound birth presentation or that Ms. [REDACTED] was administered oxytocin during labor prior to writing her child safety team inpatient consult report. Defendant Crowell's failure to review, and failure to recommend review of, L.S.'s birth records is gross negligence.

48. Defendant Crowell reported on December 2, 2011 and December 4, 2011 that "CYS and law enforcement [were] appropriately involved".

49. Defendant Crowell failed to notify Mr. [REDACTED] and Ms. [REDACTED] that she had elected to participate in the child abuse investigation and, as a result, she was immune from civil liability for breaching her fiduciary duty to keep information confidential and was immune from civil liability for breaching her contractual duty of good faith and fair dealing, immune from liability for her gross negligence for failing to review or recommend that birth records be obtained and reviewed and immune from liability for exceeding the scope of Plaintiffs' contractual consent to medical treatment of L.S.

50. Defendant PSHMC has a policy and/or practice that, in any case of suspected shaken baby syndrome/abusive head trauma, retinal photos are taken in anticipation of prosecution.

51. On December 3, 2011, at 1:03 p.m. Defendant Weinstein reported "Bilateral diffuse retinal hemorrhages in all layers of the retina + some strands of hemorrhage in the vitreous

bilaterally – There are bilateral hemorrhagic retino-schisis cavities. ... This constellation of findings is highly suggestive of repetitive shaking injury and would be extremely rare in any other setting.”

52. Defendant Weinstein fails to report that his assertion is highly controversial and that orthodox mainstream ophthalmological and medical opinion is that retinal hemorrhage and retinoschisis are related to subdural hemorrhage, increased intracranial pressure and other factors rather than to shaking.

53. Defendant Weinstein failed to notify Mr. [REDACTED] and Ms. [REDACTED] that he had elected to participate in the child abuse investigation and, as a result, he was immune from civil liability for breaching his fiduciary duty to keep information confidential and was immune from civil liability for breaching his contractual duty of good faith and fair dealing, immune from liability for his negligent report and immune from liability for exceeding the scope of Plaintiffs’ contractual consent to treat L.S.

54. A further ophthalmology note by the ophthalmology resident acting under the direction of Defendant Weinstein states “Please call ASAP prior to MRI scan so can take retinal photos while under sedation.”

55. Taking retinal photos is not for the purpose of treatment and is purely for purposes of the child abuse investigation and subsequent prosecution.

56. Mr. [REDACTED] and Ms. [REDACTED] only consented to medically necessary treatment and never consented to retinal photos for child abuse investigation and prosecution purposes.

57. December 2, 2011, Defendant Garber consulted with a Manheim Township police detective and requested that Mr. [REDACTED] submit to an interview with the detective and Defendant Garber at PSHMC. Mr. [REDACTED] declined to be interviewed by Defendant Garber and the detective.

58. Defendant PSHMC has a policy and/or practice that in any case of RDHI, PSHMC doctors are expected to order a full skeletal survey.

59. On December 3, 2011, at 2:56 p.m. a medically unnecessary skeletal survey was performed on L.S. exposing her to 17 potentially harmful doses of radiation. The skeletal survey found “no evidence for acute or healing fracture”.

60. On December 3, 2011, at 3:18 p.m. an MRI performed on L.S. reported “unremarkable MRI of the cervical spine” and “bilateral subdural hematoma”.

61. On December 3, 2011, Defendant Taroli was consulted as the Director of the PSHMC Child Safety Team to investigate whether L.S. was abused because PSHMC medical providers dismissed the history provided by Mr. [REDACTED] and Ms. [REDACTED] as false.

62. Defendant Taroli “admitted that, upon a finding of an intracranial injury of a child less than a year of age, she presumes the child was abused. She also admitted doing so in this specific case[L.S.]”

63. Defendant Taroli reported “Pt exhibits evidence of high velocity forces acting upon the brain inside the skull, causing subdural hemorrhages, retinal hemorrhages and retinoschisis (tearing of the retina off its attachment). These injuries only occur with large magnitude forces, typically seen in unrestrained motor vehicle accidents, and also in the context of violent shaking of infants... The child’s head circumference grew steadily along the 75<sup>th</sup> %ile until 1 month of age. When she was admitted at 2 months of age her head was notably macrocephalic and the circumference had increased to > 97<sup>th</sup>%ile. ... the eye injuries must be considered to be abusive in nature, the result of shaking.”

64. Defendant Taroli reported that Ms. [REDACTED] “denied shaking of the baby for any reason” by her or Mr. [REDACTED]

65. Defendant Taroli failed to report or acknowledge that, according to Dr. Norman Guthkelch, the pediatric neurosurgeon often credited with first advancing the hypotheses, the shaken

baby syndrome hypothesis and its associated shaken eye hypothesis are “simply hypotheses, not proven medical or scientific facts” and that it is not reasonable to “infer shaking (or any other form of child abuse) from a finding of retino-dural hemorrhage of infancy”.

66. Defendant Taroli deliberately misrepresented and exaggerated retinoschisis as “tearing of the retina off its attachment” to bolster her misdiagnosis of shaken baby syndrome. Defendant Taroli knew, or should have known, that retinoschisis is a small collection of blood in between the layers of the retina, not a retinal detachment.

67. Defendant Taroli deliberately misrepresented and exaggerated L.S.’s head circumference as growing “steadily along the 75<sup>th</sup> %ile until 1 month of age” when L.S.’s pediatrician documented head circumference measurements as 90<sup>th</sup> to 95<sup>th</sup> percentile at birth, 98<sup>th</sup> percentile at 1 month and well above the 98<sup>th</sup> percentile at 3 months to bolster her misdiagnosis of shaken baby syndrome. See Exhibit 1.

68. Defendant Taroli failed to review birth records resulting in Defendant Taroli’s complete ignorance of the documented fact that L.S. had a right hand compound birth presentation and that Ms. [REDACTED] was administered oxytocin during labor to augment the strength of her contractions.

69. Defendant Taroli further reported, “The initial skeletal survey did not reveal any fractures related to abuse. A followup skeletal survey needs to be obtained in 2 weeks...” recommending that L.S. be further subjected to 17 more medically unnecessary and potentially harmful doses of radiation.

70. Defendant Taroli failed to notify Mr. [REDACTED] and Ms. [REDACTED] that she had elected to participate in the child abuse investigation and, as a result, she was immune from civil liability for breaching her fiduciary duty to keep information confidential, immune from civil liability for breaching her contractual duty of good faith and fair dealing, immune for her grossly negligent



76. The new areas of subdural bleeding, or rebleeding, (red arrow) and depressed fontanel demonstrated on L.S.'s December 11, 2011 head CT confirmed the chronic nature of L.S.'s subdural collection meaning her subdural collection was likely related to her compound right hand birth presentation and oxytocin administration to A.T. during labor. See Exhibit 2.

77. The significant overlapping of L.S.'s sutures demonstrated the chronic nature of L.S.'s subdural collections in that such bone growth shown by the overlapping sutures could not have grown just within the 10 days L.S. exhibited symptoms of vomiting and poor feeding supporting the chronic nature (weeks to months old) of L.S.'s subdural collection. See Exhibit 2.

78. L.S.'s subdural drains were removed on December 12, 2011 and she was discharged by Defendant Dias from PSHMC at 1:46 p.m. on December 13, 2011.

79. Defendant Dias' December 13, 2011 discharge instructions included an instruction "You are scheduled for a follow-up skeletal survey on December 14, 2011."

80. Defendant Dias' December 13, 2011 discharge instructions included an instruction for the "Services" from Defendant Lancaster County Children and Youth Services and Defendant Garber.

81. Without any Court Order, and because of the safety plan "agreed" to by Plaintiffs, Plaintiffs could not take L.S. to their home when L.S. was discharged from PSHMC.

82. Defendant Dias' December 13, 2011 discharge instructions to Plaintiffs were printed at 3:56 p.m. and faxed to Defendant Garber on December 13, 2011 at 4:25 p.m.

83. On December 14, 2011, a second medically unnecessary skeletal survey was performed on L.S. exposing her to 17 potentially harmful doses of radiation. The skeletal survey found no fractures.

84. Upon information and belief, some or all of Defendants Garber, Redcay, Boyer, Hasselback, Murray, Huegel, Taroli, Crowell, Weinstein and Dias participated in a Child Safety Team meeting, a multi-disciplinary team meeting and/or a near death review board meeting regarding the allegations that L.S. had been abused within 30 to 60 days of December 2, 2011.

85. On March 5, 2012, Plaintiffs were notified by the Pennsylvania Department of Public Welfare that Defendants Garber and Redcay had finished their investigation and filed an administrative “indicated” report listing them as perpetrators of child abuse on ChildLine citing “[L.S.] was found to have subdural hemorrhages, retinal hemorrhages and retinoschisis. ...Neither parent could provide an explanation for the injuries. There are no known medical conditions that would have caused these injuries.”

86. The March 5, 2012 notice from the Department of Public Welfare provided Plaintiffs with due process procedural information about how to request administrative review and expungement of the “indicated” report.

87. On March 21, 2012, Plaintiffs requested a FAIR Hearing to require Defendant Lancaster County to provide evidence concerning why the administrative “indicated” report with ChildLine should not be expunged.

88. On or about April 19, 2012, Defendant Hasselback stated, “the safety plan could be lifted as soon as both parents successfully completed their Family Service Plan. To date, the parenting portion of the plan has not been completed. Therefore, the safety plan will need to be extended. ... When Ms. [REDACTED] and Mr. [REDACTED] successfully complete the COBYS parenting program and provide the Agency with a copy of their certificates, the Agency will lift the safety plan and [L.S.] can return home to her parents.”

89. Under the threat of placement of L.S. in foster care, Plaintiffs “agreed” to extend the safety plan. Defendant Murray approved the extension of the safety plan.

90. Pursuant to Defendant Lancaster County policy, neither Defendant Hasselback nor Defendant Murray provided Plaintiffs with any due process to challenge the extension of the safety plan.

91. On or about June 16, 2012, Defendant Hasselback forwarded an “extended safety plan” to Plaintiffs that continued to require the same restrictions on Plaintiffs contact with L.S.

92. Under the threat that L.S. would be placed in foster care, Plaintiffs “agreed” to the “extended safety plan”. Defendant Huegel approved the “extended safety plan” forwarded by Defendant Hasselback.

93. Pursuant to Defendant Lancaster County policy, neither Defendant Hasselback nor Defendant Heugel provided Plaintiffs with any due process to challenge the extension of the safety plan.

94. On July 12, 2012, L.S. was permitted to return home and live with her parents subject to scheduled and unscheduled home inspection visits from Defendant Hasselback or other employees of Defendant Lancaster County without probable cause.

95. On or about September 12, 2012, the safety plan was “lifted” and the Plaintiffs were no longer subject to home visits by Lancaster County employees.

96. Though the police investigated the allegations that L.S. was abused, no criminal charges were ever filed.

97. No dependency petition was ever filed.

98. For over 9 months, from the date Defendants coerced the first safety plan on December 2, 2011, until on or about September 12, 2012, when the safety plan was “lifted”, Defendants

Garber, Redcay, Hasselback, Murray, Boyer, Huegel and Lancaster County failed to provide Plaintiffs with any due process regarding any safety plan or any forum in which Plaintiffs could defend the false allegations of abuse giving rise to the safety plan.

99. Upon Information and belief, Defendant Lancaster County is a member of the County Commissioners Association of Pennsylvania.

100. The Pennsylvania Children and Youth Administrators Association is an Affiliate of the County Commissioners Association of Pennsylvania.

101. Upon information and belief, Defendant Lancaster County is a member of the Children and Youth Administrators Association. “The Pennsylvania Children and Youth Administrators Association (PCYA) is a 501(c) (4) nonprofit corporation incorporated in 1969. The Association represents all sixty-seven county children and youth agencies in activities with other organizations and government officials and facilitates on-going networking and information sharing among its membership.”

102. The Children and Youth Administrators Association has delegated its responsibility to train its employees, supervisors and administrators to the University of Pittsburgh’s Pennsylvania Child Welfare Training Program. “The Pennsylvania Child Welfare Training Program (Training Program) is a collaborative effort of the University of Pittsburgh, School of Social Work, the Pennsylvania Department of Public Welfare, and the Pennsylvania Children and Youth Administrators. It was established to train direct service workers, supervisors, administrators, and foster parents in providing social services to abused and neglected children and their families. The Training Program is centrally managed and regionally administered by the University of Pittsburgh, School of Social Work.”

103. Defendant Lancaster County delegated its duty to train its case workers, supervisors and administrators to the University of Pittsburgh's Pennsylvania Child Welfare Training Program.

104. Defendant Lancaster County's training of its direct service workers, supervisors and administrators is devoid of training that, when parents are coerced into "agreement" with a safety plan that impairs the parents' fundamental right to the care, custody and control of their child, due process considerations are triggered.

105. The ordinary physician-doctor relationship is one in which a fiduciary duty arises that includes the duty of the doctor to maintain the confidentiality of the information the doctor learns during the course of treating the patient. The relationship includes a contractual duty to act in good faith and fair dealing with the patient, or in the case of treatment of a minor, the patient's legal guardian. The patient can ordinarily vindicate any doctor's breach of a duty of care through a civil lawsuit in the Courts.

106. Under the threat of criminal prosecution for failing to do so, Pennsylvania has mandated that medical professionals "shall report or cause a report ... when the person has reasonable cause to suspect, ... that a child under the care, ...of that person ... is a victim of child abuse". Pennsylvania further mandates that specific information about the suspected victim and perpetrator must be reported, if known. Pennsylvania has granted immunity those who, in good faith, make reports of suspected child abuse.

107. In addition, Pennsylvania law provides doctor treating a patient about whom a report of suspected abuse with two options. The first option is that the doctor can choose to uphold her fiduciary duty to maintain confidentiality and contractual duty to restrict her actions strictly to treatment of the child. The second option, is that the doctor can elect to exceed the scope of the contract to treat

the child and breach their fiduciary duty of confidentiality because, if the doctor elects to do so, the government has granted the doctor immunity from civil and criminal prosecution for such actions.

108. The Plaintiffs were never notified that the ordinary doctor-physician relationship, a relationship that includes a fiduciary duty of confidentiality, a contractual duty of good faith and fair dealing and a constitutional right to pursue breaches of any standard of care in court, between Plaintiffs and Defendants Crowell, Weinstein, Taroli, Rocourt, Sheehan and Dias had been substantially impaired by the government's grant of immunity to these Defendants' because they elected to participate in a child abuse investigation.

109. Defendant Lancaster County's training of its direct service workers, supervisors and administrators is devoid of training that, when doctors elect to participate in a child abuse investigation, that the government grants those doctors immunity from civil suit thus depriving and/or impairing the parents of their liberty interest in their right to a jury trial in a civil lawsuit against the participating doctors.

110. On January 14, 2013, over one year after the allegations of abuse were first made, and five months after the safety plan was "lifted", the Pennsylvania Department Of Public Welfare Bureau of Hearings and Appeals conducted an administrative hearing about whether the "indicated" ChildLine abuse registry report against [REDACTED] [REDACTED] and [REDACTED] [REDACTED] should be expunged.

111. On January 14, 2013, Defendant Taroli testified that she presumed abuse to be the cause of L.S.'s subdural collections and retinal hemorrhages, that L.S.'s retinas were detached, that L.S.'s head circumference tracked along the 75<sup>th</sup> percentile curve until December 2, 2011 and that the hypothesis of shaken baby syndrome was not controversial.

112. On July 16, 2013, the Bureau of Hearings and Appeals attorney examiner, David Dudley, found Defendant Taroli “admitted that, upon a finding of an intracranial injury of a child less than a year of age, she presumes the child was abused. She also admitted doing so in this specific case.”

113. Mr. Dudley also made an administrative finding that to accept Defendant Taroli’s testimony he “would have to believe [Plaintiffs] abused the child around November 20, 2011, nine days later the child was completely fine, and three days after that had a complete recurrence of symptoms”.

114. Mr. Dudley stated, “A reasonable person would not accept such evidence as adequate to support a conclusion that either Appellant M.S. or Appellant A.T. actually abused he child.”

115. Mr. Dudley characterized the testimony of Defendant Taroli as “not persuasive” and made the administrative recommendation, the “Department of Public Welfare is directed to expunge the indicated report regarding both [REDACTED] and [REDACTED] cited above from the ChildLine registry.”

116. On July 24, 2013, Matthew J. McFadden, Regional Manager of the Pennsylvania Department of Public Welfare’s Bureau of Hearings and Appeals, adopted Mr. Dudley’s recommendation as a “Final Administrative Action”.

117. No appeal of the July 24, 2013 Final Administrative Action expunging the ChildLine indicated reports was ever made by any party.

118. Defendant Taroli reported, “Victims of abusive head trauma often suffer life long consequences, such as seizure disorders, learning disabilities, visual impairment, motor impairments and developmental delay. ... The extent of the neurological damage will only be determined as she grows and deficits are discovered.”

119. Contrary to the foreboding diagnosis made by Defendant Taroli, L.S. is walking, chattering like a 2 year-old, exploring her world like a 2 year-old, achieving all developmental milestones and is thriving in the care of Plaintiffs, Mr. [REDACTED] and Ms. [REDACTED]

[BEGINNING OF ADDITIONAL ALLEGATIONS OF SUBSTANTIVE DUE PROCESS CLAIM AGAINST DEFENDANT TAROLI PURSUANT TO DOC. 25]

**DEFENDANT TAROLI HOLDS HERSELF OUT AS AN EXPERT WHO CAN DIFFERENTIATE BETWEEN MEDICAL CONDITIONS THAT MIMIC THE FINDINGS OF CHILD ABUSE AND ACTUAL CASES OF CHILD ABUSE**

120. In September of 2009, Penn State Hershey established it's Child Safety Team and Defendant Crowell was appointed as a co-director along with Dr. Laura Duda.

121. Defendant Taroli came to PSHMC in August of 2011 to serve as the lead physician of the Child Safety Team.

122. Defendant Taroli holds a subspecialty certification in child abuse pediatrics.

123. Defendant Taroli is a member of the American Academy of Pediatrics and its subsection on child abuse and neglect.

124. Defendant Taroli holds herself out as an expert in distinguishing children with medical findings that mimic child abuse from actual cases of child abuse.

**DEFENDANT TAROLI DISREGARDED THE CONTROVERSY OVER WHETHER SHAKING ALONE CAN CAUSE SUBDURAL HEMORRHAGE**

125. In a now expired 2001 position paper published by the American academy of Pediatrics, Committee on Child Abuse and Neglect, the Academy claimed shaken baby syndrome was a "clearly definable form of child abuse" characterized as having subdural and retinal hemorrhage with "little or no evidence of external cranial trauma".

126. The American Academy of Pediatrics Committee on Child Abuse and Neglect issued a new position paper in 2009 superseding the 2001 position paper. The 2009 position paper

recognized “[t]he relative importance of impact as a contributor to the head injury sustained by abused children became a source of controversy”.

127. On October 31, 2011, the United States Supreme Court recognized the controversy over whether shaking alone can cause subdural and retinal hemorrhages. *Cavazos v. Smith*, 132 S. Ct. 2, 10 (U.S. 2011) (The majority dismissed the case for procedural reasons but commented “Doubts about whether Smith is in fact guilty are understandable.” The three Justice dissent said, “What is now known about shaken baby syndrome (SBS) casts grave doubt on the charge leveled against Smith . . . Doubt has increased in the medical community over whether infants can be fatally injured through shaking alone. . . . By the end of 1998, it had become apparent that there was inadequate scientific evidence to come to a firm conclusion on most aspects of causation, diagnosis, treatment, or any other matters pertaining to SBS, and that the commonly held opinion that the finding of [subdural hemorrhage] and [retinal hemorrhage] in an infant was strong evidence of SBS was unsustainable. . . . [A]n SBS diagnosis in an infant . . . without cervical spine or brain stem injury is questionable. [T]he hypothetical mechanism of manually shaking infants in such a way as to cause intracranial injury is based on a misinterpretation of an experiment done for a different purpose, and contrary to the laws of injury biomechanics as they apply specifically to the infant anatomy.” Quotations and citations omitted.)

128. In December of 2011, Defendant Taroli, though she holds herself out as an expert, disregarded the controversy and doubt over whether shaking alone can cause subdural hemorrhage or retinal hemorrhage.

**DEFENDANT TAROLI DISREGARDED THAT A PRESUMPTION OF ABUSE IS NO LONGER ENDORSED BY THE AMERICAN ACADEMY OF PEDIATRICS**

129. In the now expired 2001 position paper published by the American academy of Pediatrics, the Committee on Child Abuse and Neglect stated that a “presumption of abuse” should be made when “a child under the age of one suffers an intracranial injury”.

130. The American Academy of Pediatrics Committee on Child Abuse and Neglect issued a new position paper in 2009, superseding the 2001 position paper. The 2009 position paper does not endorse a presumption of abuse.

131. In 2011, Defendant Taroli agreed with the expired position paper from 2001 published by the American Academy of Pediatrics Committee on Child Abuse and Neglect about making a presumption of child abuse.

132. Defendant Taroli makes a presumption of abuse when she investigates a case of suspected abuse where a child under the age of one year has subdural/retinal hemorrhage with no external or other evidence of trauma.

133. Defendant Taroli testified that she presumed L.S. was abused.

134. Defendant Taroli believes a presumption of abuse should be made to protect children and ensure that no cases of child abuse are missed.

135. A presumption shifts the burden of proof away from the party asserting the claim and violates due process when a presumption is adopted by a state actor/investigator.

136. Defendant Taroli disregards that the 2009 American Academy of Pediatrics position paper does not endorse a presumption of abuse.

137. Though she holds herself out as an expert, Defendant Taroli testified that she did, in fact, make a presumption of abuse in L.S.'s case, disregarding that such a presumption is no longer endorsed by the American Academy of Pediatrics and that a presumption of abuse violates due process.

**DEFENDANT TAROLI DISREGARDED THAT BIRTH RELATED SUBDURAL HEMORRHAGES AND SPONTANEOUS SUBDURAL HEMORRHAGES ARE REPORTED IN THE MEDICAL LITERATURE**

138. Defendant Taroli was familiar with medical literature that birth commonly causes subdural hemorrhage.

139. Defendant Taroli acknowledges that an uncomplicated birth can cause subdural hemorrhage in an infant.

140. Defendant Taroli was familiar with medical literature reporting that subdural hemorrhages caused by birth “usually virtually all resolve by two months, and I think there's one case report of it not resolving until three months”.

141. The most recent medical journal article, and the study with the largest cohort of infants, to identify how long it takes birth subdural hemorrhages to resolve, is entitled, *Prevalence and Evolution of Intracranial Hemorrhage in Asymptomatic Term Infants*, by V. Rooks et al., 29 Am. J. Neuroradiology 1085 (2008). (hereinafter “Rooks”). See Exhibit 3.

142. Rooks imaged 101 infants after birth and found 46 of the 101 infants had subdural hemorrhage and, after discussing technical limitations, concluded, “the true incidence of [subdural hemorrhage] may be slightly higher than reported in this study”.

143. Rooks states there are a “few published series [that] report the finding of hemorrhages in infants who were symptomatic in the neonatal period.<sup>14-18</sup>” and declares that “[m]ost reports of [subdural hemorrhage] in the neonate appear in the larger body of literature on infants who present with symptomatic [subdural hemorrhage]s.”

144. The purpose of the Rooks study was “to determine the normal incidence, size, distribution, and natural history of SDH in asymptomatic term neonates”.

145. Defendant Taroli disregarded the “larger body of literature on infants who present with symptomatic [subdural hemorrhage]s” when she ruled out birth as the cause of L.S.’s subdural hemorrhage because L.S. had no symptoms after birth.

146. Rooks identified the administration of oxytocin as a risk factor for birth induced subdural hemorrhage.

147. Rooks reported that 75%, three out of four, of the infants in the study who were subjected to a course of labor contractions augmented by oxytocin prior to delivery by c-section had birth induced subdural hemorrhage. See Exhibit 3, Table 3.

148. Rooks followed 18 of the 46 infants who sustained birth induced subdural hemorrhages.

149. Rooks reported, “Our study suggests that SDH in an infant older than 3 months of age is unlikely to be birth-related”.

150. Rooks did not report that every birth induced subdural will always resolve by the age of 3 months based on the Rooks’ study subset of 18 infants that were followed for resolution of their birth induced subdural hemorrhages.

151. Rooks further reported that 1 of the 18 infants followed in the study experienced a spontaneous subdural hemorrhage at 26 days of age. See Exhibit 3, Figure 4.

152. The spontaneous subdural hemorrhage first identified in one infant at 26 days in the Rooks study was reported to resolve by 5 months of age. See Exhibit 3, p.7.

153. Spontaneous subdural hemorrhages in infants are reported elsewhere in the medical literature. (Vinchon M, Delestret I, DeFoort-Dhellemmes S, Desurmont M, Noulé N. “Subdural hematoma in infants: can it occur spontaneously? Data from a prospective series and critical review of the literature.” *Childs Nerv Syst* 2010; 26(9): 1195–205; Amodio J, Spektor V, Pramanik B, etal. “Spontaneous development of bilateral subdural hematomas in an infant with benign infantile hydrocephalus: color Doppler assessment of vessels traversing extra-axial spaces.” *Pediatr Radiol* 2005; 35:1113–17. Epub 2005 May 19)

154. Though she holds herself out as an expert, Defendant Taroli disregarded the medical literature when she ruled out birth as a cause of L.S.'s subdural hemorrhage because L.S. had no symptoms at birth and was 2 months old in December of 2011 when L.S. presented with symptoms.

155. Defendant Taroli also disregarded the medical literature that reports spontaneous subdural hemorrhages occur in infants.

#### **DEFENDANT TAROLI DISREGARDED REVIEWING L.S.'S BIRTH RECORDS**

156. The standard of care in a case of a two-month old infant with subdural hemorrhages and retinal hemorrhages in which there is a suspicion of abuse is for the physician to review the birth records.

157. Defendant Taroli failed to obtain or review L.S.'s birth records prior to rendering her opinion.

158. Had Defendant Taroli reviewed L.S.'s birth records, she would have learned that L.S. had a rare compound birth presentation in which L.S.'s right hand presented with her head during contractions.

159. Had Defendant Taroli reviewed L.S.'s birth records, she would have learned that L.S.'s rare compound birth presentation resulted in observable traumatic abrasions on L.S.'s right hand as a result of her birth.

160. Had Defendant Taroli reviewed L.S.'s birth records, she would have learned that L.S.'s mother was administered medication, oxytocin, to augment the strength of her contractions thus subjecting L.S.'s head to greater strength contractions as L.S.'s head was pushed through the birth canal with her right hand compressed against her head.

161. Though she holds herself out as an expert, Defendant Taroli failed to review L.S.'s birth records prior to rendering her opinion, thus disregarding the standard of care.

**DEFENDANT TAROLI DISREGARDED THAT SUBDURAL HEMORRHAGES REBLEED AND CAN CAUSE SYMPTOMS**

162. Defendant Taroli acknowledged that an infant with an existing subdural hemorrhage can rebleed with minor or no trauma.

163. It is well recognized in the medical literature that rebleeding in an existing subdural hemorrhage is a natural process without trauma and that existing subdural hemorrhages and that rebleeding can cause symptoms. (“While the diagnosis of acute ... [subdural hemorrhage] implies catastrophic forces acting upon the head and brain, acute subdural hemorrhage within an already established chronic subdural hematoma (rebleed) may occur in the absence of significant trauma.” Sargent S, Kennedy JG, Kaplan JA, “Hyperacute” Subdural Hematoma: CT Mimic of Recurrent Episodes of Bleeding in the Child Abuse”, *Journal of Forensic Sciences*, JFSCA, Vol. 41, No. 2, March 1996, pp. 314-316, 315.)

164. Existing subdural hemorrhages have been reported to rebleed from as little as 2.0%, to as much as 27.2%, per day of the existing hemorrhage’s volume with an average of 10.2% per day. (Ito, H., Yamamoto, S., Komai, T., et al. (1976). Role of local hyperfibrinolysis in the etiology of chronic subdural hematoma. *Journal of Neurosurgery*, 45, 26-31.)

165. Rooks states “[r]ebleeding may present either with or without clinical symptoms.” See Exhibit 3, p.7.

166. Defendant Taroli fails to explain, though subdurals rebleed and cause symptoms in adults, why birth subdural hemorrhages cannot rebleed and cause symptoms in infants.

167. Defendant Taroli claims birth induced subdurals do not rebleed and cause symptoms in infants.

168. Defendant Taroli dismissed birth as the cause of L.S.’s subdural hemorrhage because L.S. exhibited symptoms in December of 2011.

169. Though she holds herself out as an expert, Defendant Taroli disregards the well recognized phenomenon that subdural hemorrhages can rebleed and cause symptoms includes subdural hemorrhages caused by birth and spontaneous subdural hemorrhages in infants.

**DEFENDANT TAROLI DISREGARDED THE CONTROVERSY ABOUT WHETHER SHAKING CAN CAUSE RETINAL HEMORRHAGE AT ALL**

170. Defendant Taroli reported that L.S. had retinal hemorrhages.

171. Defendant Taroli reported that L.S.'s retinal hemorrhages "must be considered to be abusive in nature."

172. L.S. was observed to have increased intracranial pressure upon her admission to Penn State Hershey Medical Center in December of 2011.

173. Defendant Taroli agrees that increased intracranial pressure can cause retinal hemorrhage in adults.

174. Defendant Taroli does not believe increased intracranial pressure can cause retinal hemorrhage in infants.

175. Defendant Taroli cannot explain why increased intracranial pressure can cause retinal hemorrhage in adults but not in infants.

176. In 2002, the medical literature reported, "the levels of force required for retinal bleeding by shaking to damage the eye directly is biomechanically improbable. The work of Hansen & Helmke also indicates that the role of sudden rise of ICP [Intra Cranial Pressure] is more likely to cause bleeding than the 'shaken eye' hypothesis." 2002 Ommaya A, Goldsmith W, Thibault L, "Biomechanics and Neuropathology of Adult and Paediatric Head Injury", *Brit J Neurosurg* 2002; 16(3):220-242.

177. In 2005 the medical literature reported, "retinal hemorrhages are also associated with an ever-expanding list of conditions, each of which carries important implications for patients and

their families. ... Sudden increases in intracranial pressure, regardless of etiology, have been associated with retinal and optic nerve hemorrhage in adults. ... In cases of suspected child abuse, it is wise to remember that the differential diagnosis of retinal hemorrhage is vast, and to suspend judgment until all other reasonable explanations are exhausted.” Aryan et al, “Retinal hemorrhage and pediatric brain injury: etiology and review of the literature”, *Journal of Clinical Neuroscience* (2005) 12(6), 624–631 0967-5868/\$ doi:10.1016/j.jocn.2005.05.005

178. In 2007 the medical literature reported, “Conclusions: The two cases, one with accidental and the other nonaccidental injury, demonstrate very similar eye findings. This supports the argument that there may be no pathognomonic eye signs in shaken baby syndrome. 2007 Obi E, Watts P. “Are there any pathognomonic signs in shaken baby syndrome?”, *J AAPOS*. 2007;11:99-100.

179. In 2007 the medical literature reported, “The mechanism of retinal hemorrhage formation in child abuse has been the subject of great speculation and little agreement. ... much of what we think we know about the systemic and ocular findings of child abuse will continue to be the result of speculation rather than based on sound evidence. 2007 Emerson M, Jakobs E, Green R. “Ocular Autopsy and Histopathologic Features of Child Abuse”, *Ophthalmology* 2007;114:1384–1394.

180. “[I]t seems unlikely that shaking of an infant would result in significant vitreoretinal traction, or that this would lead to retinal haemorrhage. ... the eye is ‘designed’ to rotate, for example during saccadic eye movements, during which angular accelerations of up to 700° per second may be achieved, and the vestibulo-ocular reflex is likely to mitigate the effects of rotation of the head on the eye. Retinal haemorrhages are not observed after saccadic eye movements... Rotational forces are intentionally applied to the eye by some surgeons during strabismus surgery --- the ‘spring back balance test’ of Jampolsky, without causing haemorrhage. ... without a clearer understanding of the processes involved in the pathogenesis of these findings, it remains impossible, despite the assertions

of some authors, to be certain that all infants demonstrating them have been the victims of attempted, or actual, murder.” Clarke MP. “Vitreoretinal traction is a major factor in causing the haemorrhagic retinopathy of abusive head injury? No”, *Eye (Lond)*. 2009; 23(9):1761–1763. See Exhibit 4.

181. Though she holds herself out as an expert, Defendant Taroli disregards the controversy about whether shaking can cause retinal hemorrhages at all and disregards that increased intracranial pressure can cause retinal hemorrhages in infants.

**DEFENDANT TAROLI RENDERED AN UNHURRIED JUDGMENT**

182. Defendant Taroli was first consulted on December 3, 2011 and faxed her report to Lancaster County Children and Youth Services on December 16, 2011, three days after L.S. was discharged from the hospital.

183. Defendant Taroli’s report cites that a CT scan was performed on December 11, 2011, that surgical drains were removed on December 12, 2011 and that L.S. was discharged on December 13, 2011.

184. Defendant Taroli’s opinion was not rendered in a “hyperpressurized environment”, nor did Defendant Taroli have to render a “split second” decision or a decision within minutes or hours.

185. Defendant Taroli took 13 days, nearly two weeks, from December 3, 2011 until December 16, 2011, to deliberate and render her unhurried judgment.

186. Defendant Taroli could have taken even more time than 13 days to deliberate and render her unhurried judgment, if she, in her sole discretion, believed additional information or time was required.

187. Situations involving days or weeks to act require only deliberate indifference to prove conscience shocking behavior.

## SUMMARY

188. While holding herself out as an expert who can distinguish medical conditions that mimic the findings of child abuse from actual child abuse, Defendant Taroli, in an unhurried judgment, consciously disregarded a great risk that L.S. had not been abused as demonstrated by her conscious disregard of, and deliberate indifference to, the following:

- a. The controversy recognized by the American Academy of Pediatrics over whether shaking alone can cause subdural hemorrhage,
- b. That a presumption of abuse was no longer endorsed by the American Academy of Pediatrics in 2011,
- c. That oxytocin is a risk factor for birth induced subdural hemorrhages,
- d. That L.S.'s birth records show L.S. experienced a rare compound birth presentation,
- e. That L.S.'s mother, Plaintiff [REDACTED] [REDACTED] was administered the contraction strength augmenting medication, oxytocin, during L.S.'s birth,
- f. That L.S.'s birth records document that L.S. had peeling skin abrasions on her right hand from her traumatic, rare, right hand compound presentation, oxytocin augmented contraction birth,
- g. That Defendant Taroli failed to obtain and review L.S.'s birth records prior to rendering her opinion,
- h. That birth induced subdural hemorrhages are reported occur in nearly half of all births,
- i. That most, but not necessarily all, birth induced subdural hemorrhages resolve by 3 months,
- j. That infants who sustain birth induced subdural hemorrhages commonly exhibit no immediate symptoms,

k. That birth induced subdural hemorrhages cannot be ruled out because L.S. exhibited no symptoms at birth, and then later exhibited symptoms at 2 months of age,

l. That spontaneous subdural hemorrhage is reported to occur in infants,

m. That Rooks reported the spontaneous subdural hemorrhages in one patient resolved by 5 months of age,

n. That it is well recognized that subdural hemorrhages rebleed and cause symptoms in adults,

o. That Defendant Taroi cannot explain why rebleeding birth induced or spontaneous subdural hemorrhages cannot cause symptoms in infants when it is well recognized that rebleeding subdural hemorrhages cause symptoms in adults,

p. That L.S.'s symptoms were consistent with a rebleeding subdural hemorrhage,

q. That L.S. experienced increased intracranial pressure,

r. That increased intracranial pressure is well recognized to cause retinal hemorrhages in adults,

s. That Defendant Taroli cannot explain why increased intracranial pressure would not cause retinal hemorrhages in infants when it is well recognized that increased intracranial pressure causes retinal hemorrhages in adults,

t. That there is controversy about whether shaking can cause retinal hemorrhages at all, and

u. The fact that L.S.'s head circumference did not follow the 75<sup>th</sup> percentile as Defendant Taroli misrepresented in her December 16, 2011 report, but actually increased from the 90-95<sup>th</sup> percentile at birth, to the 98<sup>th</sup> percentile at one month, to well above the 98<sup>th</sup> percentile at two months as reported by L.S.'s treating pediatrician.

[END OF ADDITIONAL ALLEGATIONS OF SUBSTANTIVE DUE PROCESS CLAIM AGAINST DEFENDANT TAROLI PURSUANT TO DOC. 25]

189. Plaintiffs seeks compensatory, punitive and other damages as the court may find appropriate for Defendants' above cited conduct resulting in the infringement of Plaintiffs' right to familial association with, and the care, custody and control of, L.S.; the denial of Plaintiffs' right to due process when "agreement" to safety plans were coerced with threats of placing L.S. in foster care; denial of Plaintiffs' right to due process when medical providers exceeded the scope of consent to medical treatment and embarked on a child abuse investigation; breach of the doctor-patient fiduciary duty of confidentiality and contractual duty of good faith and fair dealing; breach of the duty to provide Plaintiffs with notice that PSHMC employee Defendants elected to participate in a child abuse investigation and, as a result, were immune from civil liability; all in violation of the United States Constitution, the Pennsylvania Constitution and Pennsylvania law.

190. The foregoing averments place all Defendants on notice that their actions have caused Plaintiffs, [REDACTED] and L.S. harm including, but not limited to, the following claims, pursuant to the First, Fourth, Fifth, Sixth, Seventh and Fourteenth Amendments of the United States Constitution, Article I of the Pennsylvania Constitution, Pennsylvania law and 42 U.S.C. §1981, §1983 & §1985, as applicable, against:

- I. Defendant Lancaster County for having a policy of not providing parents due process to challenge any safety plan and failing to train its employees that, when an employee coerces a safety plan, due process considerations are triggered.
- II. (Dismissed pursuant to Doc. 23) Defendant Lancaster County for having a policy of not notifying parents who are the subject of a child abuse investigation that physicians participating in the child abuse investigation are immune from civil suit for breaches of physician-patient fiduciary, professional and contractual duties and not training its employees to provide such notice.

- III. Defendants Garber, Redcay, Boyer, Hasselback, Murray and Huegel for failing to provide Plaintiffs with due process to challenge the safety plans.
- IV. (Dismissed with Prejudice pursuant to Doc. 23) Defendants Garber, Redcay, Boyer, Hasselback, Murray and Huegel for failing to notify Plaintiffs that physicians participating in the child abuse investigation are immune from civil suit for breaches of physician-patient fiduciary duties and breaches of the physicians' professional and contractual duties.
- V. (Dismissed pursuant to Doc. 23) Defendants Garber, Redcay, Boyer, Hasselback, Murray, Huegel, Taroli, Crowell, Dias and Weinstein, for conspiring to deny Plaintiffs their right to familial association and right to the care, custody and control of L.S. by denying Plaintiffs any forum or due process to defend false allegations that [REDACTED] and [REDACTED] [REDACTED] abused L.S.
- VI. (Dismissed pursuant to Doc. 25) Defendant Penn State Hershey Medical Center for having a policy of initiating and requiring its medical doctors to refer patients suspected being abused to the PSHMC Child Safety Team for a child abuse investigation.
- VII. (Dismissed pursuant to Doc. 25) Defendant Penn State Hershey Medical Center for failing to notify Plaintiffs that PSHMC doctors who elect to participate in the child abuse investigation are immune from civil suit for breaches of physician-patient fiduciary, professional and contractual duties.
- VIII. Defendant Taroli for her grossly negligent consultation and report, and presuming L.S. was abused due to the presence of retino-dural hemorrhage of infancy during her child abuse investigation; for failing to believe the history from the Plaintiffs that L.S. suffered no inflicted trauma; for fabricating evidence that retinoschisis is "tearing of the retina off its attachment"; for fabricating evidence that L.S.'s "head circumference grew steadily along the

75<sup>th</sup> %ile until 1 month of age”; for recommending medically unnecessary tests for the purpose of investigation rather than treatment; for willful indifference to, and failure to report, the fact that the shaken baby syndrome hypothesis and its associated shaken eye hypothesis are “simply hypotheses, not proven medical or scientific facts” and that it is not reasonable to “infer shaking (or any other form of child abuse) from a finding of retino-dural hemorrhage of infancy”; for failing to review L.S.’s birth records that documented a rare compound right hand birth presentation and that oxytocin, though contraindicated, was administered to augment Ms. [REDACTED] contractions during labor; for breaching her fiduciary duty to keep informational confidential; and for her participation in an administrative expungement proceeding against the Plaintiffs.

- IX. (Dismissed pursuant to Doc. 25) Defendant Crowell for her grossly negligent consult and breach of her contractual duty of good faith and fair dealing when she failed to believe the history provided by Mr. [REDACTED] and Ms. [REDACTED] that L.S. suffered no inflicted trauma, for breaching her contractual duty by recommending medically unnecessary tests outside the scope of the Plaintiffs’ medical consent and for breaching her fiduciary duty of keeping information confidential.
- X. (Dismissed pursuant to Doc. 25) Defendant Weinstein for his grossly negligent consult and dismissal of the history provided by Plaintiffs that L.S. did not suffer any inflicted head trauma and recommendation that retinal photos be taken for purposes of child abuse investigation rather than medical treatment, thus demonstrating willful indifference to the truth, to the Plaintiffs’ rights and to the fact that the shaken eye hypothesis is “simply [a] hypothes[is], not proven medical or scientific facts” and that it is not reasonable to “infer

shaking (or any other form of child abuse) from a finding of retino-dural hemorrhage of infancy”.

- XI. (Dismissed pursuant to Doc. 25) Defendants Rocourt and Sheehan for their negligent consult and failure to believe the history from Plaintiffs that L.S. suffered no inflicted or accidental trauma and recommending the initiation of a PSHMC child safety team investigation into suspected abuse, an investigation that was not mandated by law, was not medically necessary and was not related to the medical treatment of L.S.’s RDHI.
- XII. (Dismissed pursuant to Doc. 25) Defendant Dias for discharging L.S. to the Services of Defendant Garber of Defendant Lancaster County Children and Youth Services agency and discharging L.S. to a follow-up skeletal survey, both of which were not medically necessary and not related to the medical treatment of L.S.’s RDHI.
- XIII. (Dismissed pursuant to Doc. 25) Defendants Taroli, Crowell, Weinstein, Rocourt, Sheehan and Dias for failure to notify Plaintiffs that these Defendants each elected to participate in the child abuse investigation and, as a result, were immune from civil suit for breaches of physician-patient fiduciary duty to maintain confidentiality breaches of their contractual duty to Plaintiffs to act in good faith and fair dealing and for civil claims related to their participation in the investigation of child abuse.
- XIV. Any other claim against the Defendants for which the above averments and/or additional facts discovered during litigation provide notice.

WHEREFORE, Plaintiffs, [REDACTED] [REDACTED] [REDACTED] [REDACTED] and L.S., respectfully request the court enter judgment in favor of Plaintiffs and against Defendants.

Respectfully submitted,

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**DECLARATION**

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

Signed this \_\_\_\_\_ day of \_\_\_\_\_, 2014

\_\_\_\_\_  
[Redacted]

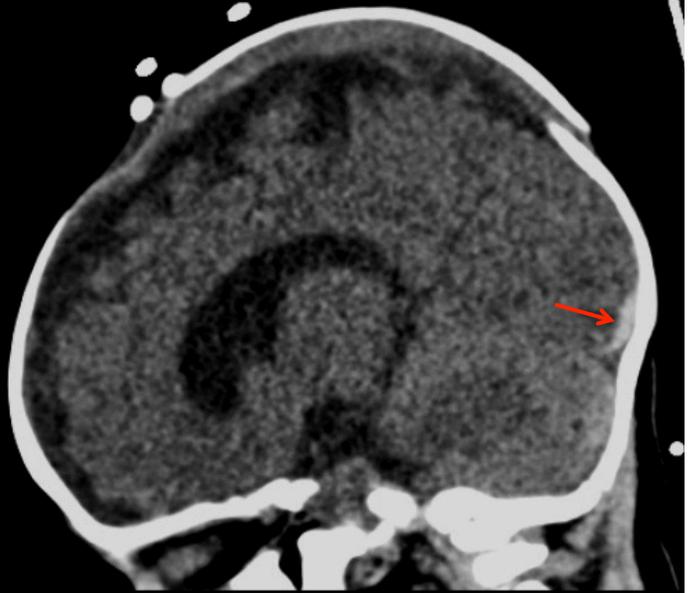
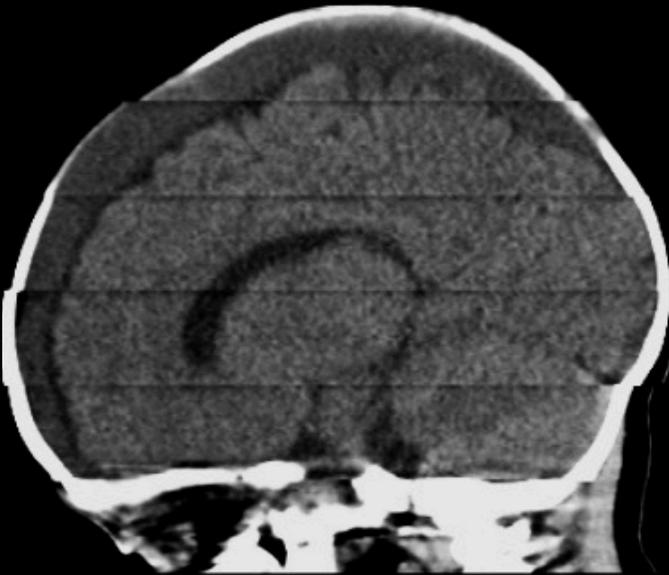
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Sagittal view same area, admission versus 12-11-11 CT

Admission CT 12-2-11  
sagittal

12-11-11



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ORIGINAL  
RESEARCH

V.J. Rooks  
J.P. Eaton  
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R.C. Pedersen



## Prevalence and Evolution of Intracranial Hemorrhage in Asymptomatic Term Infants

**BACKGROUND AND PURPOSE:** Subdural hemorrhage (SDH) is often associated with infants experiencing nonaccidental injury (NAI). A study of the appearance and natural evolution of these birth-related hemorrhages, particularly SDH, is important in the forensic evaluation of NAI. The purpose of this study was to determine the normal incidence, size, distribution, and natural history of SDH in asymptomatic term neonates as detected by sonography (US) and MR imaging within 72 hours of birth.

**MATERIALS AND METHODS:** Birth history, delivery method, duration of each stage of labor, pharmaceutical augmentation, and complications during delivery as well as postnatal physical examination were recorded. Brain MR imaging and US were performed on 101 asymptomatic term infants at 3–7 days, 2 weeks, 1 month, and 3 months. Clinical follow-up at 24 months was recorded.

**RESULTS:** Forty-six neonates had SDH by MR imaging within 72 hours of delivery. SDH was seen in both vaginal and cesarean deliveries. All neonates were asymptomatic, with normal findings on physical examination. All 46 had supratentorial SDH seen in the posterior cranium. Twenty (43%) also had infratentorial SDH. US detected 11 of the 20 (55%) infratentorial SDHs and no supratentorial SDH. Most SDHs present at birth were  $\leq 3$  mm and had resolved by 1 month, and all resolved by 3 months on MR imaging. Most children with SDHs had normal findings on developmental examinations at 24 months.

**CONCLUSION:** SDH in asymptomatic term neonates after delivery is limited in size and location.

Subdural hemorrhage (SDH) is often associated with infants experiencing nonaccidental injury (NAI).<sup>1–13</sup> Birth-related trauma is used in the court of law as an explanation for SDH in infants with suspected NAI because a variety of hemorrhages have been reported in term neonates. A study of the appearance and natural evolution of these birth-related hemorrhages, particularly SDH, is important in the forensic evaluation of NAI. A few published series report the finding of hemorrhages in infants who were symptomatic in the neonatal period.<sup>14–18</sup> Some reports suggest that the risk of SDH and other hemorrhages found on imaging of symptomatic infants varies with the method of delivery.<sup>19</sup> Sonography (US) is standard practice for detecting germinal matrix hemorrhage in the preterm neonate and has also been proved to demonstrate posterior fossa SDH.<sup>14</sup> MR imaging in general has a high sensitivity for intracranial hemorrhage, and, with its lack of ionizing radiation, is a favorable technique for the evaluation of birth trauma over CT, especially for a neonate. Previous studies conducted in an effort to determine the incidence and natural history of asymptomatic SDH in the neonate have been limited by the use of low-field-strength (0.2T) MR imaging,

small patient numbers, or variable timing of imaging after birth.<sup>20–23</sup>

The purpose of this study was to determine the normal incidence, size, appearance, and distribution of SDH in asymptomatic term neonates as detected by US and 1.5T MR imaging within 72 hours of birth. In addition, we prospectively studied the natural history of these hemorrhages. This study can then serve as a baseline for comparison with an abnormal pattern of SDH seen in abuse.

### Methods

The protocol was approved by the Scientific Review and Human Use Committees of the hospital. Neonates of at least 37 weeks gestation, with normal findings on neonate physical examination by a board-certified physician were eligible for the study. The first 101 patients whose parents gave written consent during the approved study period were included. Birth history, delivery method, duration of each stage of labor, pharmaceutical augmentation with oxytocin, and complications during delivery were recorded. All neonates had normal findings on neurologic examination by a board-certified child neurologist before imaging. Ophthalmologic examination of the retina was not performed on any neonates. The first MR imaging and US for each patient were performed at <72 hours of age.

US was performed on an Acuson Sequoia 512 (Siemens Medical Solutions, Malvern, Pa) by using 8V5 and 15L8 transducers. Standard coronal and sagittal images of the neonatal brain through the anterior fontanelle and images of the posterior fossa via the mastoid fontanelle were obtained. Color Doppler flow imaging was also used when the findings of gray-scale imaging were positive for SDH. US was performed within 1 hour of the MR imaging. SDH was defined as an extracerebral curvilinear echogenicity subjacent to the calvaria without evidence of central traversing vessels on color Doppler imaging.

Imaging was timed to occur after a morning feeding. Infants were transported to the radiology department in a mobile bassinette, placed on the MR imaging table in an 8-channel head coil, and secured with a sheet, sponges, and tape to minimize motion. Pieces of

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indicates article with supplemental on-line table.

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PEDIATRICS

ORIGINAL RESEARCH

standard foam ear protection were taped in place, and a pacifier was offered for comfort. No infants were given sedation medications. With a Signa 1.5T MR imaging scanner (software 11.0\_M4\_0403a) (GE Healthcare, Milwaukee, Wis), we used the following imaging sequences: 1) 3-plane localizer; 2) sagittal T2 single-shot fast spin-echo (SE) 2D pulse sequence imaging option with a TE of 90, TR of 3000, bandwidth of 31.25, FOV of 18, section thickness of 4, 0 skip, matrix of  $256 \times 192$ , frequency signal intensity, NEX 1, phase FOV of 0.70; 3) axial multiplanar gradient recall (MPGR) pulse sequence gradient-echo imaging option, flow comp, VBW, with a TE of 20, TR of 355, flip angle of  $20^\circ$ , bandwidth of 15.63, FOV of 18, section thickness of 4, 0 skip, matrix of  $256 \times 192$ , frequency AP, NEX 1, phase FOV of 0.75; 4) axial T1 conventional SE 2D pulse sequence imaging option, VBW, TE min, TR of 377, bandwidth of 15.63, SAT I, FOV of 18, section thickness of 4, 0 skip, matrix  $256 \times 192$ , NEX 0.75, phase FOV of 0.75, frequency AP; 5) coronal T1 (posterior fossa) 2D pulse sequence SE imaging option, VBW, TE min, TR of 502, bandwidth of 15.63, SAT I, FOV of 18, section thickness of 4, 0 skip, matrix  $256 \times 192$ , frequency direction S/I, NEX 0.75, phase FOV of 0.75; 6) axial fluid-attenuated inversion recovery (FLAIR) 2D pulse sequence IR imaging option, tailored radio-frequency fast, zip of 512, TE of 120, TR of 10,000, TI of 2200, bandwidth of 15.63, FOV of 18, section thickness of 4, 0 skip, matrix  $256 \times 224$ , frequency direction A/P, NEX 1; 7) axial diffusion-weighted echo-planar imaging (DWI EPI) 2D SE imaging option (DIFF), number of shots 1, TE min, TR of 10,000, DWI screen b-value of 1500, diffusion direction ALL, frequency of 128/128, NEX 1, FOV of 18, section thickness of 4, 0 skip, matrix  $128 \times 128$ . Conventional SE T1 was substituted for fast SE after 42 patients were scanned.

MR and US images were independently reviewed on a PACS (Centricity; GE Healthcare) by 2 board-certified radiologists each with a Certificate of Added Qualification in neuroradiology or pediatric radiology. The child neurologist discussed imaging results with parents. Infants with SDH detected on initial imaging were scheduled for follow-up MR imaging and US examinations at 3–7 days, 2 weeks, 1 month, and 3 months or until the MR imaging and US findings were both negative. If the initial US findings were normal, no further US images were obtained. Final interpretations regarding the presence of SDH on MR imaging were determined by consensus of 2 of the radiologists based on SDH seen on both the immediate postdelivery initial MR imaging and the first follow-up at 3–7 days. SDH on MR imaging was defined as an extracerebral curvilinear signal-intensity abnormality corresponding to blood products that did not extend into the sulci. For US and MR imaging, SDH location and size were recorded, with size measured as a maximal width in the axial plane by using electronic calipers. In infants with SDH in multiple locations, the size of the largest SDH was recorded. The presence of cephalohematomas was also recorded. Evaluation for coagulopathy was not routinely performed.

Comparison of the incidence of SDH among the delivery groups was made by using the Fisher exact test. The average labor times and birth weights of those with SDH and those without were compared by using a Student *t* test or the nonparametric Wilcoxon test if the variance in data was unequal between groups. The Fisher exact test was used to compare prolonged duration of labor and incidence of cephalohematoma in those with SDH versus those without. The comparison of the incidence of SDH in vaginal and cesarean deliveries augmented with oxytocin administration was also performed by using the Fisher exact test in addition to computation of the odds ratio of increased SDH associated with giving oxytocin. Data are expressed as

mean  $\pm$  standard error of the mean and/or as a median within the range of values obtained. For all tests, a value of  $P < .05$  was considered significant. The first stage of labor was defined as the duration from the onset of labor until the fetus was engaged in the birth canal. The second stage of labor was defined as the duration of fetal descent through the birth canal.

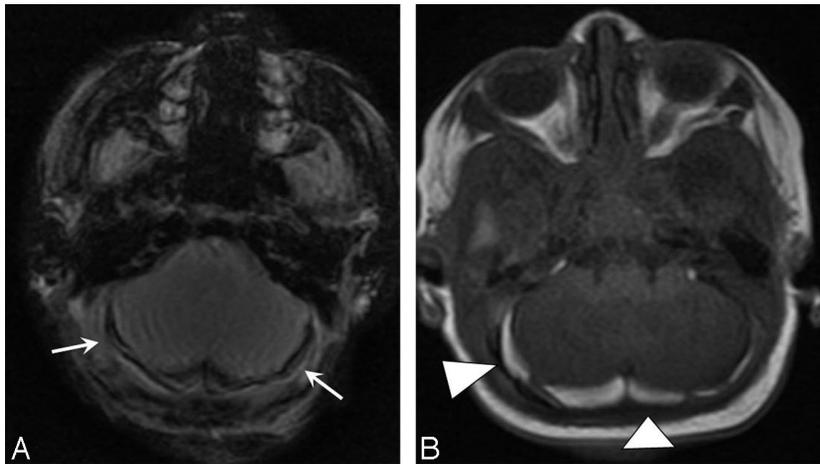
Clinical follow-up was performed in all patients who demonstrated SDH on imaging. Patients were evaluated at their 24-month well-child visit and assessed for developmental delay. A developmental delay (motor or speech) was defined as a delay in a particular developmental domain compared with expected norms for age. Developmental delay is used as a temporary diagnosis in young children at risk for developmental disabilities, indicating a failure to achieve age-appropriate neurodevelopmental milestones.<sup>24</sup> At our institution, assessing for developmental delays is part of every well-child visit, typically performed at 2, 4, 6, 12, 15, 18, and 24 months. The Denver Developmental Screening Test II is applied to each child at their well-child visit.<sup>25</sup>

## Results

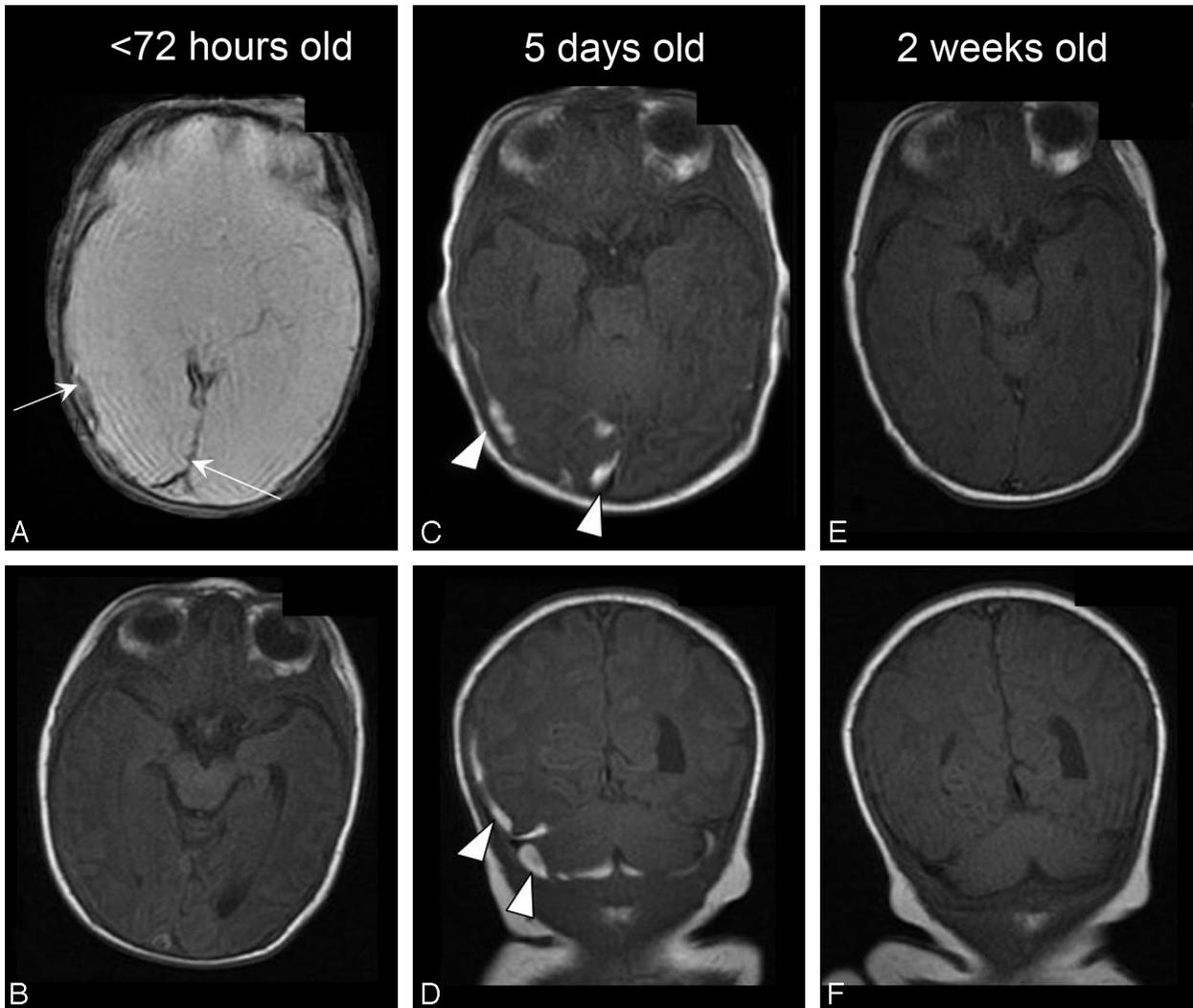
One hundred one patients were enrolled in the study between January 2005 and March 2006. There were 58 male and 43 female infants. Seventy-nine (78%) infants were born via vaginal delivery with (80%) via spontaneous delivery (SVD), 10 (12%) with vacuum assistance, and 6 (8%) with forceps assistance (supplemental on-line Table). Thirty-five vaginal deliveries were induced or augmented with oxytocin. Twenty-two (22%) infants were delivered via cesarean delivery: 13 elective cesarean deliveries and 9 for failure to progress and/or fetal distress after a trial of labor. Four of the cesarean deliveries had a trial of labor augmented with oxytocin. One cesarean delivery was assisted with forceps, and 1 was assisted with vacuum extraction. All neonates had normal findings on neurologic examinations at birth.

All 101 initial MR imaging examinations were successful, without significant motion artifact. Most infants slept through the entire examination. Examination times required  $<10$  minutes to complete. Three MR imaging examinations were thought to be positive for SDH on initial sequences, but the findings were normal at the first follow-up MR imaging by 3–7 days of life. These were presumed to be false-positive findings and were recategorized as negative findings. Forty-six (46%) infants had SDH on initial MR imaging that was confirmed on follow-up studies (supplemental on-line Table). Forty-four of 46 (95.9%) had SDH of  $\leq 3$  mm in thickness (range, 1.0–4.3 mm; mean, 2.1 mm). SDH was best visualized on the initial MR imaging MPGR sequence performed before 72 hours of life (Fig 1). All 46 patients with intracranial hemorrhage had supratentorial SDH confirmed on 2 imaging planes on follow-up imaging. All supratentorial SDHs identified within 72 hours postdelivery were seen in the posterior half of the cranium. Twelve (26%) infants had SDH noted in only 1 location, whereas most infants had SDH in 2 or 3 locations. In all, SDH was most commonly seen in the posterior interhemispheric fissure (parafalcine location) (30, 65%), with SDH also noted posteriorly along the occipital lobes in 29 (63%) and over the tentorium in 22 (48%) (supplemental on-line Table). All SDHs were homogeneous in signal intensity on all sequences.

Twenty (43%) of the neonates with supratentorial SDH also had posterior fossa SDH (Fig 2). No neonate had only



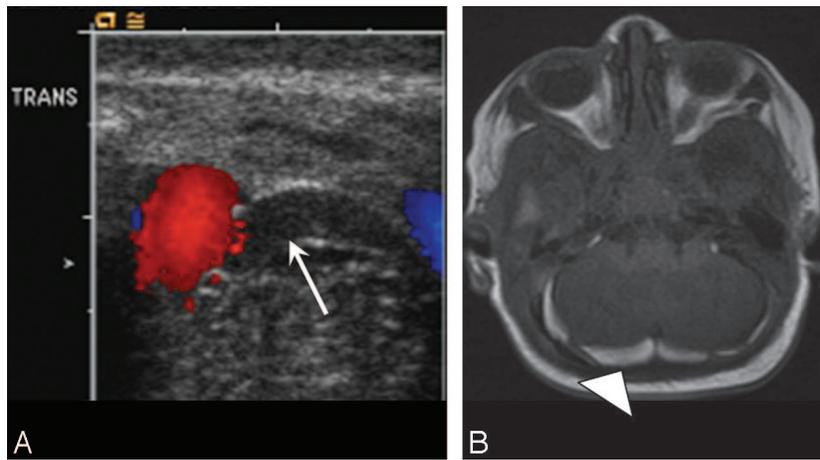
**Fig 1.** Posterior fossa SDH in a neonate delivered via SVD. *A*, Axial MPGR at <72 hours of life demonstrates lobular symmetric low signal intensity with blooming in the posterior fossa (*arrows*). *B*, Follow-up T1 images show high-signal-intensity SDH (*arrowheads*) by 7 days.



**Fig 2.** Neonate delivered via SVD with both supratentorial and infratentorial SDH. *A* and *B*, Initial examination shows the lobular occipital SDH to be very low signal intensity on MPGR (*arrows*, *A*) and isointense to gray matter and difficult to detect on the SE T1-weighted MR image (*B*). *C* and *D*, Five-day follow-up shows high T1 SDH (*arrowheads*) in 2 locations in 2 planes, axial supratentorial (*C*) and coronal, both supra- and infratentorial (*D*). *E* and *F*, Two-week follow-up shows complete resolution of hemorrhage on T1 images.

posterior fossa hemorrhage detected by MR imaging. No neonate had MR imaging evidence of subarachnoid, epidural, or intraparenchymal hemorrhage. No parenchymal contusions were seen. Two neonates had grade I germinal matrix hemor-

rhages (1 unilateral, 1 bilateral) as well as SDHs. Twenty-two neonates had a cephalohematoma noted at MR imaging. Eighteen (82%) of these neonates had SDH. Most (11/18, 61%) had posterior fossa SDH as well as supratentorial SDH. One



**Fig 3.** Neonate delivered via SVD with posterior fossa SDH seen on US and confirmed on MR imaging. *A*, Axial sonogram of the posterior fossa through the mastoid fontanel demonstrates initial curvilinear echogenic focus adjacent to the transverse sinus (arrow). *B*, Axial T1-weighted MR image confirms high-signal-intensity posterior fossa SDH (arrowhead) on day 7 of life.

**Table 1: SDH versus mode of delivery**

	SVD	Vacuum	Forceps	C/S
Total deliveries	63	10	6	22
SDH	32	6	4	4
Percentage	51	60	67	18
<i>P</i> value*	<.05	<.05	<.05	

**Note:**—C/S indicates cesarean delivery; SVD, spontaneous vaginal delivery.  
\* *P* values represent significance of comparisons with the C/S group.

had a 1.6-cm paraventricular mass incidentally detected on MR imaging, which was not seen on repeat US performed after the initial MR imaging. The mass, thought to be a hamartoma, was observed with expectant management. It remained asymptomatic and unchanged in size on 4-month follow-up at our institution before the patient’s family moved from our area.

Posterior fossa SDH was seen at US in 11 (11%) neonates, and all SDHs were confirmed on MR imaging (Fig 3). Thus, only 55% of the 20 posterior fossa SDHs seen on MR imaging were identified independently on US examination. US was focused along the lateral aspects through the mastoid fontanelle. Sensitivity of US detection of posterior fossa SDH improved when the 3 infants with posterior fossa SDH isolated to midline were excluded on MR imaging<sup>3</sup>; thus, 11/17 (65%) lateral posterior fossa SDHs were detected on US. All SDHs seen on US were also seen on MR imaging. No supratentorial hemorrhages were detected at US.

The incidence of SDH versus mode of delivery is shown in Table 1. All 4 neonates with SDH delivered by cesarean birth had supratentorial SDH only. One of the neonates with SDH and delivered by cesarean birth was born via elective cesarean delivery for macrosomia, whereas 3 of 4 (75%) neonates with SDH and delivered by cesarean birth had failed a trial of oxytocin-augmented labor before cesarean delivery. One of these cesarean deliveries required vacuum assistance. In comparison with the neonates delivered via cesarean delivery, rates of SDH were significantly higher in all the vaginal delivery groups (Table 1). There was no statistically significant difference in the presence of SDH in each of the vaginal delivery groups.

The duration of the first and second stages of labor was recorded for all neonates delivered vaginally. For neonates with SDH, the mean duration of the first stage of labor was not significantly different from that in those without SDH (Table

2). The second stage of labor was significantly longer in neonates with SDH than in those without SDH. A prolonged second stage of labor (>2 hours) was also significantly longer in the group with SDH, compared with the group without SDH. The incidence of cephalohematoma was greater in neonates with SDH than in those without SDH. There was no difference in average second-stage labor duration in those with a cephalohematoma compared with those without. The mean birth weight of neonates with SDH on MR imaging was higher than that of those with normal findings on MR imaging (Table 2).

The overall incidence of SDH in the 39 patients who received oxytocin was not different from the incidence of SDH in the 62 patients who did not receive oxytocin (Table 3). This was also true for the subgroup of vaginal deliveries. However, closer examination of cesarean delivery revealed that the incidence of SDH when oxytocin was given before cesarean delivery was much higher (Table 3).

Follow-up imaging was completed in 18/46 (39.1%) patients with SDH. All 18 patients demonstrated resolution by 3 months. Two patients were only imaged at birth and at 3 months due to scheduling conflicts. Both of these patients had normal MR imaging findings at 3 months. Fifteen of 16 patients (93.8%) whose follow-up imaging included a 1-month MR imaging had interval resolution of their SDHs. One patient had a new frontal SDH on the 2-week MR imaging follow-up examination (Fig 4). This patient had bilateral occipital and posterior fossa SDH on initial imaging at birth, confirmed on the 7-day follow-up MR imaging. He was also noted to have extra-axial collections of infancy. At 26-days postnatal age, the MR imaging demonstrated left frontal subdural collections that did not conform to CSF signal intensity. Of the 46 infants with SDH, 43 children had records of 2 years of well-baby examinations at our institution. One child was only followed to 2 months, 1 child’s family had moved out of the area, and 1 child was not eligible for continued care in our system. None of the 43 infants had gross motor delay. Six (14%) children were noted to have speech delay, and 1 (2%) is currently being evaluated for an autistic spectrum disorder.

**Discussion**

We confirmed reports that SDH occurs in the asymptomatic neonate after delivery.<sup>20-22</sup> The incidence of SDH (46%) is significantly higher in our study than in previous reports. Our

**Table 2: Vaginally delivered neonates with and without SDH: mean length of each stage of labor, birth weight, and incidence of cephalohematoma**

	1st stage (min)	2nd stage (min)	2nd stage >120 minutes	Cephalohematoma	Birth Weight (g)
No SDH (n = 55)	414, range, 37–1439; median, 357	50, range, 11–554; median, 19	3 5%	5 9%	3404, range, 2842–4379
SDH (n = 46)	448, range, 75–1397; median, 380	96, range, 1–593; median, 27	12 26%	18 39%	3589, range, 2867–4583
P value*	>.01	<.01	<.01	<.01	

\* P values represent significance of comparisons between no SDH and SDH.

**Table 3: SDH and use of oxytocin in vaginal and cesarean deliveries**

	Vaginal		Cesarean Delivery	
	No	Yes	No	Yes
Oxytocin				
No.	44	35	18	4
No SDH	18	19	17	1
Percentage	41	54	94.4	25
SDH	26	16	1	3
Percentage	59	46	5.6	75
P Value*	>.01	>.01	<.01	>.01

\* P values represent significance of comparisons between no SDH and SDH.

higher incidence may be related to improved detection and increased sensitivity with a higher magnetic-field-strength 1.5T MR imaging scanner. Whitby et al,<sup>20</sup> by using a low-field-strength 0.2T magnet, reported an SDH incidence of 8% overall and 10.5% in vaginal deliveries when they imaged within the first 48 hours of life. Our reported incidence is most like that of Holden et al,<sup>21</sup> who, in a pilot study also using 1.5T MR imaging in 1999, saw SDH in 4 of 8 (50%) asymptomatic neonates in the first 4 days of life. These results suggest that SDH after uncomplicated vaginal delivery is a common finding on MR imaging.

Patient age at the time of MR imaging is an important factor in determining the incidence of SDH in neonates. We imaged neonates within the first 72 hours of life and found SDH most readily detectable on a gradient-echo sequence, confirmed on follow-up T1 sequences at 3–7 days of life. Most of the SDHs resolved by 4 weeks. Whitby et al<sup>20</sup> also found that their 9 patients with SDH first seen within 48 hours of life had resolution of hemorrhage on MR imaging at 4-week follow-up. Recently, Looney et al,<sup>22</sup> by using 3T MR imaging, reported SDH in 26% of neonates delivered vaginally. Infants in this study were scanned between 1 and 5 weeks of age. We agree that the true incidence in the population of Looney et al may have been higher than the prevalence reported because they may have missed SDHs that were present earlier in life and had resolved by the time of first imaging. Patient age at the time of MR imaging may also be important in determining an etiology for neonate SDH. In our patients, not only were most SDHs resolved by 1 month but SDHs had resolved by 3 months in all patients. This information may be useful to the radiologist asked to comment on the etiology of SDH in an infant. Our study suggests that SDH in an infant older than 3 months of age is unlikely to be birth-related regardless of the mode of delivery.

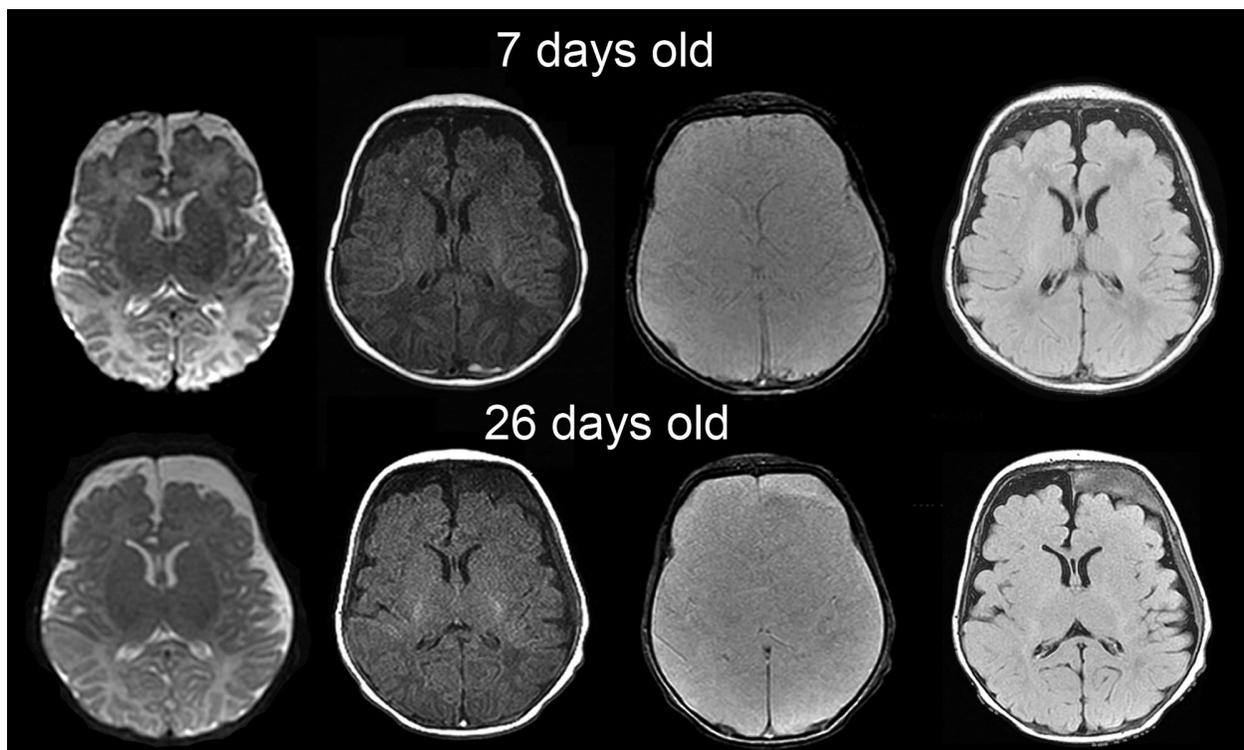
Proposed mechanisms for SDH have included tears of the falx and tentorium or bridging cortical veins secondary to stretching,<sup>11</sup> difficult delivery,<sup>26,27</sup> or abnormal labor.<sup>19</sup> One

suggested mechanism of hemorrhage after vaginal delivery is that increased circumferential pressure and squeezing of the head in the birthing canal result in overlap at the sutures, mechanical compression, and shearing of the bridging veins during delivery, resulting in SDH.<sup>28</sup> The true etiology remains unknown because there is a paucity of evidence-based literature on this subject. Most reports of SDH in the neonate appear in the larger body of literature on infants who present with symptomatic SDHs.

The forensic literature suggests that SDH can result from rupture of bridging cerebral veins; however, it is difficult to demonstrate rupture of bridging cerebral vessels at autopsy.<sup>29,30</sup> Towner et al<sup>19</sup> suggested that abnormal labor was a common risk factor for hemorrhage in infants, after a retrospective review of deliveries in nulliparous women demonstrated a low incidence of intracranial hemorrhage. Pollina et al<sup>27</sup> suggested that the method of assisted delivery rather than the urgency of the delivery or dysfunctional labor is a more important variable in cranial birth injuries. Although all types of intracranial hemorrhage were more common in vacuum extraction, not all term neonate SDHs can be explained by circumferential head squeeze and overlapping sutures. This finding is particularly true because we found SDH after cesarean delivery as well. Perhaps additional forces during parturition are at work contributing to the rupturing of veins and or capillaries.

In our study, the first and second stages of labor were longer in the infants with SDH than in those without SDH. Perhaps compressive force from the uterus during the first stage, which propels the infant into the birth canal, is a causative factor. A prolonged first stage in combination with a prolonged second stage of labor may be causative in that there may not only be increased prolonged propulsive forces but also increased molding and overlapping of sutures, which may lead to failure of tensile strength of the stretched vessels. Increased pressure during the labor process may augment the intracranial venous pressures, which also may be an additional factor leading to SDH. The incidence of SDH in our study was greater in neonates with cephalohematoma and was also associated with a longer second stage of labor. The overall birth weight of neonates with SDH was also significantly higher, which may have resulted in increased circumferential pressure forces from the birth canal. Although all of these factors or a combination of these factors is plausible for the mechanism, SDH as a product of parturition has now been documented in asymptomatic neonates in multiple studies.<sup>14-23,26-28</sup>

Although most of the asymptomatic SDHs seen at MR imaging and US were in neonates delivered vaginally, 18% (4 of



**Fig 4.** Images obtained at 7 and 26 days postnatal age for follow-up of bilateral occipital SDH in a neonate with extra-axial collections. Axial T2, T1, gradient-refocused echo (GRE), and FLAIR images (left to right, top row) show CSF-intensity frontal subarachnoid collections that were present since birth. Also note a thin linear T1 hyperintense GRE hypointense bilateral posterior occipital SDH. At 26 days postnatal age (bottom row), left frontal subdural collections that do not conform to CSF signal intensity are present, consistent with spontaneous SDH. The patient had no history of trauma and had a negative evaluation for NAI.

22) of our neonates delivered by cesarean birth also had SDH. Most infants with SDH delivered by cesarean birth (75%) had a trial of labor with oxytocin administration before the cesarean delivery. This supports the proposal that SDH may be related to labor. Presumably, the neonate experienced labor during oxytocin administration before the decision for cesarean delivery.

All previous reports of SDH associated with cesarean deliveries have been in symptomatic infants. Welch and Strand<sup>31</sup> reported a series of neonates with a variety of intraparturitional intracranial hemorrhages, including 3 who had SDH and complicated cesarean deliveries either for failure to descend, forceps failure, or fetal distress. Studies reporting the incidence or prevalence of SDH in asymptomatic neonates have not reported hemorrhages in association with cesarean deliveries. The series by Whitby et al,<sup>20</sup> using low field strength, did not report SDH after cesarean delivery even when vacuum-assisted delivery was attempted. Most recently, Looney et al<sup>22</sup> reported no SDH in 23 cesarean deliveries. The delayed initial imaging at 1–5 weeks could account for the low incidence of SDH detection in that study because most SDHs in our patients resolved by 4 weeks.

The only hemorrhages detected were SDH. The location and size of the SDHs were limited. Most SDHs in our neonates were  $\leq 3$  mm. There were 2 neonates with an initial SDH  $> 3$  mm. One of these neonates had a presumed hamartoma with an occipital SDH measuring 3.3 mm. The other infant had increased extra-axial spaces and an initial occipital SDH of 4.3 mm. We believe that these infants had factors that may have predisposed them to a larger initial SDH. Like other investiga-

tors,<sup>20,22</sup> we found most SDHs were in the posterior half of the calvaria.

In our patients, supratentorial hemorrhage was more common, with 39% also having infratentorial posterior fossa hemorrhage. Both Looney et al<sup>22</sup> and Whitby et al<sup>20</sup> reported infratentorial hemorrhage alone being significantly more common. We believe that confirmatory coronal imaging was helpful in assessing supratentorial-versus-infratentorial hemorrhage. Only if we saw the blood products below the tentorium on the coronal view, would we assess the hemorrhage as infratentorial, which is depicted in Fig 2D. This finding was difficult to assess on the initial imaging series obtained within the first 72 hours of life but was confirmed on subsequent coronal T1 imaging. Also very small 1- to 2-mm supratentorial hemorrhages, which were raised as possible SDHs on initial gradient-echo sequences, were not confirmed to be SDH unless found as hyperintense on the T1 follow-up imaging. This finding on 2 subsequent imaging studies may have increased the number of overall supratentorial SDHs that were detected in comparison with that of other investigators.

In our patients, both the infratentorial and supratentorial hemorrhages were posterior in the cranium except for 1 SDH not present on the initial MR imaging ( $< 72$  hours postdelivery) but found at a follow-up study. Initially, this patient had bilateral posterior occipital SDHs, which were being followed for resolution. At 26 days of life, the patient returned for the follow-up MR imaging and was noted to have a 1-cm extra-axial left frontal collection that did not conform to CSF attenuation, consistent with a spontaneous SDH. The patient was admitted for full evaluation for nonaccidental injury to in-

clude skeletal survey, ophthalmologic examination, coagulation panel, metabolic studies, as well as social work enquiries. These investigations did not reveal any additional injuries or findings to support NAI as an etiology of the spontaneous frontal SDH (Fig 4). At a 5-month follow-up MR imaging, the left frontal SDH resolved; however, the subarachnoid space remained prominent in this patient. This finding suggests that though not typical in a neonate, prominent extra-axial space is a predisposing factor for SDHs as has been reported by other authors.<sup>32-35</sup>

Although SDH along the interhemispheric fissure, parafalcine in location, is widely associated with NAI, we would suggest that the pattern and location of SDH alone should not be used to make a distinction between SDH due to NAI or birth injury. In the pilot study of Holden et al,<sup>21</sup> there is a description and illustration of an interhemispheric SDH in an asymptomatic neonate. The posterior location of the SDH is generally common to reports of asymptomatic SDH, including our study. Interhemispheric SDHs have been previously reported in accidental trauma as well as in birth trauma and are no longer considered specific for the type or mechanisms of injury.<sup>36-38</sup> We noted that the SDH was in a more dependent position on follow-up imaging regardless of the location of the initial hemorrhage and propose that this is likely due to the recommended practice of the American Academy of Pediatrics of placing infants on their backs for sleep.<sup>39</sup> When lying supine, gravity may account for the posterior locations of the SDH, indicating communication of the subdural space.

Although US could detect approximately half of the SDHs, the area imaged was limited to the lateral posterior fossa via the mastoid fontanelle. Midline imaging of the posterior fossa was not routinely performed and thus the utility of US for detection of SDH may have been underestimated. Still, no supratentorial SDHs were detected on US. Clearly, MR imaging is more sensitive than US for the detection of SDH.

The 2-year follow-up of the infants with SDH was reassuring because all (100%) of the 43 children with documented follow-up had no gross motor delay. In our study population, 6 (14%) of the children were noted to have speech delay, which is similar to the known incidence in the general population.<sup>40</sup> The 1 boy being evaluated for a possible autistic spectrum disorder is not unexpected because autism is currently reported to have a prevalence of 1:150, with the prevalence in boys reported as high as 1:80.<sup>41-43</sup> Normal findings on clinical follow-up are reassuring but are limited because there is no baseline for comparison in the study design. We compared normal development with that in children who met the criteria for the Denver Developmental Screening Test, which lists expected milestones at each chronological age through 5 years. This expected development is our norm when assessing children in our clinic. Children not meeting expectations are marked as having a delay and are referred for further evaluation to a subspecialty clinic.

One limitation of our study was the evaluation for rebleeding of SDH, which has been reported in the literature. Rebleeding may present either with or without clinical symptoms.<sup>44</sup> Although none of our infants re-presented clinically with an SDH rebleed, the subclinical incidence of rebleeding in our population was not studied because none of the infants were reimaged after 3 months of age. Normal development on

clinical examination is reassuring, indicating that major rebleeding did not take place.

Another limitation in our study included the need to change MR imaging sequences and timing. We found early on that SDH was isointense to gray matter and intermittently difficult to see on initial imaging within the first 72 hours of life. The SDH was seen as lobular low signal intensity with blooming on the gradient-echo imaging. The MR imaging findings were considered positive for SDH if the positive gradient-echo sequence was confirmed on subsequent T1 imaging with hyperintense signal intensity by 3–7 days. To improve MR imaging for maximal sensitivity, we changed the original T1-weighted fast spin-echo imaging sequence, spoiled gradient-recalled (SPGR), to a spin-echo T1 sequence. The initial study performed with fast spin-echo SPGR was recategorized from positive for extra-axial blood products to negative for extra-axial blood products if findings of the follow-up study performed at 3–7 days were negative. The recategorization from positive to negative for SDH may have underestimated the actual number of SDHs in our neonate population. The SDH on initial MR imaging may have been very small and resolved by the second MR imaging between 3–7 days of life. Therefore, the true incidence of SDH may be slightly higher than that reported in this study. The initial follow-up time interval was also changed from 3 days to 5–7 days to account for the signal-intensity conversion changes. The initial follow-up at 3 days did not consistently demonstrate T1 hyperintense signal intensity. We, therefore, lengthened the interval to 5–7 days to allow blood products to change to T1 hyperintense, consistent with methemoglobin. This variability of the time-interval imaging may have masked the actual timeframe in which fetal hemoglobin changes signal-intensity characteristics.

Another limitation to the study is the lack of follow-up imaging in some patients. Follow-up imaging was only completed in 18 of 46 infants with SDH. We were surprised to find that, despite the parent knowing that their infant had SDH, follow-up appointments were often missed after the first 2-week follow-up MR imaging and US. Selection bias of the patient population is also a potential limitation. We relied on a random selection process limited by our ability to obtain written consent from the parents for our sample population.

## Conclusion

SDH is a common result of parturition and may be seen after vaginal and cesarean delivery. MR imaging is more sensitive than US for the detection of SDH. The hemorrhages seen in asymptomatic term neonates are limited in size and location. SDH after 1 month of age is unlikely to be birth-related.

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## References

- Barkovich AJ. *Pediatric Neuroimaging*. 3rd ed. New York: Lippincott Williams & Wilkins; 2000
- Kleinman PK. *Diagnostic Imaging of Child Abuse*. 2nd ed. Toronto, Ontario, Canada: Mosby; 1998
- Billmire ME, Myers PA. **Serious head injury in infants: accident or abuse?** *Pediatrics* 1985;75:340–42
- Hoskote A, Richards P, Anslow P, et al. **Subdural haematoma and non-accidental head injury in children.** *Childs Nerv Syst* 2002;18:311–17. Epub 2002 Jun 26
- Jayawant S, Rawlinson A, Gibbon F, et al. **Subdural haemorrhages in infants: population-based study.** *BMJ* 1998;317:1558–61
- Caffey J. **Multiple fractures in the long bones of infants suffering from subdural hematoma.** *Am J Roentgenol* 1946;56:163–73
- Caffey J. **On the theory and practice of shaking infants: its potential residual effects of permanent brain damage and mental retardation.** *Am J Dis Child* 1972;124:161–69
- Caffey J. **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.** *Pediatrics* 1974;54:396–403
- Hadley MN, Sonntag VK, Reigate HL, et al. **The infant whiplash-shake injury syndrome: a clinical and pathological study.** *Neurosurgery* 1989;24:536–40
- Kempe CH, Silverman FN, Steele BF, et al. **The battered-child syndrome.** *JAMA* 1962;181:17–24
- Duhaime AC, Christian CW, Rorke LB, et al. **Nonaccidental head injury in infants: the “shaken-baby syndrome.”** *N Engl J Med* 1998;338:1822–29
- Barnes PD, Krasnokutsky M. **Imaging of the central nervous system in suspected or alleged nonaccidental injury, including the mimics.** *Top Magn Reson Imaging* 2007;18:53–74
- Feldman KW, Bethel R, Shugerman RP, et al. **The cause of infant and toddler subdural hemorrhage: a prospective study.** *Pediatrics* 2001;108:636
- Huang CC, Shen EY. **Tentorial subdural hemorrhage in term newborns: ultrasonographic diagnosis and clinical correlates.** *Pediatr Neurol* 1991;7:171–77
- Hayashi T, Hashimoto T, Fukuda S, et al. **Neonatal subdural hematoma secondary to birth injury: clinical analysis of 48 survivors.** *Childs Nerv Syst* 1987;3:23–29
- Hanigan WC, Powell FC, Miller TC, et al. **Symptomatic intracranial hemorrhage in full-term infants.** *Child Nerv Syst* 1995;11:698–707
- Huang AH, Robertson RL. **Spontaneous superficial parenchymal and leptomeningeal hemorrhage in term neonates.** *AJNR Am J Neuroradiol* 2004;25:469–75
- Miall LS, Cornette LG, Tanner SF, et al. **Posterior fossa abnormalities seen on magnetic resonance brain imaging in a cohort of newborn infants.** *J Perinatol* 2003;23:396–40
- Towner D, Castro MA, Eby-Wilkens E, et al. **Effect of mode of delivery in nulliparous women on neonatal intracranial injury.** *N Engl J Med* 1999;341:1709–14
- Whitby EH, Griffiths PD, Rutter S, et al. **Frequency and natural history of subdural haemorrhages in babies and relation to obstetric factors.** *Lancet* 2004;363:846–51
- Holden KR, Titus MO, Van Tassel P. **Cranial magnetic resonance imaging examination of normal term neonates: a pilot study.** *J Child Neurol* 1999;14:708–10
- Looney CB, Smith JK, Merck LH, et al. **Intracranial hemorrhage in asymptomatic neonates: prevalence on MR images and relationship to obstetric and neonatal risk factors.** *Radiology* 2006;242:535–41. Epub 2006 Dec 19
- Tavani F, Zimmerman RA, Clancy RR, et al. **Incidental intracranial hemorrhage after uncomplicated birth: MRI before and after neonatal heart surgery.** *Neuroradiology* 2003;45:253–58. Epub 2003 Mar 15
- Batshaw ML, Pellegrino L, Roizen NJ, eds. *Children with Disabilities*. 5th ed. Baltimore: Paul H. Brooks Publishing Co; 2002
- Frankenburg WK, Dodds J, Archer P, et al. **The Denver II: a major revision and restandardization of the Denver Developmental Screening Test.** *Pediatrics* 1992;89:91–97
- Doward W, Sgouros S. **Acute subdural haematomas following ventouse-assisted delivery.** *Pediatr Neurosurg* 2001;35:335
- Pollina J, Dias MS, Li V, et al. **Cranial birth injuries in term newborn infants.** *Pediatr Neurosurg* 2001;35:113–19
- Menezes AH, Smith DE, Bell WE. **Posterior fossa hemorrhage in the term neonate.** *Neurosurg* 1983;13:452–56
- Maxeiner H. **Detection of ruptured cerebral bridging veins at autopsy.** *Forensic Sci Int* 1997;89:103–10
- Stein KM, Ruf K, et al. **Representation of cerebral bridging veins in infants by postmortem computed tomography.** *Forensic Sci Int* 2006;163:93–101. Epub 2005 Dec 20
- Welch K, Strand R. **Traumatic parturition intracranial hemorrhage.** *Dev Med Child Neurol* 1986;28:156–64
- McNeely PD, Atkinson JD, Saigal G, et al. **Subdural hematomas in infants with benign enlargement of the subarachnoid spaces are not pathognomonic for child abuse.** *AJNR Am J Neuroradiol* 2006;27:1725–28
- Amodio J, Spector V, Pramanik B, et al. **Spontaneous development of bilateral subdural hematomas in an infant with benign infantile hydrocephalus: color Doppler assessment of vessels traversing extra-axial spaces.** *Pediatr Radiol* 2005; 35:1113–17. Epub 2005 May 19
- Pittman T. **Significance of a subdural hematoma in a child with external hydrocephalus.** *Pediatr Neurosurg* 2003;39:57–59
- Ravid S, Maytal J. **External hydrocephalus: a probable cause for subdural hematomas in infancy.** *Pediatr Neurol* 2003;28:139–42
- Tung GA, Kumar M, Richardson RC, et al. **Comparison of accidental and non-accidental traumatic head injury in children on noncontrast computed tomography.** *Pediatrics* 2006;118:626–33
- Vinchon M, Noulé N, Tchofo PJ, et al. **Imaging of head injuries in infants: temporal correlates and forensic implications for the diagnosis of child abuse.** *J Neurosurg* 2004;101(1 suppl):44–52
- Steinbok P, Singhal A, Poskitt K, et al. **Early hypodensity on computed tomographic scan of the brain in an accidental pediatric head injury.** *Neurosurgery* 2007;60:689–94, discussion 694–95
- American Academy Of Pediatrics Task Force on Sudden Infant Death Syndrome. **The changing concept of sudden infant death syndrome: diagnostic coding shifts, controversies regarding the sleeping environment, and new variables to consider in reducing risk.** *Pediatrics* November 10, 2005;116:1245–55
- Feldman HM. **Evaluation and management of language and speech disorders in preschool children.** *Pediatr Rev* 2005;26:131–42
- Autism and Developmental Disabilities Monitoring Network Surveillance Year 2000 Principal Investigators; Centers for Disease Control and Prevention. **Prevalence of autism spectrum disorders: autism and developmental disabilities monitoring network, six sites, United States, 2000.** *MMWR Surveill Summ* 2007;56:1–11
- Autism and Developmental Disabilities Monitoring Network Surveillance Year 2002 Principal Investigators; Centers for Disease Control and Prevention. **Prevalence of autism spectrum disorders: autism and developmental disabilities monitoring network, 14 sites, United States, 2002.** *MMWR Surveill Summ* 2007;56:12–28
- Autism and Developmental Disabilities Monitoring Network Surveillance Year 2002 Principal Investigators; Centers for Disease Control and Prevention. **Evaluation of a methodology for a collaborative multiple source surveillance network for autism spectrum disorders: autism and developmental disabilities monitoring network, 14 sites, United States, 2002.** *MMWR Surveill Summ* 2007;56:12–28.
- Hymel KP, Jenny C, Block RW. **Intracranial hemorrhage and rebleeding in suspected victims of abusive head trauma: addressing the forensic controversies.** *Child Maltreat* 2002;7:329–48

# Vitreoretinal traction is a major factor in causing the haemorrhagic retinopathy of abusive head injury? – No

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For the past 20 years, vitreoretinal traction has been held to be a major mechanism for the generation of retinal haemorrhage in non-accidental injury in infancy. The presence of circinate macular folds, and a lesion termed ‘traumatic retinoschisis’ have been proposed as indicating severe vitreoretinal traction due to shaking, and by implication, only to result from the application of extreme violence. Some recent evidence, and clinical experience of the behaviour of partially detached vitreous, casts doubt on this hypothesis and this has implications for the degree of certainty with which the presence of retinal haemorrhage and circinate macular folds can be used as a marker for extreme violence done to an infant.

Child abuse is likely to be as old as humanity, but only recently has the combination of subdural and retinal haemorrhages with encephalopathy in infancy been recognised as being due, in some, if not all, cases, to inflicted trauma. The mechanism by which the trauma gives rise to the clinical findings remains the subject of hypothesis and conjecture.

Subdural haemorrhage (SDH) in abused children was first described by Tardieu.<sup>1</sup> Over 80 years later, Caffey described unexplained fractures of the long bones and SDH in 6 children,<sup>2</sup> but did not overtly cite inflicted trauma as the cause. It was a further 16 years before Kempe coined the term ‘Battered Child Syndrome’ to explain such findings.<sup>3</sup>

The first description of retinal haemorrhage (RH) in abused children was by Gilkes and Mann.<sup>4</sup> They suggested that RH arose as a result

of a rise in the intracranial and the intraocular venous pressure, which could arise because of chest compression while the child was being shaken. Further descriptions of RH in abused children were given by Harcourt and Hopkins, who also described the visual impairment which could result not only because of ocular but also cerebral injury.<sup>5,6</sup>

Guthkelch, a British neurosurgeon, first postulated that the cause of SDH in Battered Child Syndrome was a shaking injury, causing rotational forces within the cranium which disrupted vessels bridging the subdural space.<sup>7</sup> He commented that, at the time, a ‘good shaking’ was considered by many British parents socially more acceptable and less dangerous than a blow to the head.

In 1974, Caffey coined the term ‘Whiplash Shaken Infant Syndrome’<sup>8</sup> and postulated that many battered babies were really shaken babies. In commenting on the pathogenic significance of ocular lesions in these children, Caffey agreed with other authors of the time that ‘some of the affected infants are the victims of over vigorous manipulations (sic), not battering.’ He went on to comment that: ‘The pathogenesis of retinal haemorrhages in the manual WLS (whiplash shaking) of infants and children cannot be evaluated satisfactorily without a consideration of the incidence, nature and persistence of idiopathic retinal haemorrhages of the newborn.’ before going on to cite an increase in blood viscosity and polycythaemia as the major causal factors.

The concept that retinal haemorrhage arose in shaking injuries because of vitreous traction on the retina was first proposed by Greenwald *et al* in 1986.<sup>9</sup> They coined the term ‘traumatic retinoschisis’ to refer to the appearances described in their series, which consisted of five

children with features compatible with inflicted trauma (although criminal prosecution occurred in only one case). Cystic retinal lesions, partially or completely filled with blood, were described at the posterior pole in four cases, in two of which cysts developed (in one case after clearance of delayed vitreous haemorrhage) after an initial evaluation had shown retinal haemorrhage only. All five cases had reduced or electronegative ERGs in at least one eye, indicating damage to the inner layers of the retina. They proposed that back and forth movement of the lens during a shaking episode transferred tractional forces through the vitreous to the posterior pole of the eye, causing splitting of retinal layers. Further descriptions of circinate perimacular folds, considered to result from vitreoretinal traction attributable to shaking, followed.<sup>10</sup>

Pathological support for the vitreous traction theory came from papers by Massicote *et al*<sup>11</sup> and Green *et al*.<sup>12</sup> Massicote *et al* noted partial detachment of the vitreous except at the apices of retinal folds—confirming, in their view, the role of vitreous traction in the formation of folds.

Green *et al* found subhyaloid haemorrhage and retinal detachment to be most frequent at the retinal periphery and around the optic nerve—the sites of the strongest vitreoretinal adhesion. They did not, however, describe retinoschisis.

Massicote *et al* also noted massive retinal haemorrhage at the vitreous base in one of their cases, and described a haemorrhagic cavity beneath the internal limiting membrane in one of their patients, which they described as schitic. In fact, despite the continued use of the term ‘traumatic retinoschisis’, true retinoschisis, as opposed to separation of the internal limiting membrane, has never been described pathologically due to inflicted head trauma in children.

In contrast, Emerson *et al*,<sup>13</sup> found retinal haemorrhage to be more common in the mid periphery of the retina rather than at the vitreous base. Furthermore, Emerson *et al* did not find vitreous detachment peripheral to macular folds and cast doubt on vitreomacular traction as the aetiology of circumferential macular fold formation. They proposed that venous leakage led to the formation of a haemorrhagic schisis cavity, which expanded, pulling surrounding retina centripetally into a circumferential fold.

In other respects, it seems unlikely that shaking of an infant would result in significant vitreoretinal traction, or that this would lead to retinal haemorrhage.

Clinical experience of the behaviour of partially detached vitreous, and of vitrectomy surgery, where attached vitreous may have to be peeled away from the retinal surface, suggests that vitreous traction on the retina causes retinal tears rather than haemorrhage.

Furthermore, the eye is ‘designed’ to rotate, for example during saccadic eye movements, during which angular

accelerations of up to 700° per second may be achieved, and the vestibulo-ocular reflex is likely to mitigate the effects of rotation of the head on the eye.<sup>14</sup> Retinal haemorrhages are not observed after saccadic eye movements, nor in cases of nystagmus, or opsoclonus. Rotational forces are intentionally applied to the eye by some surgeons during strabismus surgery—the ‘spring back balance test’ of Jampolsky,<sup>15</sup> without causing haemorrhage.

Neither does vitreoretinal traction explain the frequent finding of RH (and when looked for, SDH<sup>16</sup>) in normal neonates, nor why the frequency of RH is significantly increased (reaching up to 75%) after Ventouse delivery,<sup>17,18</sup> indicating a role for venous congestion by suctional forces transmitted through the fontanelle.

Does the precise mechanism whereby retinal haemorrhage occurs, in cases of inflicted trauma, matter? It is clear that inflicted trauma can give rise to subdural haemorrhage, encephalopathy, retinal haemorrhage, subhyaloid and sub internal limiting membrane haemorrhage, and circinate macular folds; and it is very likely that these findings can arise from shaking an infant without any impact or injury. However without a clearer understanding of the processes involved in the pathogenesis of these findings, it remains impossible, despite the assertions of some authors,<sup>19</sup> to be certain that all infants demonstrating them have been the victims of attempted, or actual, murder.

### Conflict of interest

The author has been a paid expert witness in court cases related to child abuse.

### References

- 1 Tardieu A. Etude médico-légale sur les services et mauvais traitements exercés sur des enfants. *Ann Hyg Publ Med Leg* 1860; **13**: 361–398.
- 2 Caffey J. Multiple fractures in the long bones of infants suffering from subdural hematoma. *Amer J Roentgen* 1946; **56**: 163.
- 3 Kempe C, Silverman F, Steele B, Droegmueller W, Silver H. The battered-child syndrome. *JAMA* 1962; **181**: 17–24.
- 4 Gilkes MJ, Mann TP. Fundi of battered babies. *Lancet* 1967; (2): 468–469.
- 5 Harcourt B, Hopkins D. Ophthalmic manifestations of the battered-baby syndrome. *Br Med J* 1971; **3**(771): 398–401.
- 6 Harcourt B, Hopkins D. Permanent chorio-retinal lesions in childhood of suspected traumatic origin. *Trans Ophthalmol Soc U K* 1973; **93**: 199–205.
- 7 Guthkelch A. Infantile subdural haematoma and its relationship to whiplash injuries. *BMJ* 1971; **ii**: 430–431.
- 8 Caffey J. 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. *Pediatrics* 1974; **54**(4): 396–403.

- 9 Greenwald MJ, Weiss A, Oesterle CS, Friendly DS. Traumatic retinoschisis in battered babies. *Ophthalmology* 1986; **93**(5): 618–625.
- 10 Gaynon M, Koh K, Marmor M, Frankel L. Retinal folds in the shaken baby syndrome. *Am J Ophthalmol* 1988; **106**: 423–425.
- 11 Massicotte SJ, Folberg R, Torczynski E, Gilliland M, Luckenbach MW. Vitreoretinal traction and perimacular retinal folds in the eyes of deliberately traumatized children. *Ophthalmology* 1991; **98**(7): 1124–1127.
- 12 Green M, Lieberman G, Milroy C, Parsons M. Ocular and cerebral trauma in non-accidental injury in infancy: underlying mechanisms and implications for paediatric practice. *Br J Ophthalmol* 1996; **80**: 282–287.
- 13 Emerson M, Jakobs E, Green W. Ocular autopsy and histopathologic features of child abuse. *Ophthalmology* 2007; **114**(7): 1384–1394.
- 14 Wong A. *Eye Movement Disorders*. Oxford University Press, US, 2008.
- 15 Pittar G. Multiple operations for strabismus. *Clin Exp Ophthalmol* 2008; **8**(1): 15–19.
- 16 Whitby E, Griffiths P, Rutter S, Smith MF, Sprigg A, Ohadike P *et al*. Frequency and natural history of subdural haemorrhages in babies and relation to obstetric factors. *Lancet* 2003; **362**: 846–851.
- 17 Emerson M, Pieramici D, Stoessel K, Berreen J, Gariano R. Incidence and rate of disappearance of retinal haemorrhages in newborns. *Ophthalmology* 2001; **108**(1): 36–39.
- 18 Hughes L, May K, Talbot J, Parsons M. Incidence, distribution and duration of birth-related retinal hemorrhages: a prospective study. *J AAPOS* 2006; **10**(2): 102–106.
- 19 Levin A. Retinal haemorrhages and child abuse. In: David T (ed). *Recent Advances in Paediatrics*. Edinburgh Churchill Livingstone, 2000; 151–219.

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