

12-18-2011

Unexplained Fractures in Infants and Child Abuse: The Case for Requiring Bone-Density Testing Before Convicting Caretakers

Matt Seeley

Follow this and additional works at: <https://digitalcommons.law.byu.edu/lawreview>



Part of the [Family Law Commons](#), and the [Medical Jurisprudence Commons](#)

Recommended Citation

Matt Seeley, *Unexplained Fractures in Infants and Child Abuse: The Case for Requiring Bone-Density Testing Before Convicting Caretakers*, 2011 BYU L. Rev. 2321 (2011).

Available at: <https://digitalcommons.law.byu.edu/lawreview/vol2011/iss6/13>

This Comment is brought to you for free and open access by the Brigham Young University Law Review at BYU Law Digital Commons. It has been accepted for inclusion in BYU Law Review by an authorized editor of BYU Law Digital Commons. For more information, please contact hunterlawlibrary@byu.edu.

Unexplained Fractures in Infants and Child Abuse: The Case for Requiring Bone-Density Testing Before Convicting Caretakers

I. INTRODUCTION

Imagine that you are a young parent taking your four-month-old son to a routine checkup. During the examination, the pediatrician notices a bruise on his right leg. Her visage subtly changes as she closely examines the bruise. She informs you that she has reason to believe that your baby may have been physically abused and that you will have to immediately take him to the hospital for further testing. You are a bit insulted that the doctors would even think it was possible that you would hurt your baby, but you are confident that carefully researched, scientific tests that will be conducted at the hospital will quickly dispel this misunderstanding.

You wring your hands as you wait for the result of the x-ray at the hospital. A doctor then approaches you and says that your baby has a large number of fractures in his legs and his ribs. Before you can ask, the doctor explains that these types of fractures in a baby of this age suggest, with near certainty, child abuse. He asks you how these fractures occurred. You are so aghast and surprised at the situation that you stutter as you say that you don't know. You suggest that he must have some type of condition that makes his bones break very easily. This suggestion is met with dismissive incredulity. No, the doctor explains. The x-rays didn't show any signs of such a condition. These types of fractures indicate that your baby has been abused. A child protection agent says that your baby will have to be taken into protective custody. Your baby will be placed with foster parents. You and your spouse will soon be charged with felony assault. You and your spouse will be labeled child abusers by incredulous authorities who simply shake their heads when you insist that your child's fractures must have been caused by a medical condition. In short, your life will be turned upside down.

How likely is this scenario? Prevalent beliefs in the medical and legal communities about unexplained infantile fractures actually make this type of scenario more likely to occur than is necessary in order to protect children who are legitimately abused. The scenario

you have just read is actually not hypothetical; it describes a case that occurred several years ago.¹ Fortunately, this case eventually had a happy ending because a simple, commonly available bone-density test was performed. The test demonstrated that the child had abnormally low bone density of unknown origin that predisposed him to fractures.²

Such tests, however, are seldom administered in alleged child abuse cases involving unexplained fractures.³ Many expert medical witnesses and prosecutors believe that child abuse can be diagnosed using only ordinary x-rays.⁴ This perception fails to account for the serious inaccuracy of ordinary x-rays for measuring bone density⁵ and collagen integrity.⁶ It also fails to incorporate a modern understanding of many conditions that can cause bone fragility in infants—an understanding that has evolved substantially since the publication of the sixty-five-year-old study that first suggested that unexplained infantile fractures indicated child abuse with near certainty.⁷

This Comment highlights the problems inherent in making a diagnosis of child abuse based solely on findings of unexplained fractures in x-rays and ultimately proposes a solution to the problem that will help protect innocent children and caretakers alike: a statute requiring that bone-density tests be performed when the prosecution

1. *In re* Stephan H., No. L15-CP00-007572-A, 2002 WL 31083579, at *1–5 (Conn. Super. Ct. Aug. 23, 2002).

2. *Id.* at *12.

3. See, e.g., *In re* Erik R., No. B215474, 2010 Cal. App. Unpub. LEXIS 1712, at *2–3 (Cal. Ct. App. March 10, 2010); *In re* A.B., 999 A.2d 36, 39–40 (D.C. 2010); *In re* A.R., 651 S.E.2d 467, 468–469 (Ga. Ct. App. 2007); *In re* J.V., 526 S.E.2d 386, 389 (Ga. Ct. App. 1999); *State v. M.F.*, No. 06-05-0945, 2009 WL 2192231, at *3 (N.J. Super. Ct. App. Div. July 24, 2009); *N.J. Div. of Youth and Family Servs. v. F.H.*, 914 A.2d 318, 325 (N.J. Super. Ct. App. Div. 2007); *In re* Julia BB., 837 N.Y.S.2d 398, 402 (N.Y. App. Div. 2007); *In re* Gaven R., No. M2005-01868-COA-R3-CV, 2007 WL 2198288, at *4 (Tenn. Ct. App. July 23, 2007).

4. See D.L. Griffiths & F.J. Moynihan, *Multiple Epiphyseal Injuries in Babies* (“Battered Baby” Syndrome), 2 BRIT. MED. J. 1558, 1560 (1963).

5. See generally William H. Harris & Robert P. Heaney, *Skeletal Renewal and Metabolic Bone Disease*, 280 NEW ENG. J. MED. 193 (1969).

6. Anne Johnstone, *When Doctors Disagree*, THE HERALD (Glasgow, Scotland), Jan. 26, 2001, at 23, available at <http://www.heraldscotland.com/sport/spl/aberdeen/when-doctors-disagree-1.200918>.

7. See John Caffey, *Multiple Fractures in the Long Bones of Infants Suffering from Chronic Subdural Hematoma*, 56 AM. J. ROENTGENOLOGY 163 (1946).

wishes to use unexplained infantile fractures as evidence of child abuse.

This Comment proceeds as follows. Part II discusses the problem of child abuse and the strategies used to combat it, as well as the reasons why more effective strategies are often not possible. It then discusses how unavoidable weaknesses in these strategies led to a widespread misappraisal of the degree of certainty with which abuse could be inferred in cases where infants have a set of symptoms once thought to be pathognomonic⁸ for abusive shaking. Part III then outlines critical problems with some of the initial studies that gave rise to the commonly held belief that unexplained infantile fractures indicate child abuse with near certainty. Also included in this Part is a brief description of a number of medical conditions now known to cause bone fragility in infants, including the controversial diagnosis of Temporary Brittle-bone Disease. Part IV follows by explaining why unexplained fractures, even in combination with the intracranial bleeding once thought to be caused only by shaking, may not indicate child abuse with as high a degree of certainty as is commonly believed because of problematic assumptions of independence. This Part also highlights a case by the California Supreme Court that explains how unsupported assumptions of independence can be improperly prejudicial to justice. Parts V and VI conclude by suggesting that requiring a commonly available bone-density test in cases involving unexplained fractures would both decrease the likelihood of convicting innocent caretakers and increase the likelihood of convicting guilty ones.

II. THE PROBLEM OF CHILD ABUSE

Child abuse is common⁹ and has far-reaching effects on children's physical health and happiness.¹⁰ It also affects society at large because people who are abused as children are more likely to

8. For a definition of this term, see HAROLD C. SOX ET AL., *MEDICAL DECISION MAKING* 17 (1988) (“[A] pathognomonic finding establishes a diagnosis. A pathognomonic finding occurs in only one disease.”).

9. CINDY L. MILLER-PERRIN & ROBIN D. PERRIN, *CHILD MALTREATMENT: AN INTRODUCTION* 8 (2d ed. 2007) (stating that there were approximately 900,000 cases of child maltreatment confirmed by child protective services in the United States in 2003).

10. Kathleen Kendall-Tackett, *The Health Effects of Childhood Abuse: Four Pathways by Which Abuse Can Influence Health*, 26 *CHILD ABUSE & NEGLECT* 715, 715–16 (2002).

become criminals¹¹ or abusers¹² themselves. Furthermore, from a moral standpoint, one of the very purposes of our law is to protect the innocent—especially those who are unable to protect themselves. Thus, our society has both a vested interest in preventing child abuse as well as a moral duty to do so.

In order to combat the plague of child abuse, Congress passed the Child Abuse Prevention and Treatment Act in 1974, which allocated funds for the identification, treatment, and prevention of child abuse.¹³ By 1978, all fifty states had passed statutes mandating that physicians report suspected cases of child abuse.¹⁴ Most of these statutes also required teachers, school officials, and social workers to report suspected cases.¹⁵ A substantial minority of these states even required “any person” to report.¹⁶ In addition, every state provided immunity from civil and criminal liability to anyone who reported suspected child abuse in good faith, and, in fifteen states, good faith was a rebuttable presumption.¹⁷ A majority of states even passed legislation that imposed criminal penalties on people who had the duty to report suspected child abuse but failed to do so.¹⁸ Thus, state and federal laws have been carefully structured to incentivize reporting and discourage any failure to report.

Child abuse, however, is often difficult to identify. Abusers seldom testify against themselves voluntarily and cannot be compelled to do so.¹⁹ Since abusers are often family members, the victims and witnesses of some forms of domestic abuse are often reluctant to testify against them.²⁰ Furthermore, not all cultures have

11. Cathy Spatz Widom, *Child Abuse, Neglect, and Violent Criminal Behavior*, 27 *CRIMINOLOGY* 251, 251 (1989).

12. John F. Knutson, *Psychological Characteristics of Maltreated Children: Putative Risk Factors and Consequences*, 46 *ANN. REV. PSYCHOL.* 401, 419 (1995).

13. Brian G. Fraser, *A Glance at the Past, a Gaze at the Present, a Glimpse at the Future: A Critical Analysis of the Development of Child Abuse Reporting Statutes*, 54 *CHI.-KENT L. REV.* 641, 648 (1978).

14. *Id.* at 657.

15. *Id.* at 657–58.

16. *Id.* at 658.

17. *Id.* at 663–64 (noting that fifteen states provide those that report child abuse a rebuttable presumption of good faith).

18. *Id.* at 665.

19. U.S. CONST. amend. V.

20. See AM. BAR ASS'N, *THE STATE OF CRIMINAL JUSTICE 2007–2008*, at 334 (Victor Streib ed., 2008) (explaining why elderly victims of domestic abuse are often reluctant to testify against abusers who are family members.)

the same definition of abuse.²¹ As a result, it may not always be clear to victims and witnesses when reporting is appropriate. Furthermore, in the case of an abused baby, the victim may not yet understand spoken and written language and is therefore not even capable of testifying.

As a result, circumstantial evidence of child abuse must be used—even if it is not always corroborated by direct evidence—because it is often the only evidence in such cases that can be reasonably obtained.²² To do otherwise would risk letting a large swath of abusive conduct go unpunished and potentially leave children in abusive situations.

Physical child abuse can result in bruising, broken bones, head injuries, and burns.²³ By themselves, however, such injuries are not always indicative of abuse. Most childhood injuries do not result from abuse, and some accidents “may produce injuries that are not characteristically seen from accidental causes.”²⁴ In other words, the population of all children with such injuries undoubtedly includes many children who have been physically abused, but it also includes a substantial number of children who have not. With this reality in mind, a number of medical researchers have earnestly sought to identify certain types of injuries and circumstances that are highly correlated with child abuse.²⁵

21. Tamar Cohen, *Israel*, in CHILD ABUSE: A GLOBAL VIEW 85, 86 (Beth M. Schwartz-Kenney et al. eds., 2001).

22. LAW REFORM COMM’N OF SASK., TENTATIVE PROPOSALS FOR COMPENSATION OF ACCUSED ON ACQUITTAL 21 (1987).

23. JONATHAN S. OLSHAKER ET AL., FORENSIC EMERGENCY MEDICINE 158–165 (2d ed. 2007).

24. Nancy D. Kellogg & Comm. on Child Abuse & Neglect, *Evaluation of Suspected Child Physical Abuse*, 119 PEDIATRICS 1232, 1235 (2007).

25. See, for example, the following studies: William A. Altemeier et al., *Prediction of Child Abuse: A Prospective Study of Feasibility*, 8 CHILD ABUSE & NEGLECT 393 (1984) (aiming to predict the likelihood that children will be abused based on familial circumstances measured before birth); Shervin R. Dashti et al., *Current Patterns of Inflicted Head Injury in Children*, 31 PEDIATRIC NEUROSURGERY 302 (1999) (aiming to identify the signs of inflicted head injuries); A.M. Kemp et al., *Diagnosing Physical Abuse Using Bayes’ Theorem: A Preliminary Study*, 7 CHILD ABUSE REV. 178 (1998) (aiming to identify patterns of bruising that are indicative of abuse); Peter Worlock et al., *Patterns of Fractures in Accidental and Non-accidental Injury in Children: A Comparative Study*, 293 BRIT. MED. J. 100 (1986) (aiming to identify which types of fractures are indicative of abuse).

A. The Approach Used to Identify Injuries Associated with Child Abuse

Childhood injuries can be placed along a spectrum in terms of the likelihood that they were caused by abuse. At one end are injuries considered relatively unlikely to have been caused by abuse such as a single, superficial scratch on a child's left forearm in the shape of the child's own right thumbnail. At the other end are injuries often referred to as being pathognomonic²⁶ for child abuse—that is, injuries for which the only possible cause is child abuse. Examples include slap marks in the shape of an adult hand or, in an extreme hypothetical, bruises in the exact imprint of a trademark for brass knuckles. Where pathognomonic injuries are present, law enforcement is generally quick to intervene on behalf of the child and prosecute the abuser.

In the prosecution of child abuse, the injuries themselves are often the principle evidence used to meet the State's burden of proof.²⁷ As noted above, because it is so difficult to find witnesses willing to testify, showcasing physical injuries may often be the only option available to bring abusers to justice. In fact, some courts have even gone so far as to take a *res ipsa loquitur* approach by permitting an inference of abusive neglect when a child's condition "is such as in the ordinary course of things does not happen if the parent who has the responsibility and control of an infant is protective and non-abusive."²⁸ As a result, it is imperative that medical researchers properly assess whether injuries are truly pathognomonic for child abuse. Is the presence of the injury itself proof beyond a reasonable doubt of child abuse, or could the injury be caused by other factors? Ideally, an accurate appraisal would correctly identify when a child's injuries are the result of abuse and serve as evidence against the abuser. In contrast, an inaccurate appraisal could lead to a second tragic event: the false accusation and imprisonment of an innocent caretaker who may already be suffering immense grief because of the child's injuries.

26. See SOX ET AL., *supra* note 8.

27. See *In re Erik R.*, No. B215474, 2010 Cal. App. Unpub. LEXIS 1712, at *2–3 (Cal. Ct. App. March 10, 2010).

28. Allyson B. Levine, *Failing to Speak for Itself: The Res Ipsa Loquitur Presumption of Parental Culpability and Its Greater Consequences*, 57 BUFF. L. REV. 587, 590 (2009) (quoting *In re S.*, 259 N.Y.S.2d 164, 165 (N.Y. Fam. Ct. 1965)) (internal quotation marks omitted).

B. The Inherent Difficulty in Identifying Pathognomonic Child-Abuse Injuries

A number of cases have shown that appraisal of pathognomoncity is often not a trivial task, even when very damning injuries occur. Multiple bruises and apparent cigarette burns, for example, can be caused by conditions unrelated to abuse.²⁹ Even injuries to a young girl's genitalia are not always the result of abuse.³⁰ Thus, careful scientific studies are necessary to evaluate the likelihood of alternative causes for almost any injury that might seem to indicate abuse at first blush.

There is, however, a major obstacle that prevents scientific studies about child abuse from being performed in an ideal way. In an ideal scientific study, researchers develop a hypothesis and test it against results that can be measured by some means that does not require the hypothesis to be accurate in order to accurately measure the results. A scientific study on child abuse is difficult to fit into this framework because obtaining absolute "proof" that an injury resulted from child abuse is inherently difficult.³¹

Ethically, doctors cannot bus in a statistically significant number of children; divide them into various control and experimental groups; whip, shake, bludgeon, molest, and otherwise abuse the children in the abuse experimental groups; deliberately cause "accidental" injuries to the control group; and then carefully study what distinguishes the "accidental" injuries from the abusive ones. Because this cannot (and must not) be done, child-abuse researchers must, in most cases, conclude that abuse did or did not occur based on some indicia other than direct observation. Video evidence would be ideal, but it is seldom available. Other indicia, such as eyewitness testimony (when it can be obtained) or confessions, are somewhat reliable—though not infallible—since eyewitness testimony is often inaccurate³² and false confessions³³ are frequently given. It is not

29. David M. Wheeler & Christopher J. Hobbs, *Mistakes in Diagnosing Non-accidental Injury: 10 Years' Experience*, 296 BRIT. MED. J. (CLINICAL RES. EDITION) 1233, 1234–35 (1988).

30. Victoria Levine et al., *Localized Vulvar Pemphigoid in a Child Misdiagnosed as Sexual Abuse*, 128 ARCHIVES DERMATOLOGY 804, 804 (1992).

31. See J.F. Geddes & J. Plunkett, *The Evidence Base for Shaken Baby Syndrome: We Need to Question the Diagnostic Criteria*, 328 BRIT. MED. J. 719, 719 (2004).

32. Gary L. Wells & Elizabeth A. Olson, *Eyewitness Testimony*, 54 ANN. REV. PSYCHOL. 277, 278 (2003).

hard to imagine that a trembling caretaker might feel overmatched by police, child protection agents, and medical experts who, with their intimidating welter of credentials, insist that abuse *must* have taken place. A false confession in order to get a plea bargain might seem like the only way to get a lighter sentence for what may seem to be an inevitable conviction.

A problem of circularity can occur when certain types of injuries are used as proof of abuse in studies that are aiming to identify where those same injuries lie along the spectrum of pathognomoncity for child abuse. In large measure, this is precisely what happened with shaken-baby syndrome, a diagnosis whose history is discussed in the following section. The history of shaken-baby syndrome highlights the type of problems in child-abuse research that have also influenced the way that the medical and legal communities perceive unexplained infantile fractures.

C. The Lessons of Shaken-Baby Syndrome

In 1972, Dr. John Caffey, a pediatric radiologist, first theorized that shaking infants could cause them to have intracranial hemorrhaging.³⁴ Caffey based this theory on a previous study that showed that intracranial hemorrhaging could occur in rhesus monkeys due to whiplash induced by a simulated rear-end automotive collision.³⁵ Caffey contacted Dr. Ommaya, the author of the monkey study, who advised him that there was no information at the time to suggest whether a human could generate enough angular acceleration through shaking to induce the type of injuries caused in the monkeys by the simulated rear-end collision.³⁶ Nevertheless, Caffey still presented the theory in his original paper. In a follow-up study, he opined that the presence of subdural and retinal hemorrhaging alone, even without any signs of external trauma,

33. See Richard P. Conti, *The Psychology of False Confessions*, 2 J. CREDIBILITY ASSESSMENT & WITNESS PSYCHOL. 14, 15 (1999), available at [http://www.nasams.org/forensics/for_lib/Documents/1104868281.86/Conti article the psychology of false confessions.pdf](http://www.nasams.org/forensics/for_lib/Documents/1104868281.86/Conti%20article%20the%20psychology%20of%20false%20confessions.pdf).

34. John Caffey, *On the Theory and Practice of Shaking Infants: Its Potential Residual Effects of Permanent Brain Damage and Mental Retardation*, 124 AM. J. DISEASES CHILDREN 161, 166 (1972).

35. Ronald Uscinski, *Shaken Baby Syndrome: Fundamental Questions*, 16 BRIT. J. NEUROSURGERY 217, 218 (2002).

36. *Id.*

could be sufficient to diagnose child abuse.³⁷ However, Caffey acknowledged that the current supporting evidence was “*manifestly incomplete and largely circumstantial.*”³⁸ His theory, in other words, was still just a theory. It was a noble attempt to correlate a specific type of childhood injury with abuse in hopes of helping society more effectively protect children and identify those who harmed them—but it was, as the theory’s own creator acknowledged, not adequately proven.

Notwithstanding the reservations Caffey expressed, his theory gained momentum and general acceptance in the medical community. By 1984, the first appeal of a shaken-baby syndrome conviction was reported.³⁹ Over the next two decades, thousands of people were sent to prison after being convicted of shaking babies.⁴⁰ Many of these convictions were made based only on the presence of (1) subdural hemorrhaging, (2) retinal hemorrhaging, and (3) brain swelling;⁴¹ it was not considered necessary for the child to have any outward injuries, such as bruising or scratching.⁴² This triad of injuries was considered to be pathognomonic for abusive shaking.⁴³ Prosecutors often argued that the injuries were sufficient evidence to satisfy the mens rea for murder because shaking sufficient to cause these injuries must have been so excessively violent that the perpetrator had to have known it could cause severe harm.⁴⁴ Also, the perpetrator must have been the most recent caretaker, experts testified, because symptoms would immediately occur after the

37. John Caffey, *The Whiplash Shaken Infant Syndrome: Manual Shaking by the Extremities with Whiplash-Induced Intracranial and Intraocular Bleedings, Linked With Residual Permanent Brain Damage and Mental Retardation*, 54 PEDIATRICS 396, 397 (1974) (emphasis added).

38. Genie Lyons, *Shaken Baby Syndrome: A Questionable Scientific Syndrome and a Dangerous Legal Concept*, 2003 UTAH L. REV. 1109, 1119 (emphasis added) (citation omitted).

39. Deborah Tuerkheimer, *The Next Innocence Project: Shaken Baby Syndrome and the Criminal Courts*, 87 WASH. U. L. REV. 1, 9 (2009) (citing *Ohio v. Schneider*, No. L-84-214, 1984 Ohio App. LEXIS 11988 (Ohio Ct. App. Dec. 21, 1984)).

40. Lee Scheier, *Shaken Baby Syndrome: A Search for Truth*, CHI. TRIB., June 12, 2005, at 10.

41. See Tuerkheimer, *supra* note 39, at 4. Subdural hemorrhaging, retinal hemorrhaging, and brain swelling form the primary medical evidence presented in many shaken-baby cases. *Id.*

42. See *id.* at 8.

43. *Id.* at 11.

44. *Id.* at 5.

injuries were supposedly inflicted.⁴⁵ On the basis of such expert medical testimony, juries convicted thousands of alleged abusers.⁴⁶ To many, the shaken-baby diagnosis had become a valuable tool in society's arsenal to identify and punish child abusers.

Even as its momentum soared, though, problems with the shaken-baby diagnosis quietly began to surface as early as 1987. That year, a biomechanical modeling study demonstrated that a human could not shake an infant hard enough to produce the brain hemorrhaging seen in primate models. Brain hemorrhaging could occur, the researchers opined, only upon impact against a hard surface.⁴⁷

Problems continued to trickle out over the next two decades. Conditions such as vitamin K deficiency,⁴⁸ glutaric aciduria,⁴⁹ Terson's Syndrome,⁵⁰ hemophagocytic lymphohistiocytosis,⁵¹ benign enlargement of the subarachnoid spaces,⁵² idiopathic thrombocytopenic purpura,⁵³ hemophilia,⁵⁴ Von Willebrand's Disease,⁵⁵ infective endocarditis,⁵⁶ Apnea,⁵⁷ Bradycardia,⁵⁸ and even the

45. *Id.*

46. Scheier, *supra* note 40.

47. Ann-Christine Duhaime et al., *The Shaken Baby Syndrome: A Clinical, Pathological, and Biomechanical Study*, 66 J. NEUROSURGERY 409, 414 (1987).

48. Tonia J. Brousseau et al., *Vitamin K Deficiency Mimicking Child Abuse*, 29 J. EMERGENCY MED. 283, 283 (2005).

49. L. M. Hartley et al., *Glutaric Aciduria Type I and Nonaccidental Head Injury*, 107 PEDIATRICS 174, 174 (2001).

50. Giulio Cesare Pinnola et al., *Terson's Syndrome: Report of a Case with a Favorable Outcome*, 56 ARQUIVOS DE NEURO-PSIQUIATRIA 133, 133 (1998), available at <http://www.scielo.br/pdf/anp/v56n1/1880.pdf>.

51. Betty Spivack, *The Differential Diagnosis of Abusive Head Trauma Widens*, 10 AM. ASSOC. PEDIATRICS GRAND ROUNDS 33, 33 (2003), available at <http://aapgrandrounds.aapublications.org/cgi/reprint/10/3/33-a> (purchase required for download).

52. P.D. McNeely et al., *Subdural Hematomas in Infants with Benign Enlargement of the Subarachnoid Spaces Are Not Pathognomonic for Child Abuse*, 27 AM. J. NEURORADIOLOGY 1725, 1728 (2006).

53. Sarah Joan Woerner et al., *Intracranial Hemorrhage in Children with Idiopathic Thrombocytopenic Purpura*, 67 PEDIATRICS 453, 453 (1981).

54. Gordon L. Bray & Naomi L. C. Luban, *Hemophilia Presenting with Intracranial Hemorrhage: An Approach to the Infant with Intracranial Bleeding and Coagulopathy*, 141 AM. J. DISEASES CHILDREN 1215, 1215 (1987), available at <http://archpedi.ama-assn.org/cgi/content/abstract/141/11/1215> (purchase required for download).

55. Walid S. Almaani & Abdulla S. Awidi, *Spontaneous Intracranial Hemorrhage Secondary to Von Willebrand's Disease*, 26 SURGICAL NEUROSURGERY 457, 457 (1986).

56. R. G. Hart et al., *Mechanisms of Intracranial Hemorrhage in Infective Endocarditis*, 18 STROKE J. AM. HEART ASS'N 1048, 1048 (1987), available at

performance of CPR⁵⁹ were all shown to cause spontaneous intracranial hemorrhaging in infants.

Other studies also showed that “lucid intervals” of up to seventy-two hours⁶⁰ could occur between the infliction of a brain injury and the onset of symptoms. This was significant because, even if the symptoms had been caused by abuse, it was no longer certain that the person who was caring for a baby at the time of the onset of symptoms must have been the abuser. Oddly, the fact that weeks or months could occur between an injury and the onset of the clinical signs of a chronic subdural hematoma was recognized in a study in 1945—and that study was noted by Caffey in a 1946 study of his own, several decades before the shaken-baby hypothesis was made.⁶¹ Furthermore, another study showed that asymptomatic chronic subdural hematomas could go undetected and could be reactivated into symptomatic hemorrhages by relatively minor bumps on the head.⁶² Other studies showed these chronic subdural hematomas could occur in utero⁶³ (before birth) and as the result of birth trauma.⁶⁴ The latter study even showed that asymptomatic intracranial hemorrhages often occur as the result of normal vaginal delivery.⁶⁵

Among a growing number of studies that began to question whether the shaken-baby triad was pathognomonic of child abuse, an extensive review of the scientific literature on shaken-baby syndrome

<http://stroke.ahajournals.org/cgi/reprint/18/6/1048>.

57. Christopher B. Looney et al., *Intracranial Hemorrhage in Asymptomatic Neonates: Prevalence on MR Images and Relationship to Obstetric and Neonatal Risk Factors*, 242 *RADIOLOGY* 535, 536 (2007), available at <http://radiology.rsna.org/content/242/2/535.full.pdf+html>.

58. *Id.*

59. Victor W. Weedn et al., *Retinal Hemorrhage in an Infant After Cardiopulmonary Resuscitation*, 11 *AM. J. FORENSIC MED. & PATHOLOGY* 79, 79 (1990).

60. Scott Denton & Darinka Mileusnic, *Delayed Sudden Death in an Infant Following an Accidental Fall: A Case Report with Review of the Literature*, 24 *AM. J. FORENSIC MED. & PATHOLOGY* 371, 371 (2003), available at <http://journals.lww.com/amjforensicmedicine/toc/2003/12000> (purchase required for download).

61. Caffey, *supra* note 7, at 172.

62. Uscinski, *supra* note 35.

63. Ciaran J. Powers et al., *Chronic Subdural Hematoma of the Neonate: Report of Two Cases and Literature Review*, 43 *PEDIATRIC NEUROSURGERY* 25, 25 (2007), available at <http://content.karger.com/produktedb/produkte.asp?typ=pdf&file=000097521> (purchase required for download).

64. Looney, *supra* note 57.

65. *Id.*

published between 1966 and 1998 was completed in 2003.⁶⁶ This study made a sobering observation: shaken-baby syndrome's evidentiary base was like "an inverted pyramid" plagued with circular assumptions and other serious deficiencies.⁶⁷ In particular, many studies lacked adequate controls and many used selection criteria based on presumption or suspicion.⁶⁸ While these weaknesses are unsurprising in light of the ethical constraints inherent in child-abuse research, "repeated opinions based on poor-quality data cannot improve the quality of evidence."⁶⁹ In addition, many of the shaken-baby researchers committed the logical flaw of affirming the consequent: they assumed that if retinal and subdural hemorrhages were always seen in shaken-baby cases, then the presence of retinal and subdural hemorrhages proved that a baby had been shaken.⁷⁰ Regarding these shaken-baby studies, a leading national expert woefully admitted, "[W]e as a group that wrote those papers *assumed what we were concluding*."⁷¹ In other words, the existence of shaken-baby syndrome was based on research that was not scientifically strong enough to justify its long-accepted status in the medical community.⁷²

The debate about how frequently the triad is caused by abusive versus nonabusive causes still rages on, as does the debate about whether shaking alone can cause the triad. One thing, however, is certain: by itself, the so-called shaken-baby triad is not *pathognomonic* for abusive shaking. A number of people have been exonerated in recent years as a result of these new findings,⁷³ but it is unclear how many innocent caretakers still remain behind bars. At the very least, some states are now adjusting their laws to forestall erroneous accusations of child abuse based only on the triad once thought to be pathognomonic of shaken-baby syndrome.

66. Mark Donohoe, *Evidence-Based Medicine and Shaken Baby Syndrome: Part I: Literature Review, 1966–1998*, 24 AM. J. FORENSIC MED. & PATHOLOGY 239, 239 (2003).

67. *Id.* at 241.

68. *Id.* at 240.

69. *Id.* at 241.

70. *Id.*

71. Tuerkheimer, *supra* note 39, at 13 n.78 (emphasis added) (citation omitted).

72. Shaken-baby syndrome's acceptance in the medical community was an example of a phenomenon known as the "informational cascade." For an excellent explanation about cascades, see WARD FARNSWORTH, *THE LEGAL ANALYST: A TOOLKIT FOR THINKING ABOUT THE LAW* 136–43 (2007).

Colorado,⁷³ West Virginia,⁷⁴ Kentucky,⁷⁵ and New Mexico,⁷⁶ for example, now require that infants be tested for glutaric aciduria (which, as mentioned above, is one of the medical conditions that is now known to cause the shaken-baby triad).

There are many lessons to be learned from the history of shaken-baby syndrome, not all of them within the scope of this Comment. One lesson, though, is clear: a misappraisal of whether a certain injury or combination of injuries is pathognomonic can lead to the conviction and imprisonment of innocent caretakers. As a result, careful consideration should be given in each case to alternative causes—even extremely rare ones—of injuries whose presence alone has traditionally been sufficient to secure convictions of accused caretakers. The fact that an abusive cause is more likely—perhaps even much more likely—than a nonabusive one should not inevitably lead to the conclusion that the evidentiary standard of proof, beyond a reasonable doubt, has been met. In the words of William Blackstone, “it is better that ten guilty persons escape, than that one innocent suffer.”⁷⁷

Awareness is now growing that consideration should be given to possible alternative causes of the shaken-baby triad. But the shaken-baby triad is not the only type of alleged injury that is often mistakenly used, even in the absence of other evidence, to convict caretakers of child abuse. There is also a growing body of scientific literature that suggests that unexplained infantile fractures may not be as pathognomonic for abuse as is currently believed.

III. UNEXPLAINED FRACTURES AND CHILD ABUSE

A. History

In 1946, Dr. Caffey (the same researcher who would later hypothesize that shaking could cause intracranial hemorrhaging⁷⁸) published a study in which he elucidated a suspicious correlation

73. SHAFEEK S. SANBAR, *LEGAL MEDICINE* 525 (2004).

74. W. VA. CODE § 16-22-3(a) (2009).

75. KY. REV. STAT. ANN. § 214.155(2) (West 2006).

76. N.M. STAT. ANN. § 24-1-6(A)(2) (1978).

77. 4 WILLIAM BLACKSTONE, *COMMENTARIES ON THE LAWS OF ENGLAND* 352, 358 (Univ. of Chi. Press 1979) (1769).

78. See Caffey, *supra* note 34, 164–68.

between multiple long-bone⁷⁹ fractures and chronic subdural hematomas in infants.⁸⁰ While the known association between cranial fractures and subdural hematomas seemed reasonable, Caffey had been puzzled for years about the connection between long-bone fractures and subdural hematomas.⁸¹ Using x-rays (“roentgenograms”) and some simple tests of the blood and cerebrospinal fluid, Caffey expressed confidence that scurvy and localized skeletal diseases could be excluded as factors that might have predisposed the six infants he examined to fractures.⁸² In light of these facts, he concluded that the fractures had been caused by trauma that was “either not observed or denied when observed.”⁸³ Though he stopped short of explicitly suggesting that such trauma had been intentionally inflicted, his reluctance to make such a suggestion may have been because he feared legal repercussions.⁸⁴ In 1957, however, Caffey published another study where he suggested that abusive trauma could be inferred from x-rays that showed multiple fractures in different stages of healing; “[i]n many cases,” he asserted, “radiological changes are pathognomonic of trauma and an immediate conclusive diagnosis can be made from them *without recourse to clinical and laboratory investigations*.”⁸⁵ Thus, the seed for the notion that multiple unexplained fractures might be pathognomonic for child abuse was planted.

Other studies began to follow suit. In 1962, a landmark study by Dr. C. Henry Kempe was published that finally openly addressed the issue of child abuse.⁸⁶ Kempe opined that physical abuse was a frequent cause of permanent injury and death to children.⁸⁷ Furthermore, he advised that battered-child syndrome “should be

79. The term “long bones” refers to bones that are longer than they are wide, such as femurs, tibias, and fibulas (legs) and the humeri, radii, and ulnas (arms).

80. Caffey, *supra* note 7, at 173.

81. *Id.* at 163.

82. *See id.* at 161–69.

83. *Id.* at 172.

84. Marilyn Heins, *The “Battered Child” Revisited*, 251 J. AM. MED. ASS’N 3295, 3296 (1984).

85. John Caffey, *Some Traumatic Lesions in Growing Bones Other Than Fractures and Dislocations: Clinical and Radiological Features—The Mackenzie Davidson Memorial Lecture*, 30 BRIT. J. RADIOLOGY 225, 228 (1957) (emphasis added).

86. C. Henry Kempe et al., *The Battered-Child Syndrome*, 181 J. AM. MED. ASS’N 17 (1962).

87. *Id.*

considered in any child exhibiting evidence of fracture of any bone.”⁸⁸ Kempe seemed to be of the opinion that multiple unexplained fractures were on the extreme end of the pathognomoncity spectrum for abuse; as he put it, “the bones tell a story the child is too young or too frightened to tell.”⁸⁹ In the meantime, a study published by Dr. D.Ll. Griffiths the following year further developed the issue by discussing four suspicious cases of infants with unexplained fractures.⁹⁰ Using x-rays and blood tests like the original Caffey study, Griffiths felt scurvy could be excluded as a possible cause.⁹¹ Reaching a conclusion parallel to Caffey’s, Griffiths opined that child abuse could be diagnosed from x-rays rather than from clinical history.⁹²

The hypothesis that multiple unexplained fractures in different stages of healing were essentially pathognomonic for child abuse gained momentum and general acceptance in the medical community.⁹³ However, because of the same obvious ethical considerations that preclude direct experimentation to verify the symptoms of physical child abuse, many subsequent studies that have attempted to identify the distinguishing features of abusive fractures have been forced to use the same types of problematic inclusion criteria that have plagued comparable studies on shaken-baby syndrome.⁹⁴

Those studies largely stand on the shoulders of the starting point of Caffey’s seminal study. However, a close examination of that study shows bias toward the diagnosis of child abuse that is reflected in more than just the inclusion criteria. A careful reading reveals some problems with Caffey’s assertion that, in the cases of all six infants studied, scurvy and other conditions predisposing the infants to fractures could be ruled out. In the conclusion, he stated that “[t]here was no [x-ray] or clinical evidence of general or localized skeletal disease which would have predisposed the bones to

88. *Id.*

89. *Id.* at 18.

90. Griffiths & Moynihan, *supra* note 4, at 1558–59.

91. *Id.* at 1559–60.

92. *Id.* at 1561.

93. See, e.g., I. Blumenthal, *Shaken Baby Syndrome*, 78 POSTGRADUATE MED. J. 732, 733 (2002) (citing Caffey in support of assertion that metaphyseal fractures are pathognomonic of inflicted injury in infants).

94. See Alison M. Kemp et al., *Patterns of Skeletal Fractures in Child Abuse: Systematic Review*, BRIT. MED. J. 2 (Oct. 2, 2008), <http://www.bmj.com/content/337/bmj.a1518.full>.

pathological fractures.”⁹⁵ However, he also noted in the text of his study that x-rays had shown some localized osteoporosis in two of the six cases.⁹⁶ Osteoporosis, as is commonly known, predisposes bones to fractures.⁹⁷ In addition, he also noted that one of the patients had bleeding gums and bloody feces,⁹⁸ symptoms which are associated with scurvy;⁹⁹ scurvy, in turn, is associated with bone fragility.¹⁰⁰ While the bloody stools might have been attributed to abusive internal injury, bleeding gums do not easily lend themselves to an abusive explanation. Furthermore, the assertion that a diagnosis could be made *without recourse to clinical and laboratory investigations* is also problematic because it seems to implicitly assume that all types of conditions that can cause bone fragility are detectable in normal x-rays.

The Griffiths study also demonstrated a bias toward the diagnosis of abuse by asserting that “[t]he x-ray changes in the ‘battered baby’ are so like those often described in infantile scurvy (Barlow’s disease) that, in our opinion, many of the illustrations in textbooks of radiology and of orthopaedics which purport to show typical changes of scurvy are in fact examples of [battered-child syndrome].”¹⁰¹ He did not consider the possibility that the similarity of the x-ray findings in his study to x-rays of fractures attributed to scurvy in textbooks might indicate that some of the children in his study might have had undetected scurvy—even though fractures can be the first symptoms of scurvy that appear.¹⁰² Furthermore, the Griffiths researchers’ assertion that “[m]ultiple unexplained shaft fractures in babies are clearly outside ordinary domestic hazards”¹⁰³ seems to presume either the nonexistence of bone fragility in infants or at least the statistical independence of one unexplained fracture from another. That assumption of statistical independence also seems to underlie Caffey’s conclusions.

95. Caffey, *supra* note 7, at 173.

96. *Id.* at 163, 168.

97. JOHN C. STEVENSON & MICHAEL S. MARSH, AN ATLAS OF OSTEOPOROSIS 71 (3d ed. 2007).

98. Caffey, *supra* note 7, at 164.

99. ALFRED F. HESS, SCURVY, PAST AND PRESENT 180, 208 (1920).

100. See James F. Brailsford, *Some Radiographic Manifestations of Early Scurvy*, 28 ARCHIVES DISEASE CHILDHOOD 81, 84 (1953).

101. Griffiths & Moynihan, *supra* note 4, at 1560.

102. See Brailsford, *supra* note 100, at 86.

103. Griffiths & Moynihan, *supra* note 4, at 1559.

A full review of all the literature on unexplained multiple fractures in infants is beyond the scope of this Comment. However, the problems highlighted in the initial and subsequent studies on unexplained infantile fractures suggest that some consideration should be given to the existence and prevalence of conditions that might cast doubt on medical and legal appraisals of where unexplained fractures are placed on the spectrum of pathognomoncity.

Recent and not-so-recent findings have demonstrated that there are, in fact, a number of conditions that can cause bone fragility in children. Some of these conditions are not immediately obvious, even to trained radiologists and other doctors. Some are well established, while others are controversial. They are, however, common enough that broken bones—even multiple rib fractures in different stages of healing or metaphyseal fractures—should not be considered to be fail-safe indicators of child abuse. The following section describes some of these conditions that can cause bone fragility in children.

B. Known Conditions that Can Cause Bone Fragility

1. Premature birth and osteopenia of prematurity

The bones of very premature infants are so fragile that “[e]ven without definite evidence of bone disease, which is almost universal in infants born at less than twenty-eight weeks’ gestation, premature infants are at risk for fractures and extreme care in handling them is essential.”¹⁰⁴ A common problem that affects premature infants is osteopenia of prematurity (OP),¹⁰⁵ which refers to the reduced skeletal mineralization seen in preterm infants compared to infants who were born at a normal gestational age.¹⁰⁶ This occurs because the rate of the bone accretion in a baby increases throughout pregnancy,¹⁰⁷ so the time in utero that is lost by a preterm birth

104. Rachel R. Phillips & Stephen H. Lee, *Fractures of Long Bones Occurring in Neonatal Intensive Care Units*, 301 BRIT. MED. J. 225, 226 (1990).

105. Also known as “metabolic bone disease of prematurity” or “rickets of prematurity.”

106. Frank R. Greer, *Osteopenia of Prematurity*, 14 ANN. REV. NUTRITION 169, 169 (1994), available at <http://www.annualreviews.org/doi/abs/10.1146/annurev.nu.14.070194.001125> (purchase required for download).

107. N. Bishop, *Bone Disease in Preterm Infants*, 64 ARCHIVES DISEASE CHILDHOOD 1403, 1403 (1989), available at <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1590102/pdf/archdisch00899-0067.pdf>.

results in a larger deficit in bone mineralization than a linear model would predict. In fact, approximately eighty percent of fetal bone mineral accretion occurs during the last trimester of pregnancy.¹⁰⁸ In other words, a baby born three months premature has lost a third of what should have been her time in the womb, but she has lost four-fifths of what would have been her accumulated bone mineralization level if she had been born full term. As a result, “*all* infants born at [less than thirty-two] weeks gestation have some degree of hypomineralization during and subsequent to the prolonged period of hospitalization” associated with premature birth.¹⁰⁹ Since modern medical care has been able to save the lives of more premature infants in recent years, the number of surviving infants with OP has increased.¹¹⁰

“Osteopenic [infants] are susceptible to fractures with normal handling during routine care.”¹¹¹ Sometimes these fractures occur in the hospital and are identified before the babies are sent home.¹¹² Other times, fractures occur with relatively minor trauma after the babies have been home with their parents.¹¹³

There is a danger that OP can be overlooked because conventional radiological methods (like standard x-rays) cannot detect low bone mineralization unless it is thirty to forty percent¹¹⁴ lower than normal. When used to measure bone density, standard x-rays (like those used in the 1946 Caffey study) and some other methods are said to have error rates ranging from thirty to fifty percent.¹¹⁵ In the words of one researcher, “To depend on radiology

108. Greer, *supra* note 106, at 169.

109. *Id.* at 173 (emphasis added).

110. Hüseyin Çaksen et al., *Letter to the Editor*, 23 J. EMERGENCY MED. 305 (2002).

111. Wendy L. Sievert, Joint Compression Therapy in the Prevention of Osteopenia of Prematurity: Current Research and Future Considerations 3 (Apr. 2011) (unpublished M.A. thesis, St. Catherine University), available at http://sophia.stkate.edu/ma_nursing/7; see also J. Mercy et al., *Relationship of Tibial Speed of Sound and Lower Limb Length to Nutrient Intake in Preterm Infants*, 92 ARCHIVES DISEASE CHILDHOOD FETAL NEONATAL ED., at F381, F381 (2007) (“Metabolic bone disease of prematurity (MBDP) is characterised by skeletal demineralisation and fractures that can occur during normal handling.”).

112. See, e.g., Mercy, *supra* note 111, at F381; Sievert, *supra* note 111.

113. See, e.g., Greer, *supra* note 106, at 170.

114. *Id.* at 176; see Andrew K. Poznanski, *Radiologic Evaluation of Bone Mineral in Children*, in PRIMER ON THE METABOLIC BONE DISEASES AND DISORDERS OF MINERAL METABOLISM 115, 115 (2d ed. 1993).

115. C. Conrad Johnston & L. Joseph Melton III, *Bone Density Measurement and the Management of Osteoporosis*, in PRIMER ON THE METABOLIC BONE DISEASES AND DISORDERS OF MINERAL METABOLISM, *supra* note 114, at 137, 137.

for the diagnosis of osteopenia will result in serious underestimation of the extent of this condition.”¹¹⁶ A more accurate test of bone density is needed to avoid such underestimation of the incidence of OP.¹¹⁷

2. Metabolic bone diseases

a. Liver disorders. The liver produces bile, which enhances the absorption of vitamin D, vitamin K, and other fat-soluble vitamins.¹¹⁸ Without bile, “a good proportion of fat-soluble vitamins” that enter the digestive tract are not absorbed by the body.¹¹⁹ Vitamin D, in turn, is needed for the body to efficiently absorb calcium.¹²⁰ Because calcium is required for bone mineralization, a disruption in the liver’s production or delivery of bile to the digestive tract may impede the body’s calcium intake and therefore the body’s bone mineralization.¹²¹ Some studies also link vitamin K deficiency with bone fragility.¹²²

Unsurprisingly, liver dysfunction is linked to bone fragility in infants.¹²³ Biliary atresia, a condition resulting from a congenital malformation of the bile ducts, can cause bone fragility and lead to fractures that can be erroneously attributed to abuse.¹²⁴ In one case,

116. J.R. James et al., *Osteopenia of Prematurity*, 61 ARCHIVES DISEASE CHILDHOOD 871, 875 (1986).

117. *See id.* at 871.

118. *See* Sidney Cohen, *Cholestasis*, THE MERK MANUAL HOME HEALTH HANDBOOK, <http://tinyurl.com/3e6nyez> (last updated Aug. 2006).

119. David W. Hobson & Valerie L. Hobson, *Normal and Abnormal Intestinal Absorption by Humans*, in TOXICOLOGY OF THE GASTROINTESTINAL TRACT 279, 292 (Shayne C. Gad et al. eds., 2007).

120. *See* Michael F. Holick, *Photobiology of Vitamin D*, in 1 VITAMIN D 37, 37 (David Feldman et al. eds., 2d ed. 2005).

121. *See* Akio Kobayashi et al., *Bone Disease in Infants and Children with Hepatobiliary Disease*, 49 ARCHIVES DISEASE CHILDHOOD 641, 645 (1974).

122. *See* Susanne Bügel, *Vitamin K and Bone Health*, 62 PROC. NUTRITION SOC’Y 839, 840–41 (2003).

123. *See* Kobayashi, *supra* note 121, at 641; *see also* Eric A. Argao et al., *d-Alpha-Tocopheryl Polyethylene Glycol-1000 Succinate Enhances the Absorption of Vitamin D in Chronic Cholestatic Liver Disease of Infancy and Childhood*, 31 PEDIATRIC RES. 146, 146 (1992) (“Bone disease (rickets and osteopenia) is a common complication of chronic childhood cholestatic liver disease.”).

124. Patricia A. DeRusso et al., *Fractures in Biliary Atresia Misinterpreted as Child Abuse*, 112 PEDIATRICS 185, 185 (2003).

a baby suffered a fractured tibia from being held down by a phlebotomist so blood could be drawn from her foot.¹²⁵

Biliary atresia, though, is not the only liver disorder that is linked to bone fragility or long-bone fractures. Others include Alagille syndrome,¹²⁶ Wilson's disease,¹²⁷ Byler disease,¹²⁸ and hepatitis.¹²⁹

In addition to liver dysfunction's effect on vitamin D and vitamin K absorption, it negatively affects bone mass in a less commonly known way. Jaundice, a yellowing of the skin and eyes, is associated with liver dysfunction.¹³⁰ It occurs when levels of conjugated or unconjugated bilirubin are elevated in the blood.¹³¹ Jaundice does not always indicate serious liver problems; about one in three babies experience it in a temporary form during the first week of life.¹³² Breast-milk jaundice, a common condition, may persist for more than three weeks for up to 2.4 % of breastfed infants.¹³³ Significantly, there was a study (the Janes study) done on adults with chronic cholestatic liver disease (CCLD) that demonstrated that a high level of unconjugated bilirubin impedes the function of osteoblasts¹³⁴ (the cells that form bone). The researchers noted that the effect of depressing osteoblast function did not just occur in plasma from patients with CCLD, but also in the plasma "from patients with jaundice due to other causes."¹³⁵ "This suggests," the researchers explained, "that depressed osteoblast function may be related to the

125. *Id.* at 185–86.

126. See Ruben E. Quiros-Tejeira et al., *Variable Morbidity in Alagille Syndrome: A Review of 43 Cases*, 29 J. PEDIATRIC GASTROENTEROLOGY & NUTRITION 431, 431 (1999).

127. A.G. Bearn, *Wilson's Disease: An Inborn Error of Metabolism with Multiple Manifestations*, 22 AM. J. MED. 747, 756 (1957); D. Hegedus et al., *Decreased Bone Density, Elevated Serum Osteoprotegerin, and β -Cross-Laps in Wilson Disease*, 17 J. BONE AND MIN. RES. 1961, 1966 (2002).

128. B.M. Winkhofer-Roob et al., *Progressive Idiopathic Cholestasis Presenting with Profuse Watery Diarrhoea and Recurrent Infections (Byler's Disease)*, 81 ACTA PAEDIATRICA 637, 637 (1992).

129. Kobayashi, *supra* note 121, at 644.

130. See PETER G. B. JOHNSTON ET AL., *NEWBORN CHILD* 188–90 (9th ed. 2003).

131. *Id.*

132. *Id.* at 189.

133. C.R. Winfield & R. MacFaul, *Clinical Study of Prolonged Jaundice in Breast- and Bottle-fed Babies*, 53 ARCHIVES DISEASE CHILDHOOD 506, 507 (1978).

134. Christine H. Janes et al., *Role of Hyperbilirubinemia in the Impairment of Osteoblast Proliferation Associated with Cholestatic Jaundice*, 95 J. CLINICAL INVESTIGATION 2581, 2585 (1995).

135. *Id.*

jaundice itself and is not a specific hallmark of CCLD.”¹³⁶ If jaundice alone—the mere presence of an elevated level of bilirubin—depresses osteoblast function, it is possible that a condition like breast-milk jaundice may negatively affect infantile bone density. Though no studies have yet been done on this point, some studies have shown that some infants with breast-milk jaundice have serum bilirubin concentrations that are within the range of those of the patients with CCLD in the Janes study.¹³⁷

b. Rickets. Rickets is caused by a deficiency of vitamin D and results in “poor global mineralization of the skeleton.”¹³⁸ A 2008 study highlighted several cases where fractures from rickets have been mistaken for child abuse.¹³⁹ As mentioned previously, this study also noted that ordinary x-rays are inadequate to measure bone density unless thirty to forty percent demineralization has already occurred.¹⁴⁰

Vitamin D deficiency is often associated with a lack of exposure to sunlight, since ninety percent of the vitamin D the body receives comes from sunlight.¹⁴¹ Those at greatest risk are people who live in higher latitudes or low sunshine climates and have darker skin pigmentation.¹⁴² Recent studies have demonstrated that “vitamin D deficiency is widespread in industrialized nations.”¹⁴³ Even in Australia, a country with abundant sunlight, vitamin D deficiency is a problem among women.¹⁴⁴ When mothers are vitamin D deficient during pregnancy, their babies are at an increased risk of vitamin D

136. *Id.*

137. *Compare id.* at 2581 (serum bilirubin concentrations of 1.5 to 34.8 mg/dl), with Lawrence M. Gartner & Irwin M. Arias, *Studies of Prolonged Neonatal Jaundice in the Breast-fed Infant*, 68 J. PEDIATRICS 54, 54 (1966) (serum bilirubin concentrations that are the equivalent of 9.0 to 26.0 mg/dl).

138. Lisa M. Bodnar et al., *High Prevalence of Vitamin D Insufficiency in Black and White Pregnant Women Residing in the Northern United States and Their Neonates*, 137 J. NUTRITION 447, 447 (2007).

139. Kathy A. Keller & Patrick D. Barnes, *Rickets vs. Abuse: A National and International Epidemic*, 38 PEDIATRIC RADIOLOGY 1210, 1213–14 (2008).

140. *Id.* at 1212.

141. Josephine M. Nozza & Christine P. Rodda, *Vitamin D Deficiency in Mothers of Infants with Rickets*, 175 MED. J. AUSTRALIA 253, 253 (2001).

142. Lucy Bowyer et al., *Vitamin D, PTH and Calcium Levels in Pregnant Women and Their Neonates*, 70 CLINICAL ENDOCRINOLOGY 372, 372 (2009).

143. Anne Merewood et al., *Widespread Vitamin D Deficiency in Urban Massachusetts Newborns and Their Mothers*, 125 J. AM. ACAD. PEDIATRICS 640, 641 (2010).

144. Bowyer et al. *supra* note 142, at 372.

deficiency.¹⁴⁵ Furthermore, “black and white pregnant women residing in [sic] northern United States and their neonates are at high risk of vitamin D insufficiency, even when they regularly use a prenatal vitamin or multivitamin.”¹⁴⁶ Because breast milk supplies little vitamin D, infants who are exclusively breastfed are also at greater risk.¹⁴⁷ Infants who are exclusively breastfed are also at greater risk for vitamin K deficiency,¹⁴⁸ which is another risk factor for bone fragility.¹⁴⁹

Vitamin D deficiency is widespread in North America.¹⁵⁰ A 2010 study of 459 mother-infant pairs in Boston showed that “more than half of the infants and approximately one third of the mothers . . . were vitamin D deficient at the time of delivery.”¹⁵¹ The use of prenatal vitamins “was protective,” but even when they were taken, “considerable proportions of infants and mothers remained deficient.”¹⁵²

c. Other metabolic bone diseases. Severe kidney dysfunction can also lead to bone demineralization that can cause deficient bone mineral density, leading to fractures that may mimic child abuse.¹⁵³ Thyroid dysfunction can also lead to bone fragility.¹⁵⁴ Intestinal disorders, which can impede absorption of essential vitamins, can also affect the bones; Crohn’s disease, for example, has been linked to an increased risk for fractures.¹⁵⁵

145. *Id.* at 374.

146. Bodnar et al., *supra* note 138, at 451.

147. Nozza & Rodda, *supra* note 141, at 253.

148. Frank R. Greer, *Are Breast-Fed Infants Vitamin K Deficient?*, 501 *ADVANCES EXPERIMENTAL & MED. BIOLOGY* 391, 394 (2001).

149. *See* Bügel, *supra* note 122.

150. David A. Hanley & K. Shawn Davison, *Vitamin D Insufficiency in North America*, 135 *J. NUTRITION* 332, 336 (2005).

151. Merewood, *supra* note 143, at 646.

152. *Id.*

153. Jerry R. Dwek, *The Radiographic Approach to Child Abuse*, 469 *CLINICAL ORTHOPAEDICS & RELATED RES.* 776, 785 (2011).

154. Douglas C. Bauer et al., *Risk for Fracture in Women with Low Serum Levels of Thyroid-Stimulating Hormone*, 134 *ANNALS INTERNAL MED.* 561, 561 (2001), available at <http://www.annals.org/content/134/7/561.abstract>.

155. Peter Vestergaard & Leif Mosekilde, *Fracture Risk in Patients with Celiac Disease, Crohn’s Disease, and Ulcerative Colitis: A Nationwide Follow-up Study of 16,416 Patients in Denmark*, 156 *AM. J. EPIDEMIOLOGY* 1, 1–2 (2002), available at <http://aje.oxfordjournals.org/content/156/1/1.full.pdf+html>.

3. Collagen disorders

a. Osteogenesis imperfecta. The best-known disorder that causes bone fragility in infants is osteogenesis imperfecta (OI). There are nine different types, most of which are linked to problems with the formation or quality of collagen (a protein prevalent in connective tissue).¹⁵⁶ Forms of OI that are recognizable at birth occur at a rate between one in sixteen thousand and one in twenty thousand; milder forms that are typically not recognized until later in life occur at the same frequency.¹⁵⁷ OI, along with Marfan syndrome, is the most common heritable connective tissue disorder.¹⁵⁸ While ordinary x-rays can often reveal signs of most types of OI, some OI types are not recognizable until later in life.¹⁵⁹ Common signs of OI, such as blue sclera (blueness in the white part of the eyes), are present in some, but not all, forms of OI.¹⁶⁰ A labor-intensive skin biopsy can be used to test for it, though the procedure is only about eighty five percent accurate.¹⁶¹

There have been a number of cases where unexplained fractures in infants with OI have likely been mistaken for child abuse.¹⁶² In *Velasquez v. Goodwin*, a Virginia court reversed an administrative finding that a father had abused his son after the child tested positive for OI; the administrative finding was based on the presence of unexplained rib fractures in different stages of healing as identified by normal x-rays.¹⁶³

b. Scurvy. Scurvy is a disease resulting from a vitamin C deficiency. "Vitamin C is . . . essential for collagen formation and fractures, including metaphyseal fractures, have been reported in

156. Joan Marini, *Osteogenesis Imperfecta*, ENDOTEXT.ORG 1-4 (Mar. 1, 2010), <http://www.endotext.org/parathyroid/parathyroid17/parathyroid17.pdf>.

157. *Id.* at 1.

158. *Id.*

159. *See id.*

160. Colin R. Paterson, *Brittle Bone Disease (Osteogenesis Imperfecta)*, NETDOCTOR (Sept. 15, 2010), <http://www.netdoctor.co.uk/diseases/facts/brittlebones.htm>.

161. *Id.*

162. Colin R. Paterson & Susan J. McAllion, *Child Abuse and Osteogenesis Imperfecta*, 295 BRIT. MED. J. 1561, 1561 (1987), available at <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1248690/pdf/bmjcred00050-0063c.pdf>.

163. *Velasquez v. Goodwin*, No. 0033-03-4, 2004 WL 1773647, at *1-2 (Va. Ct. App. Aug. 10, 2004).

scurvy.”¹⁶⁴ An early-onset variant of vitamin C deficiency can occur at eight to twelve weeks of age.¹⁶⁵

Scurvy can cause bone fragility before any changes in bone texture or density are evident in ordinary x-rays.¹⁶⁶ Indeed, bone fractures can occur before any definite clinical evidence of scurvy becomes apparent.¹⁶⁷ This fact undercuts the assurances of Caffey¹⁶⁸ and Griffiths¹⁶⁹ that none of the children in their studies were affected by scurvy.

4. Some other conditions that have been linked to bone fragility

Osteomyelitis, copper deficiency, Menkes syndrome, osteopetrosis, hypophosphatasia, congenital syphilitic periostitis, leukemia, and vitamin A toxicity have all been linked to bone fragility, though these conditions have distinctive characteristics that can be identified through normal radiographic techniques.¹⁷⁰ Disuse osteoporosis, which occurs when a person's movement is restricted for a long period of time, causes bone fragility.¹⁷¹ Cole-Carpenter syndrome, Bruck syndrome, McCune-Albright syndrome, and congenital cytomegalovirus infection are also linked to bone fragility,¹⁷² as are cerebral palsy¹⁷³ and cystic fibrosis.¹⁷⁴ Even some hematological diseases, such as congenital erythropoietic porphyria,

164. Colin R. Paterson, *Multiple Fractures in Infancy: Scurvy or Nonaccidental Injury?*, 2 ORTHOPEDIC RES. AND REVS. 45, 45 (2010).

165. C. Alan B. Clemetson, *Caffey Revisited: A Commentary on the Origin of "Shaken Baby Syndrome"*, 11 J. AM. PHYSICIANS AND SURGEONS 20, 20 (2006).

166. Brailsford, *supra* note 100, at 81.

167. *Id.* at 86.

168. Caffey, *supra* note 85.

169. Griffiths & Moynihan, *supra* note 4, 1558–59.

170. Carole Jenny, *Evaluating Infants and Young Children With Multiple Fractures*, 118 PEDIATRICS 1299, 1301 (2006).

171. Shinjiro Takata & Natsuo Yasui, *Disuse Osteoporosis*, 48 J. MED. INVESTIGATION 147, 147 (2001), available at <http://tinyurl.com/3srcupz>.

172. Nick Bishop et al., *Unexplained Fractures in Infancy: Looking for Fragile Bones*, 92 ARCHIVES DISEASE CHILDHOOD 251, 253 (2007).

173. S. Lingam & Jane Joester, *Spontaneous Fractures in Children and Adolescents with Cerebral Palsy*, 309 BRIT. MED. J. 265, 265 (1994), available at <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2540761/pdf/bmj00450-0057.pdf>.

174. Tarak Srivastava & Uri S. Alon, *The Role of Bisphosphonates in Diseases of Childhood*, 162 EUR. J. PEDIATRICS 735, 740 (2003).

Diamond-Blackfan syndrome, and Job syndrome are associated with bone fragility.¹⁷⁵

C. Temporary Brittle-Bone Disease: The Controversy

Most of the aforementioned conditions, such as rickets, OP, and OI, are known and accepted medical diagnoses. Temporary Brittle Bone Disease (TBBD), by contrast, is a hypothesis that has been controversial and polarizing since its inception. An in-depth examination of the history of TBBD is beyond the scope of this Comment, but it is mentioned here because it ultimately remains a plausible, though unpopular hypothesis,¹⁷⁶ and its history demonstrates that the medical and legal communities are often biased by strong emotions when considering possible nonabusive causes of child abuse.

TBBD is a term coined by Dr. Colin Paterson of Dundee, Scotland. Paterson, who has done a great deal of research on brittle-bone diseases, was puzzled by a number of cases of unexplained infantile fractures that shared some common traits: (1) an absence of external bruising, (2) a similar age range, (3) similar fracture types (predominantly rib fractures), (4) vomiting, (5) diarrhea, (6) enlarged fontanelles, and (7) a family history of double-jointedness.¹⁷⁷ Many of the infants in these studies were born premature or were twins.¹⁷⁸ Furthermore, unlike children with OI, the children in these cases generally did not suffer new fractures after being returned to their parents.¹⁷⁹ Paterson therefore hypothesized that these infants had been affected by a temporary condition of bone fragility of unknown origin—a temporary brittle-bone disease. He opined that copper deficiency, a known cause of bone fragility, might play a role.¹⁸⁰ Dr. Marvin Miller of Ohio soon joined Paterson in believing that TBBD existed; he suggested that intrauterine confinement prior to birth might lead to temporarily low bone density in infants because it could prevent bone loading that might

175. *See id.*

176. David Ayoub et al., *Are Paterson's Critics Too Biased to Recognize Rickets?*, 99 ACTA PAEDIATRICA 1282, 1282 (2010).

177. Johnstone, *supra* note 6.

178. *Id.*

179. *Id.*

180. Colin R. Paterson, *Temporary Brittle Bone Disease: Fractures in Medical Care*, 98 ACTA PAEDIATRICA 1935, 1937 (2009).

be essential for normal bone formation during pregnancy.¹⁸¹ Armed with the conviction that many innocent caretakers were being wrongfully convicted, Paterson and Miller both began to testify as defense experts in cases of alleged child abuse involving infantile fractures.¹⁸²

The TBBD theory, however, alarmed a medical community whose constituents had long been indoctrinated with Caffey's notion that unexplained long-bone and rib fractures were pathognomonic for child abuse. A doctor who presented an opinion that conflicted with decades of medical textbooks was viewed with suspicion, especially when he was being paid—no doubt handsomely—to give this opinion in court. Dr. Stephen Chapman and Dr. Christine Hall became two of Paterson's most vehement critics, suggesting that believing in the existence of TBBD was like believing the earth was flat.¹⁸³

181. M.E. Miller & T.N. Hangartner, *Temporary Brittle Bone Disease: Association with Decreased Fetal Movement and Osteopenia*, 64 *CALCIFIED TISSUE INT'L* 137, 140–142 (1999).

182. *See* State v. Talmadge, 999 P.2d 192, 193 (Ariz. 2000) (reversing the lower court's decision to exclude both Paterson and Miller as possible expert witnesses in a case involving unexplained infantile fractures).

183. Johnstone, *supra* note 6. In response to this hyperbolic comparison, it should be pointed out that Paterson's ability to build an evidentiary base for the TBBD theory is constrained by many of the same practical barriers that constrained Caffey and other researchers from building a strong evidentiary base for shaken-baby syndrome. Namely, Paterson could not directly experiment on human infants for obvious ethical reasons, so he was forced to assume, at some level, what he was trying to conclude—that a certain number of infants with unexplained fractures who shared some other characteristics have temporary brittle-bone disease. In light of the weaknesses in the evidentiary base of shaken-baby syndrome, it is very unlikely that shaken-baby syndrome would have survived—much less become a foundational element of modern medical child-abuse diagnostic dogma—if it had been subject to the same level of scientific scrutiny at its inception as TBBD. A thorough investigation of why shaken-baby syndrome was readily accepted and why TBBD was readily rejected by the medical community despite the similar weaknesses in their evidentiary bases is beyond the scope of this Comment. Perhaps one of the key differences is that shaken-baby syndrome did not have to directly supplant a contradictory traditional diagnosis. Rather, the shaken-baby syndrome was a plausible explanation for an array of symptoms that had no firmly entrenched competing explanations. TBBD, by contrast, flew in the face of a competing explanation that held near-biblical status in an ivory tower of medical diagnostic dogma. Furthermore, shaken-baby syndrome was never hampered by the possibility that its alleged victims might have some degree of culpability. Helpless infants who cannot protect themselves, speak, walk, or fairly be held accountable for anything they do clearly need protection and simply cannot be at fault even if they harm themselves or others. Caretakers, on the other hand, are generally adults who can be culpable on all sorts of levels and have some power (at least in theory) to protect themselves. Perhaps our sympathies more readily attach to infants as a result.

The war of opinions about TBBB continues to rage. Hall and Paterson have served as opposing expert witnesses in nearly thirty different alleged child-abuse cases.¹⁸⁴ On different occasions, medical and legal authorities have sharply criticized both Paterson¹⁸⁵ and his most vocal critics, Chapman and Hall,¹⁸⁶ for failing, at times, to be objective in their positions. Understandably, emotions run high on account of the ironic fact that both sides in this war ultimately want to protect innocent people; one side is focused on protecting innocent children, while the other side is focused on protecting innocent but falsely-accused caretakers. The point of contention between the two sides is about how to properly distinguish legitimate cases of abuse from false ones. The TBBB debate is unlikely to be resolved in the near future. However, given that *In re Stephan H.* illustrated that there are yet unknown conditions that can cause low bone density in infants,¹⁸⁷ the existence of TBBB remains a possibility.

IV. THE PROBLEM WITH ASSUMING INDEPENDENCE IN CASES OF MULTIPLE INFANTILE FRACTURES

The existence of the aforementioned conditions that can cause infantile bone fragility without showing radiographic signs other than fractures casts a broad shadow across the longstanding assumption that child abuse can be diagnosed from x-rays alone. In addition to questioning the capacity of normal x-rays to detect bone density, it is also necessary to question Caffey's supposition that the mere presence of multiple fractures in different stages of healing necessarily makes accidental causes highly unlikely. It is also necessary to scrutinize the supposition that the combination of fractures and intracranial hemorrhaging is pathognomonic for abuse.

How the conclusion is reached that a certain combination of injuries is pathognomonic for child abuse is of paramount importance: improper methods could lead to the erroneous conclusion that a given combination is pathognomonic and that no further evidence is necessary to prove abuse (such as in the case of

184. *Id.*

185. JIM FISHER, FORENSICS UNDER FIRE: ARE BAD SCIENCE AND DUELING EXPERTS CORRUPTING CRIMINAL JUSTICE? 91, 96 (2008).

186. Johnstone, *supra* note 6.

187. *In re Stephan H.*, No. L15-CP00-007572-A, 2002 WL 31083579, at *12 (Conn. Super. Ct. Aug. 23, 2002).

the shaken-baby triad). One error in particular that must be avoided is the assumption of independence. It may seem intuitive to think that if injury A has a one-in-ten chance of being caused by an accident and injury B has a one-in-ten chance of being caused by an accident, the chance of both injuries appearing simultaneously on the same person due to accidental causes is one in one hundred. The reality, however, is that this is only correct if injuries A and B are statistically independent of each other.

A. The California Supreme Court's View About the Assumption of Independence and Convictions

Improper assumptions of independence can unfairly bias the administration of justice in a number of contexts. The California Supreme Court has dealt with such assumptions on at least one occasion. In *People v. Collins* witnesses saw an African American man with a beard and a Caucasian woman with a blond ponytail steal a woman's purse and escape in a yellow automobile.¹⁸⁸ The defendant and his wife matched the description of the couple and drove a yellow automobile.¹⁸⁹ The prosecution provided an expert witness, who opined that the probability of there being another couple in the area matching this description could be calculated using the following probabilities: (1) a partly yellow automobile: 1/10; (2) a man with a mustache: 1/4; (3) a girl with a ponytail: 1/10; (4) a girl with blond hair: 1/3; (5) an African American man with a beard: 1/10; and (6) an interracial couple in a car: 1/1000.¹⁹⁰ Assuming that all these descriptive factors were independent, he multiplied the probabilities together using the product rule and opined that the probability that the defendants were innocent was one in twelve million.¹⁹¹ The defendant and his wife were convicted.

The Court reversed the conviction, stating that “[n]o proof was presented that the characteristics selected were mutually independent, even though the [prosecution's] witness himself acknowledged that such condition was essential to the proper application of the ‘product rule’”¹⁹² As a result, the Court explained,

188. 438 P.2d 33, 34 (Cal. 1968).

189. *Id.*

190. *Id.* at 37.

191. *Id.*

192. *Id.* at 39.

“the ‘product rule’ would *inevitably* yield a wholly erroneous and exaggerated result *even if* all the individual components had been determined with precision.”¹⁹³ “[U]nder the circumstances,” the Court explained, “the ‘trial by mathematics’ so distorted the role of the jury and so disadvantaged counsel for the defense, as to constitute in itself a miscarriage of justice.”¹⁹⁴

B. Implicit Assumptions of Independence in Child-Abuse Cases Involving Unexplained Fractures and the Problems They Present

It may seem reasonable to presume that, if the probability that any given infant in the population has a condition that causes bone fragility is one in a million, then the probability that a given infant with unexplained fractures has bone fragility is also one in a million. The problem with this approach, though, is that it assumes that the presence of fractures and the presence of bone fragility are statistically independent of one another—that is, the presence of one does not make the other more likely. It should be intuitive, however, that the presence of bone fragility and the presence of bone fractures are most likely *not* independent because infants with fragile bones are much more likely to suffer fractures than their normal counterparts.¹⁹⁵ One should expect that the set of infants with bone fragility would be overrepresented in the set of infants with fractures. In fact, the record in the *Velasquez* case strongly suggests that such overrepresentation exists.¹⁹⁶ In that case, the child was one of eleven who tested positive for OI in a larger study of 262 infants that authorities suspected were victims of abuse.¹⁹⁷ (Keep in mind that skin biopsy OI tests only accurately identify cases of OI eighty-five percent of the time.)¹⁹⁸ Furthermore, in that same study, there were

193. *Id.* (emphasis added).

194. *Id.* at 41.

195. To use a more intuitive example, consider the likelihood that a person randomly selected from the population is over six feet six inches tall versus the likelihood that a person randomly selected from the set of all NBA players is over six feet six inches tall. Clearly, the likelihood is much greater in the latter selection because the presence of a certain nonindependent factor is obvious. While not all people over six foot six—or even the majority of them—play in the NBA, they are a set of people that is heavily overrepresented within the set of all NBA players.

196. *Velasquez v. Goodwin*, No. 0033-03-4, 2004 WL 1773647 (Va. Ct. App. Aug. 10, 2004).

197. *Id.* at *2.

198. Paterson, *supra* note 160.

eleven more children tested for whom the diagnosis of OI “could not be excluded.”¹⁹⁹ If the incidence of children with OI in the general population is one in sixteen thousand²⁰⁰ and the incidence of OI in the set of children with unexplained fractures is somewhere between eleven in 262 and twenty-two in 262, the incidence of OI is somewhere between 671 and 1343 times greater²⁰¹ in the set of infants with unexplained fractures than it is in the general population.²⁰² If one accounts for the fifteen percent false negative rate of the OI test used in the study, the actual incidence is likely even higher. And remember, OI was the only condition tested for out of the many conditions that can cause infantile bone fragility.

In light of the lack of independence between bone fractures and bone fragility, due consideration should also be given to the possibility that multiple fractures in different stages of healing are not independent of one another. Underlying bone fragility does not presumably disappear once a single fracture has occurred, so it is also likely that infants with bone fragility are overrepresented in the set of infants with *multiple* fractures just as it is likely that they are overrepresented in the set of infants with single fractures. As a result, it does not necessarily follow that the presence of multiple fractures in the same infant makes an abusive cause more likely than the presence of only one fracture.

What may not be as intuitive is that bone fragility and intracranial hemorrhaging are also probably not independent—a significant consideration, given that the combination of fractures and intracranial hemorrhaging is commonly considered to be pathognomonic for abusive shaking. First, and most obviously, intracranial hemorrhaging often occurs from injuries sustained due

199. A. Marlowe et al., *Testing for Osteogenesis Imperfecta in Cases of Suspected Non-accidental Injury*, 39 J. MED. GENETICS 382, 383 (2002).

200. Marini, *supra* note 156.

201. $(1/16,000)x = 11/262$. Solving for x in this equation yields a result of 671.756. If 22 is substituted for 11, the solution for x is 1343.51.

202. Because child abuse was suspected in all these cases, the National Center for the Prosecution of Child Abuse’s assertions that (1) “the likelihood of OI presenting without typical symptoms in a way likely to be indistinguishable from child abuse is approximately 1 in 3,000,000” and (2) that “[s]tatistically, it makes no sense for the defense to claim that OI can easily be mistaken for child abuse” cannot withstand scrutiny. Joëlle Anne Moreno, *Einstein on the Bench?: Exposing What Judges Do Not Know About Science and Using Child Abuse Cases to Improve How Courts Evaluate Scientific Evidence*, 64 OHIO ST. L.J. 531, 574–75 (2003) (citation omitted) (internal quotation marks omitted).

to skull fractures²⁰³—a fact even acknowledged by Caffey.²⁰⁴ Thus, a person whose bones are more likely to get fractured—from accidental or nonaccidental causes—might also be more likely to have a skull fracture and, therefore, more likely to have intracranial hemorrhaging as a result.

Second, and more importantly, fractures in other parts of the body and intracranial hemorrhaging are likely not independent. At the time of his 1946 study, Caffey was apparently unaware of this. Indeed, Caffey believed that the apparent lack of a logical relationship between these factors suggested that they were independent, and that the combination of the two conditions was therefore highly suspicious for trauma.²⁰⁵ It is now understood, however, that many of the conditions that can cause bone fragility are also linked to intracranial hemorrhaging even in the absence of skull fractures. For example, OI causes diminished vascular strength and diminished platelet function (platelets are involved in clotting). This combination makes the blood vessels within the brain more likely to rupture and makes the blood less likely to clot effectively, thereby increasing the likelihood of intracranial hemorrhaging.²⁰⁶ Furthermore, biliary atresia,²⁰⁷ bile-duct defects,²⁰⁸ and even less-severe subclinical liver dysfunction²⁰⁹ are associated with vitamin K deficiency because vitamin K, like vitamin D, is a fat-soluble vitamin

203. Steven A. Shane & Susan M. Fuchs, *Skull Fractures in Infants and Predictors of Associated Intracranial Injury*, 13 PEDIATRIC EMERGENCY CARE 198, 198 (1997).

204. Caffey, *supra* note 7, at 172.

205. See *id.*

206. W.G. Cole & T.P. Lam, *Arachnoid Cyst and Chronic Subdural Haematoma in a Child with Osteogenesis Imperfecta Type III Resulting from the Substitution of Glycine 1006 by Alanine in the Pro Alpha 2(I) Chain of Type I Procollagen*, 33 J. MED. GENETICS 193, 193 (1996); Anuradha Ganesh et al., *Retinal Hemorrhages in Type I Osteogenesis Imperfecta After Minor Trauma*, 111 OPHTHALMOLOGY 1428 (2004); Deann Sasaki-Adams et al., *Neurosurgical Implications of Osteogenesis Imperfecta in Children*, 1 J. NEUROSURGERY: PEDIATRICS 229 (2008).

207. Hideyuki Akiyama et al., *Intracranial Hemorrhage and Vitamin K Deficiency Associated with Biliary Atresia: Summary of 15 Cases and Review of the Literature*, 42 PEDIATRIC NEUROSURGERY 362, 362 (2006).

208. Tilman Humpl et al., *Fatal Late Vitamin K-Deficiency Bleeding After Oral Vitamin K Prophylaxis Secondary to Unrecognized Bile Duct Paucity*, 29 J. PEDIATRIC GASTROENTEROLOGY & NUTRITION 594, 594 (1999).

209. Ichiro Matsuda et al., *Late Neonatal Vitamin K Deficiency Associated with Subclinical Liver Dysfunction in Human Milk-fed Infants*, 114 J. PEDIATRICS 602, 604 (1989).

which bile helps the body absorb.²¹⁰ Vitamin K deficiency, in turn, can cause spontaneous intracranial hemorrhaging.²¹¹ Scurvy can also cause bone fragility and “subdural hemorrhag[ing] has been conclusively demonstrated as a complication of [scurvy].”²¹² Additionally, Menkes disease, which is also linked to bone fragility, can cause spontaneous intracranial hemorrhaging.²¹³

The existence of multiple conditions that can predispose infants to both multiple fractures and intracranial hemorrhages is highly significant because it suggests a lack of independence between these types of injuries. The defendant in *People v. Collins* did not even have to make a showing that there were known phenomena that might call into question the independence of traits such as having a moustache or driving a yellow car. The prosecution’s mere unsupported assumption of independence itself was enough to justify reversing the conviction.²¹⁴ The knowledge that there are conditions that can cause all the injuries associated with a combination thought to be essentially pathognomonic for child abuse makes the case against convicting caretakers solely on the basis of that combination even stronger. A “trial by mathematics”²¹⁵ based on combined probabilities—whether implicitly or explicitly stated—that assume multiple fractures are mutually independent of each other or of intracranial hemorrhaging can therefore unfairly bias the administration of justice.

But the lack of independence between a combination of fractures and hemorrhages does not necessarily mean that it is more likely that an infant was not abused. However, the crime of child abuse must be proven beyond a reasonable doubt. While courts have consistently rejected any numerical definition of what percentage of certainty meets the standard of reasonable doubt, the following example

210. See Martin J. Shearer et al., *Chemistry, Nutritional Sources, Tissue Distribution and Metabolism of Vitamin K with Special Reference to Bone Health*, 126 J. NUTRITION, at 1181S, 1184S (1996).

211. G.N. Ruddy et al., *Late Form Hemorrhagic Disease of the Newborn: A Fatal Case with Illustration of Investigations that May Assist in Avoiding the Mistaken Diagnosis of Child Abuse*, 20 AM. J. FORENSIC MED. & PATHOLOGY 48, 48 (1999).

212. Theodore Hunt Ingalls, *The Role of Scurvy in the Etiology of Chronic Subdural Hematoma*, 215 NEW ENG. J. MED. 1279, 1281 (1936).

213. Marie-Cécile Nassogne et al., *Massive Subdural Haematomas in Menkes Disease Mimicking Shaken Baby Syndrome*, 12 CHILD’S NERVOUS SYS. 729, 731 (2002).

214. *People v. Collins*, 438 P.2d 33, 39 (Cal. 1968).

215. *Id.* at 41.

illustrates how small percentages can matter a great deal when lives are at stake. The Federal Aviation Administration “handles more than 70,000 flights per day.”²¹⁶ If 0.5% of those flights crashed, there would be 350 plane crashes per day. How many consumers would feel that it was safe to fly beyond a reasonable doubt if there were that many crashes per day?

This analogy should not be taken too far, however. A 0.5% margin of error in this hypothetical would result in more lives being affected than an equal margin of error in child abuse convictions because there are many more plane flights than trials for child abuse. However, in child abuse cases, innocent people’s lives can be destroyed in a number of ways when wrongful convictions are made. Because of the possibility of wrongful convictions, even a small likelihood that single and multiple fractures are not pathognomic for abuse should be taken very seriously by our justice system.²¹⁷

V. REQUIRED BONE-DENSITY TESTING: A SIMPLE WAY TO PROTECT BOTH INNOCENT INFANTS AND INNOCENT CARETAKERS

The war to protect innocent caretakers and innocent children does not have to be a zero-sum game. Both sides can win if the legislative and judicial processes can be used to create laws that require bone-density tests—not just x-rays—to be administered in every case where unexplained fractures are used as evidence of child abuse. Such tests would focus directly on the best evidence: the bones themselves. These tests could definitively establish in each case

216. Vaynu Osuri, *Successes of Collaborative Decision Making at the Traffic Flow Management Program Office and the Advantages of Adopting Toolkits 12* (May 2006) (Master’s Thesis, Massachusetts Institute of Technology), *available at* <http://dspace.mit.edu/handle/1721.1/35104>.

217. It is difficult to estimate precisely how many wrongful convictions do occur because of unexplained infantile fractures. However, if we assume that one-fifth of all children under the age of five in the United States are between zero and eleven months old, census data from the year 2000 would suggest that there are about 3,800,000 children in that age range. *See Age: 2000*, CENSUS.GOV 4 (Oct. 2001), <http://www.census.gov/prod/2001pubs/c2kbr01-12.pdf> (19,175,798 children under the age of five). If the incidence of suspicious infantile fractures in this age range is 48.3 per 100,000 children, one could expect there to be about 1835 suspicious cases in the United States per year. *See* John M. Leventhal et al., *Incidence of Fractures Attributable to Abuse in Young Hospitalized Children: Results From Analysis of a United States Database*, 122 PEDIATRICS 599, 600 (2008) (estimating rate of suspicious fracture cases for children of zero to eleven months of age to be 48.3 per 100,000). Assuming caretakers would be convicted in all 1835 cases and that eleven in every 262 of those caretakers would be factually innocent, there would be about seventy-seven wrongful convictions in the United States per year.

whether an infant with fractures actually did have low bone density. Fortuitously, in cases where low bone density was found, there would not be a need to discover its precise cause in order for it to be relevant to the legal defense of innocent caretakers. This would help promote justice, since there are some cases—such as the *In re Stephan* case, upon which the hypothetical at the beginning of this Comment is based—in which the cause might not yet be known to medical literature. In cases where intracranial hemorrhaging is present along with low bone density, such testing could also alert doctors of the need to look for underlying conditions that are linked to both fractures and hemorrhages. If tests show that an injured infant does not have low bone density, then an abusive caretaker would not be able to cast doubt on the prosecution's evidence by claiming that the injuries were due to other factors. An ideal bone density test for infants is the single-photon absorptiometry method; single-photon absorptiometers (SPAs) have been widely used for many years²¹⁸ and their use is “a well substantiated technique for measuring bone density in children.”²¹⁹ In a study unrelated to child abuse, researchers who needed to measure the bone density of many children explained:

The affordability, low maintenance cost, and ease of use of a modern single photon absorptiometer should help to make bone mineral density measurements readily available. Newer single photon absorptiometers do not require handling of radioactive material and require only minimal training for operation. No trained technician is required, and single photon absorptiometers are typically used by office or clinic personnel.²²⁰

Some SPAs are portable,²²¹ so they could be shared between multiple clinics or hospitals. This could further lower the cost of compliance with new laws requiring bone density tests in cases of suspicious infantile fractures.

218. Robert M. Neer, *The Utility of Single-Photon Absorptiometry and Dual-Energy X-ray Absorptiometry*, 33 J. NUCLEAR MED. 170, 170 (1992), available at <http://jnm.snmjournals.org/cgi/reprint/33/1/170.pdf>.

219. Albert Quan et al., *Bone Mineral Density in Children with Myelomeningocele*, PEDIATRICS 4 (Sept. 1998), <http://pediatrics.aappublications.org/content/102/3/e34.full.pdf>.

220. *Id.* at 5.

221. See J.G. Truscott et al., *A Portable System for Measuring Bone Mineral Density in the Pre-term Neonatal Forearm*, 69 BRIT. J. RADIOLOGY 532, 532 (1996).

In addition to accuracy and affordability, SPAs have another imperative advantage: some portable models can be used on infants whose health is so fragile that they cannot be moved. In one study unrelated to child abuse, for example, researchers used a compact SPA to measure the bone density of premature infants without even having to remove the infants from their incubators.²²² Since infants with bone fragility can be susceptible to suffering new fractures even when they are being passively moved by hospital personnel, a system that obviates the need to move infants with fractures is highly desirable.

As an alternative, a dual x-ray absorptiometry (DXA) scanner might be a reasonable alternative; a DXA scanner was used in the *In Re Stephan* case.²²³ However, proper calibration would be absolutely essential; DXA scanners that are calibrated for adults are not accurate on infants.²²⁴ Standard x-rays, however, should not be considered an adequate alternative because of their notorious inaccuracy in assessing bone density.²²⁵

An ideal law would require that a bone-density scan be performed as soon as suspect fractures are identified. If a significant amount of time passes between the incidence of the fractures and the scan, then the bone mineralization in the infant at the time of the scan might not be representative of the bone mineralization at the time of the fractures. Tests for OI and other collagen disorders should also be administered separately, if possible, since up to forty percent of infants with OI have normal bone density in spite of their bone fragility.²²⁶

It may even be prudent for the legal system to require that all infants have such a bone scan at birth. This could help identify children with low bone density early enough to alert caretakers and doctors that certain babies must be handled more carefully so that accidental fractures could be more effectively avoided in the first place. It may also alert doctors that they need to look for underlying disorders that a given child with low bone density might have, such

222. *Id.* at 537.

223. *In re Stephan H.*, No. L15-CP00-007572-A, 2002 WL 31083579, at *12 (Conn. Super. Ct. 2002).

224. See Larry A. Binkovitz et al., *Pediatric DXA: Technique, Interpretation, and Clinical Applications*, 38 PEDIATRIC RADIOLOGY S227, S227-28 (Supp. 2 2008).

225. Johnston & Melton, *supra* note 115.

226. Binkovitz, *supra* note 224, at S234.

as liver problems. If such disorders were then found in a given case, the infant might receive the necessary lifesaving treatment as promptly as possible. Lastly, if an infant born in the jurisdiction was subsequently hospitalized with multiple fractures and intracranial hemorrhaging, doctors would have a valuable piece of clinical history at hand that would help them more accurately assess the likelihood of abuse.

VI. CONCLUSION

Ultimately, researchers may have little power to prove or disprove by direct evidence whether child abuse is the cause of unexplained infantile fractures in a given case because of ethical and practical constraints. Medical practitioners do, however, have the technology to prove, by direct evidence and with a high degree of accuracy, whether a given infant with fractures has low bone density that may have predisposed the child to fractures during nonabusive handling. Mandating the use of this commonly available and relatively inexpensive technology would close a major evidentiary gap, which, ironically, allows room for both innocent caretakers to be convicted and guilty caretakers to be acquitted. Our understanding of nonabusive conditions that can mimic child abuse has evolved to the point where it is simply no longer appropriate to presume child abuse based solely on the presence of unexplained fractures—even when those fractures are paired with intracranial hemorrhaging. Since the technology to measure infant bone density exists and is relatively ubiquitous, its use should be mandated in cases where unexplained fractures will be used as evidence of abuse so that “beyond a reasonable doubt” will mean in practice what it says on paper in cases of alleged infant abuse.

*Matthew B. Seeley**

* J.D., April 2011, J. Reuben Clark Law School, Brigham Young University.