



1-1-1996

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Thomas D. Lyon

University of Southern California

Elizabeth E. Gilles

Ohio State University

Larry Cory

Los Angeles County Counsel's Office

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Medical Evidence of Physical Abuse in Infants and Young Children

Thomas D. Lyon*
Elizabeth E. Gilles, M.D.**
Larry Cory***

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* Assistant Professor of Law, University of Southern California. J.D., 1987, Harvard University; Ph.D., 1994, Stanford University. Correspondence concerning this Article should be sent to the first author. The first author’s address is: University of Southern California Law School, University Park, Los Angeles, CA 90089-0071. E-mail address is: tlyon@law.usc.edu. Thanks to Dr. Ralph S. Lachman, Professor of Pediatric Radiology at Harbor-UCLA Medical Center, for reviewing a draft of Parts I, II, and IV, and to Gary Seiser, for reviewing an earlier draft of the entire Article.

** Assistant Professor of Pediatrics and Neurology/Ohio State University, Columbus Children’s Hospital/ Division of Neurology, 700 Children’s Drive, Columbus, Ohio 43205-2696, (614) 722-4638.

*** Division Chief, Children’s Services Division, Los Angeles County Counsel’s Office. The views expressed herein do not necessarily reflect those of the Los Angeles County Counsel’s Office.

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I. INTRODUCTION

Determining whether a young child's injuries are due to physical abuse is often extremely difficult. Frequently, the child is nonverbal, and there are no witnesses other than the caretakers that are suspected of abuse. Expert medical opinion is often necessary to diagnose abuse. However, the process by which physicians diagnose physical abuse is something of a mystery to many attorneys, even to those who routinely handle such cases. The medical literature is often impenetrable to those without special training, leading attorneys to defer to expert opinion without fully understanding the basis for such opinion. This is unfortunate. Without understanding the research that underlies expert medical judgment, an attorney can neither make full use of the physician's expertise, nor adequately cross-examine an opposing expert.

The purpose of this Article is to provide attorneys with a basic understanding of the current research literature on the diagnosis of physical abuse in infants and young children.¹ Physicians consider several factors, including the explanation for the injury provided by the caretakers (the "history"), whether there are other injuries indicative of abuse (*e.g.*, fractures, bruising), the types of injury, and the locations of the injury. Recognition of the pattern of injuries is helpful in both confirming the intention behind the injury and giving valuable clues as to the instrument used or the mechanism of injury. This, in turn, can sometimes assist in the identification of the perpetrator. If accidents (such as a fall) and disease are ruled out, then physicians are confident in stating that the child's injury is "nonaccidental," that is, due to abuse.

Part II discusses the importance of the caretakers' explanation in diagnosing abuse. Subsequent parts focus on specific injuries and the circumstances under which they should be attributed to abuse. Part III focuses on bruises; Part IV covers skeletal fractures. Finally, Part V discusses injuries inside the skull, also known as intracranial injuries. Because attorneys are often unfamiliar with medical terminology, definitions of frequently used terms are provided at the end of this part, and in Parts III, IV, and V.

In each part, we discuss the likelihood that various types of injuries are attributable to abuse. The following terminology is used. In medical literature, commentators often say that certain conditions are "strongly associated" with abuse or "highly suggestive" of abuse. In this Article, we call such conditions "highly suggestive" of abuse. Conditions described as "highly suggestive" of abuse are much more common among abused children than among nonabused children, are not attributable to minor trauma (such as falls off a bed or couch), and should lead one to conclude that abuse occurred unless the condition may be attributable to birth trauma, an auto accident, a serious fall (more than ten feet), or a disease. "Highly suggestive" is also used to refer to conditions that some physicians call "pathognomonic" of abuse, or "specific" to abuse, terms that suggest that the condition in question could only have been caused by abuse. As we discuss, very few (if any) markers of injury are pathognomonic of abuse.

When a condition occurs more frequently among abused children than among nonabused children, we refer to the condition as "supportive" of abuse. Such a condition increases the likelihood that a child was abused, and may lead an expert to conclude that abuse occurred when taken together with other suspicious findings. By

1. The Article is not intended to be exhaustive, and multiple references are included for further reading. In addition, reviews of material not covered (such as burn and bite injuries) can be found elsewhere. *See generally* THE BATTERED CHILD (Ray Helfer & Ruth S. Kempe eds., 4th ed. 1987); CHILD MALTREATMENT: A CLINICAL GUIDE AND REFERENCE (Joan C. Reinbott et al. eds., 1994); ROBERT M. REECE, CHILD ABUSE: MEDICAL DIAGNOSIS AND MANAGEMENT (1994).

itself, however, a condition supportive of abuse does not always lead to the conclusion that abuse occurred.²

Glossary of terms:³

Clinical course: The onset and progression of clinical signs and symptoms over time. Includes medical and surgical interventions and the subsequent clinical response.

Differential: The identification of a patient's condition or disease.

Etiology: Cause.

Intentional injury: An injury due to abuse. Also called inflicted injury or nonaccidental injury. As used here, the term "intentional" does not require a specific intent to injure.

Pathogenesis: The development of a condition or disease.

Sign: Objective evidence of a disease or condition (*i.e.*, a bruise).

Symptoms: Functional evidence of a disease or condition (*i.e.*, coughing).

Syndrome: A set of symptoms occurring together; a symptom complex.

Unintentional injury: Accidental injury.

II. THE CARETAKER'S EXPLANATION

Doctors rarely diagnose abuse solely on the basis of the appearance of an injury, but rely on a combination of the history, physical examination, and laboratory and imaging findings. It is essential to ask the caretakers how the injury occurred, and to determine if the explanation is either consistent or inconsistent with the injury. Deciding whether an injury could have happened the way the caretakers say it did depends on having a thorough understanding of how different kinds of injuries occur (their pathogenesis). Physicians often diagnose abuse on the grounds that the caretakers gave a history that was inconsistent with the injury, vague, or unclear;

2. We avoid use of the term "consistent with" abuse in this Article because there appears to be some disagreement among expert witnesses regarding whether the term is synonymous with "supportive of" or merely "not inconsistent with."

3. The definitions herein are the authors', with the assistance of various medical sources. For further medical definitions, we recommend DORLAND'S ILLUSTRATED MEDICAL DICTIONARY (28th ed. 1994).

changed over time; or varied depending upon which family member was asked.⁴

Often, the caretakers have no explanation for the injury, but report that the infant or young child appeared injured or behaved unusually. In several studies comparing abused and nonabused children, a much higher percentage of abused children are brought in with symptoms but without an explanation.⁵ One study found that in cases in which children were presented with no history, 80% were subsequently determined to be due to abuse.⁶

In all cases, it is important to consider the possibility that an alternate caretaker injured the child. Has the child been with a babysitter or other caregiver? When did this occur? It may be possible to rule out alternative caretakers by the dating of the injury and by developing a timeline of events. Dating of bruises is discussed in Part III, dating of fractures in Part IV, and dating of intracranial injuries in Part V.

III. BRUISES

A. *Glossary of Terms*

Abrasion: Scratch of the epidermis (the superficial part of the skin).

Blunt force trauma: Impact force.

Contusion: A bruise. Damage to the dermal part of the skin due to blunt force trauma with rupture of capillaries (small blood vessels).

Laceration: A tearing of the skin from blunt force trauma. Differentiated from an incised wound from a blade.

4. Helen M. L. Carty, *Fractures Caused by Child Abuse*, 75-B J. BONE & JOINT SURGERY 849 (1993) [hereinafter Carty, *Fractures Caused by Child Abuse*]; John M. Leventhal et al., *Fractures in Young Children: Distinguishing Child Abuse from Unintentional Injuries*, 147 AM. J. DISEASES CHILDREN 87, 89-90 (1993).

5. Leventhal et al., *supra* note 4, at 91 (52% of abused children with no explanation versus 3% of nonabused children); Frederick P. Rivara et al., *Injuries to Children Younger than 1 Year of Age*, 81 PEDIATRICS 93, 94 (1988) (36% of abused children with no explanation versus 0% of nonabused children); Norman Rosenberg & Gerald Bottenfield, *Fractures in Infants: A Sign of Child Abuse*, 11 ANNALS EMERGENCY MED. 178 (1982) (45% of abused children with no explanation versus 0% of nonabused children).

6. Leventhal et al., *supra* note 4, at 90.

B. Bruises and Abuse

Bruises are the most common manifestation of physical abuse,⁷ but are also a typical consequence of accidental falls and bumps. In order to distinguish between abusive and nonabusive bruising, one considers the character, number, and location of the bruises, the child's level of motor development (*i.e.*, can the child crawl, walk, climb?), and the history given by the child's caretakers as to how the bruising occurred.

As a general rule, children who cannot walk either on their own or with support (generally up to nine months of age) are rarely bruised.⁸ "Bruising seen in children at this stage of development should be taken seriously, and a good explanation sought for the bruises before the bruises are accepted as accidental."⁹ Once children become mobile, they are subject to injury from minor falls and from bumping into objects, and bruise frequently.¹⁰ Injuries from unintentional falls and bumps lead to bruising over the "bony prominences," where the bone is closer to the surface of the skin (*e.g.*, the knees as opposed to the thighs).¹¹ The most common accidentally injured parts of the body are the knees and shins.¹²

The most suspicious locations for bruises are the back of the head, the face

7. See Edward Goldson et al., *Nonaccidental Trauma and Failure to Thrive: A Sociomedical Profile in Denver*, 130 AM. J. DISEASES CHILDREN 490, 491-92 (1976) (140 children abused or with failure to thrive or both: 53% had only bruises, and bruises are the most common injury observed); Allan E. Kornberg, *Skin and Soft Tissue Injuries*, in CHILD ABUSE: A MEDICAL REFERENCE 91, 91 (Stephen Ludwig & Allan E. Kornberg eds., 2d ed. 1992) ("The skin is the most common site of involvement of physical abuse."); Eric B. Leavitt et al., *Otolaryngologic Manifestations of Child Abuse*, 118 ARCHIVES OTOLARYNGOLOGICAL HEAD & NECK SURGERY 629, 629 (1992) (85 children admitted to hospital for abuse or neglect, mean age four years); *id.* at 631 ("The skin is by far the most frequently traumatized system.").

8. See D.M. Robertson et al., *Unusual Injury? Recent Injury in Normal Children and Children With Suspected Non-Accidental Injury*, 285 BRIT. MED. J. 1399, 1400 (1982) (400 nonabused children up to 11 years of age seen at routine pediatric visit: 4/60 or 7% of children up to two months had bruises, abrasions or minor lacerations; 8/62 or 13% of children three to nine months of age showed evidence of injury); *id.* at 1401 ("Injuries of any type are uncommon before nine months, with a steady increase to a plateau at three years."); John Wedgwood, *Childhood Bruising*, 234 PRAC. 598, 600 (1990) (56 nonabused children in hospital under four years of age, 11 of whom could only crawl; "children who can only crawl seem to show no bruising whatsoever . . . The average number of bruises increased as motor development improved.").

9. Wedgwood, *supra* note 8, at 600.

10. See Robertson et al., *supra* note 8, at 1400 (400 nonabused children up to 11 years of age seen at routine pediatric visit: 37% had some sort of injury, most commonly bruises; over 50% if 18 months or older).

11. See John M. Pascoe et al., *Patterns of Skin Injury in Nonaccidental and Accidental Injury*, 64 PEDIATRICS 245, 247 (1979) ("In preschool and school aged children with accidental injuries, lesions are usually not found on the 'soft' sites such as cheeks, trunk, genitals and upper legs.").

12. See Pascoe et al., *supra* note 11, at 246 (438 children: 223 abused children with soft tissue injury, 110 nonabused seen in emergency room, 105 nonabused seen in routine visit; "[s]oft tissue injuries of the knee and shin were noted more frequently [in the routine visit group than in the abused group]"); Robertson et al., *supra* note 8, at 1400 (400 nonabused children up to 11 years of age seen at routine pediatric visit: over 20% of children 19 months or older had lower leg injuries); Barton D. Schmitt, *The Child with Nonaccidental Trauma*, in THE BATTERED CHILD, *supra* note 1, at 186; Wedgwood, *supra* note 8, at 600 (56 nonabused children in hospital under four years of age; 59% of children had bruising of knees and shins).

(excluding the forehead), the buttocks and thighs, the lower back (or lumbosacral region), and the genitalia. Bruises to the head and face are much more common among abused children than among nonabused children.¹³ In general, unintentional bruising to the face is rare in children under one year of age,¹⁴ becomes fairly common among children eighteen months to three years of age, and becomes rare again thereafter.¹⁵ When accidental bruises to the face do occur, they tend to be over bony prominences, such as the forehead and over the cheekbones, common points of injury when a child falls or runs into an object.¹⁶ In young children, bruises to the ear, the soft portions of the face, and the neck are highly suggestive of abuse.¹⁷

The buttocks and thighs are common sites of abusive injury; nonabused children infrequently exhibit such bruises.¹⁸ They are therefore supportive of abuse, particularly among children less than eighteen months of age.¹⁹ The lower back or

13. Leavitt et al., *supra* note 7, at 630 (85 abused children admitted to hospital; 32% with injuries to the face); Pascoe et al., *supra* note 11, at 246 (438 children; 223 abused children with soft tissue injury, 110 nonabused seen in emergency room, 105 nonabused seen in routine visit; cheek injuries in 31% of abused, 1% of nonabused in emergency room, 1% of nonabused in routine visit); Robertson et al., *supra* note 8, at 1401 (484 children up to 11 years of age, 84 physically abused, 400 seen for routine pediatric visit; injuries to head and face in 60% of abused, 7% nonabused).

14. Patricia E. Mortimer & Myra Freeman, *Are Facial Bruises in Babies Ever Accidental?*, 58 ARCHIVES DISEASE CHILDHOOD 75, 76 (1983) (20 children under one year of age at clinic; six with facial bruises, two in children who had learned how to walk; "facial bruising is rare in infancy"); Robertson et al., *supra* note 8, at 1400 (400 children up to 11 years of age seen at routine pediatric visit; less than 5% of 122 children up to nine months old had injuries to face or head).

15. Robertson et al., *supra* note 8, at 1400 (400 nonabused children up to 11 years of age at routine visit; "[t]he peak prevalence [of head and facial bruising] was between 18 months and [three] years with one in six children being affected").

16. Mortimer & Freeman, *supra* note 14, at 76 (620 nonabused children under one year of age; six had facial bruises, all of them "on the central forehead, or point of the chin and showed, or had shown, swelling due to 'hard, contact' injury. They were quite dissimilar to the . . . bruises seen in child abuse."); Schmitt, *supra* note 12, at 180 ("Accidental falls rarely cause bruises to the soft tissue of the cheek, but instead involve the skin overlying bony prominences such as the forehead or cheekbone.").

17. Kenneth W. Feldman, *Patterned Abusive Bruises of the Buttocks and the Pinnae*, 90 PEDIATRICS 633, 635-36 (1992) (rim of bruises on the top of the ear is a strong indicator of physical abuse; the ears are not frequently injured accidentally); Leavitt et al., *supra* note 7, at 630 (85 abused children admitted to hospital; 25% had ear injury); Pascoe et al., *supra* note 11, at 247 (438 children, 223 abused children with soft tissue injury, 110 nonabused seen in emergency room, 105 nonabused seen in routine visit; injuries to the neck in 12% of abused children; 0% of both nonabused groups); Schmitt, *supra* note 12, at 180 ("Bruises on the cheek are usually secondary to being slapped.").

18. Pascoe et al., *supra* note 11, at 247 (438 children, 223 abused children with soft tissue injury, 110 nonabused seen in emergency room, 105 nonabused seen in routine visit; 27% of abused children had bruising of buttocks, 0% of nonabused emergency room, 3% of nonabused routine visit); Robertson et al., *supra* note 8, at 1400 (484 children up to 11 years of age, 84 physically abused, 400 seen for routine pediatric visit; 42% of abused children had bruising of thigh and buttocks, 9% of nonabused children); see Norman S. Ellerstein, *The Cutaneous Manifestations of Child Abuse and Neglect*, 133 AM. J. DISEASES CHILDREN 906, 906 (1979); Feldman, *supra* note 17, at 635; Schmitt, *supra* note 12, at 180.

19. Robertson et al., *supra* note 8, at 1400 (400 nonabused children up to 11 years of age seen at routine pediatric visit; 2.4% of nonabused children under 18 months had bruising to buttocks, 17% of children over 18 months); Wedgwood, *supra* note 8, at 600 (56 nonabused children in hospital under four years of age; less than 1% of all children had bruises on buttocks).

lumbosacral region is also a common location for abusive bruising and an uncommon site of injury among nonabused children. Bruises to the lumbosacral region are therefore also supportive of abuse, particularly among children less than three years of age.²⁰

Bruises to the genitalia are highly suggestive of abuse unless there is a clear history explaining the injury. Although injuries to the genitalia are not as frequently encountered in physical abuse as injuries to other parts of the body, they are highly suspicious because they only rarely occur accidentally.²¹ Sexual abuse should also be considered in the differential diagnosis of these injuries.

Bruises are more suspicious if they are not merely in suspicious locations, but are also of different ages.²² "The color of a contusion changes from blue-red-purple through green, yellow, and brown as hemoglobin is broken down."²³ Bruises may begin to turn yellow and/or green after twenty-four hours,²⁴ and tend to completely resolve (disappear) in one to four weeks.²⁵ Precise dating of bruises is difficult, due

20. Pascoe et al., *supra* note 11, at 247 (438 children, 223 abused children with soft tissue injury, 110 nonabused seen in emergency room, 105 nonabused seen in routine visit; bruises to lower back in 25% of abused children, 0% of nonabused emergency room, and 5% of routine visit); Robertson et al., *supra* note 8, at 1400-01 (484 children up to 11 years of age, 84 physically abused, 400 seen for routine pediatric visit; bruises to lumbar in 21% of abused children, 4% of nonabused; less than 1% of nonabused children under three had bruising of lumbar, 14% of school-aged children; "[i]njury to the lumbar region differed in age distribution in comparison with normal children, being much more common [among abused children than nonabused children] between 9 months and five years"); Wedgwood, *supra* note 8, at 601 (56 nonabused children in hospital under four years of age; "[b]ruising of the lower back was not seen in children with poor motor development but did occur in very mobile children"; 0% of children with median ages up to 17 months, 5% of children with median age of 25 months, and 18% of children with median age three years three months); see Schmitt, *supra* note 12, at 180.

21. Pascoe et al., *supra* note 11, at 247 (438 children, 223 abused children with soft tissue injury, 110 nonabused seen in emergency room, 105 nonabused seen in routine visit; bruises to genitalia among 7% of abused children, 0% of emergency room, 0% of routine visit); Wedgwood, *supra* note 8, at 600 (56 nonabused children in hospital under four years of age; less than 1% of all children had genital bruising); see Schmitt, *supra* note 12, at 180 ("[G]enital or inner thigh bruises are usually inflicted for toileting mishaps").

22. Ellerstein, *supra* note 18, at 906; Anthony Shaw, *Surgical Management in Child Abuse*, in CHILD ABUSE 180 (Eli H. Newberger ed., 1982).

23. E.F. Wilson, *Estimations of the Age of Cutaneous Contusions in Child Abuse*, 60 PEDIATRICS 750, 751 (1977). "Free hemoglobin appears red. Biliverdin and bilirubin appear green to yellow. Hemosiderin appears yellow to brown." *Id.*

24. Terrence Stéphenon & Yvona Bialas, *Estimation of the Age of Bruising*, 74 ARCHIVES DISEASE CHILDHOOD 53 (1996) (examination of photographs of 36 bruises among 23 children). A number of medical sources have asserted that bruises begin to turn yellow and/or green after about five days. See Wilson, *supra* note 23 (summarizing five sources); see also F.E. CAMPS, GRADWOHL'S LEGAL MEDICINE 265 (3d ed. 1976); CYRIL J. POLSON & D.J. GEE, THE ESSENTIALS OF FORENSIC MEDICINE 97-105 (3d ed. 1985) (greenish tinge by end of one week; Schmitt, *supra* note 12, at 192 (green-yellow within five to 10 days, resolution in two to four weeks); Werner U. Spitz, *Blunt Force Injury*, in MEDICOLEGAL INVESTIGATION OF DEATH: GUIDELINES FOR THE APPLICATION OF PATHOLOGY TO CRIME INVESTIGATIONS 124, 128 (Werner U. Spitz & Russell S. Fisher eds., 2d ed. 1974) (greenish yellow by end of one week). However, these sources have been criticized as based on clinical experience rather than research. See Ari J. Schwartz & Lawrence R. Ricci, *How Accurately Can Bruises Be Aged in Abused Children? Literature Review and Synthesis*, 97 PEDIATRICS 254 (1996).

25. See CAMPS, *supra* note 24, at 265 (resolution with 14-15 days); POLSON & GEE, *supra* note 24, at 109 (resolution within a month); Kornberg, *supra* note 7, at 92 (resolution in two to four weeks); Schmitt, *supra* note 12, at 192 (resolution in two to four weeks); Spitz, *supra* note 24, at 128 (resolution in two weeks to one month).

to the influence of various factors,²⁶ but it may be possible to determine if multiple bruises could have been inflicted at the same time.²⁷ Multiple bruises of the same age are suspicious if they are on different planes of the body (*e.g.*, front and back).²⁸

The shapes of bruises are occasionally significant in diagnosing abuse. "Bruises that take the shape of a recognizable object are not usually accidental. The most common of these is the 'loop' mark that is caused by a flexible object, such as a belt, electric cord, or clothesline."²⁹ Moreover, the appearance of a bruise may be inconsistent with the history, suggesting abuse. An example would be a black eye with bruising extending around the orbit onto the cheek and the forehead, and a history of running into the edge of a door. The edge of a door is a single linear plane. A bruise with this distribution requires impact to have occurred on several facial planes.

Besides unintentional trauma, other possible causes of bruising or apparent bruising that are not due to abuse must be ruled out. Many skin conditions may be misdiagnosed as due to abuse, including birth marks and various types of contact dermatitis. Mongolian spots are often confused with bruises. They are nontender, flat areas of grey-blue discoloration due to hyperpigmentation of the skin, and are quite common in darker complexioned infants, particularly over their buttocks and lower back.³⁰ They are present from birth.

If children present skin lesions that are uniformly deep brown in color and/or occur with blistering, then phytophotodermatitis must be considered. Phytophotodermatitis occurs when the skin comes in contact with plants containing psoralen (most notably lemons and limes) and is then exposed to sunlight.³¹ In six of the twelve cases reported in the literature, children with phytophotodermatitis suffered

However, as described in note 24, *supra*, these sources have been criticized as being based on clinical judgment rather than controlled research.

26. POLSON & GEE, *supra* note 24, at 109.

No precise estimate of the age of a bruise is practicable because the appearance of the colour changes which occur in an aging bruise will depend upon one or more of several factors. The size and depth of the bruise are especially important; a large bruise or one which is deep-seated may remain red or dark red for days or weeks.

Id.; Shaw, *supra* note 22, at 184 (appearance of bruise depends upon the force of the blow, age and physical condition of the child, site of injury, length of bleeding, depth of bleeding, age of lesion, rate of breakdown, and absorption of blood pigments"); Spitz, *supra* note 24, at 128 (Dating "depends on numerous factors, including among others the extent of the bruise, its depth and the efficiency of the local circulation.").

27. Shaw, *supra* note 22, at 184 (discussing the difficulty in dating bruises, but nevertheless concluding that physicians can "make a reasonable guess as to whether bruises are the result of one or more traumatic episodes"). *But see* Stephenson & Bialas, *supra* note 24, at 54 ("This study shows that several different colours can be present at the same time within one bruise and that bruises can change colour at very different rates, presumably depending on the nature of the injury and the child's response to this injury.").

28. See Schmitt, *supra* note 12, at 186.

29. Ellerstein, *supra* note 18, at 906; see Kornberg, *supra* note 7, at 94; Shaw, *supra* note 22, at 180.

30. Kornberg, *supra* note 7, at 100.

31. Kathryn Coffman et al., *Phytophotodermatitis Simulating Child Abuse*, 139 AM. J. DISEASES CHILDREN 239, 239; see Maki Okamura Goskowicz et al., *Endemic "Lime" Disease: Phytophotodermatitis in San Diego County*, 93 PEDIATRICS 828, 828 (1994).

from discoloration with the configuration of fingers or hands, leading physicians to initially suspect that abuse had occurred.³²

Bleeding disorders should always be considered in children with bruising. If a child exhibits multiple or extensive areas of serious bruising, or bruises easily, this increases the likelihood of a bleeding disorder.³³

IV. FRACTURES

Skeletal injury is a common manifestation of child abuse. As with abusive bruises, the incidence of nonaccidental fractures is highest in infants and young children. Children under eighteen months of age suffer 80% of the fractures attributable to child abuse.³⁴ In all cases of suspected child abuse among children under two years of age, a skeletal survey should be performed. A skeletal survey is a series of radiographs (x-rays) encompassing the entire skeleton.³⁵

A. Glossary of Terms

For skull fracture definitions see subpart IV.E., below.

1. Key Bones

Clavicle: The collar bone, extending from the shoulder to the breast bone.

Femur: The bone of the upper leg (above the knee).

Fibula: One of the two bones of the lower leg (below the knee).

32. Coffman et al., *supra* note 31, at 239 (two cases, both with hand print configurations); Goskowitz et al., *supra* note 31, at 829 (hand prints or finger marks were observed in six of 10 cases).

33. Shaw, *supra* note 22, at 184 ("Leukemia, idiopathic thrombocytopenia purpura, and hemophilia may be presented with cutaneous and other bleeding manifestations, giving rise to unjustified accusations."). But see A.E. O'Hare & O.B. Eden, *Bleeding Disorders and Non-Accidental Injury*, 59 ARCHIVES DISEASE CHILDHOOD 860 (1984) (found abnormal results on bleeding tests in 8/50 or 16% of children initially suspected of being abused, but nevertheless found evidence that seven of the eight had in fact been abused). The authors suggest that abnormal test results (particularly extended partial thromboplastin time) may be attributed to difficulty in obtaining a blood sample (in which case clotting can occur in the syringe, leading to a reduction in the amount of detectable platelets and coagulation factors), to the effects of minor viral infections on anticoagulants in the blood, and possibly to effects of the abuse itself. *Id.* at 863.

34. Peter Worlock et al., *Patterns of Fracture in Accidental and Non-Accidental Injury in Children: A Comparative Study*, 293 BRIT. MED. J. 100, 102 (1986). In contrast, children in this age group suffer only 2% of all unintentional fractures among children up to 18 years of age. *Id.*

35. Although this Article emphasizes the use of x-rays in imaging skeletal fractures, a number of imaging methods are useful, including skeletal scintigraphy (bone scan), ultrasonography, computed tomography (CT), and magnetic resonance imaging (MRI). A complete discussion of these methods is beyond the scope of this Article. See generally David F. Merten et al., *Skeletal Manifestations of Child Abuse*, in CHILD ABUSE: MEDICAL DIAGNOSIS AND MANAGEMENT, *supra* note 1, at 23, 24-25 [hereinafter Merten et al., *Skeletal Manifestations*].

Humerus: The bone of the upper arm (above the elbow).

Long bones: The tubular bones found in the arms and legs. Includes the humerus, radius, and ulna in the arms, and the femur, fibula, and tibia in the legs.

Radius: The shorter of two bones of the lower arm, on the thumb side of the arm at the wrist.

Scapula: The shoulder blade.

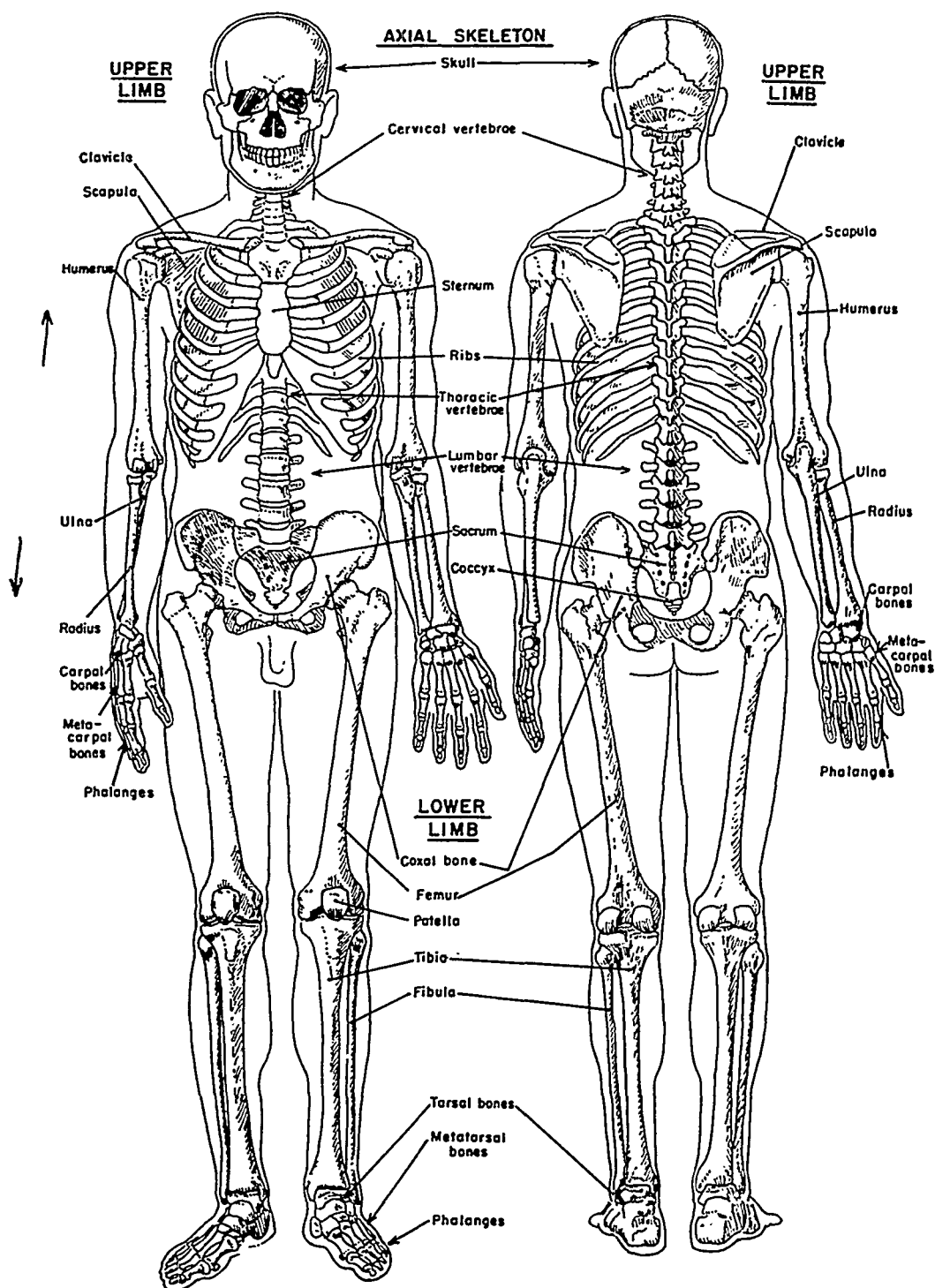
Sternum: The breast bone.

Tibia: One of the bones of the lower leg (below the knee).

Ulna: The “funny bone.” It is the longer of two bones of the lower arm, located on the opposite side of the thumb at the wrist.

Vertebrae: The bones of the spinal column.

Figure 1: The Skeleton



2. Bone Regions and Healing

Callus: Woven bone which forms where a bone is healing; ultimately absorbed into the hard new bone via a process called remodeling.

Distal: The part of the bone (or other structure) farthest from the center of the body (for example, the part of the femur near the knee is the “distal femur”).

Diaphysis (die-AFF-uh-sis): The shaft of a long bone between the metaphyseal ends. When used as an adjective, it is “diaphyseal.”

Epiphysis (eh-PIFF-uh-sis): The end of a long bone. When used as an adjective, it is “epiphyseal.”

Metaphysis (meh-TAFF-uh-sis): The flaring ends of the central shaft of a long bone. When used as an adjective, it is “metaphyseal.”

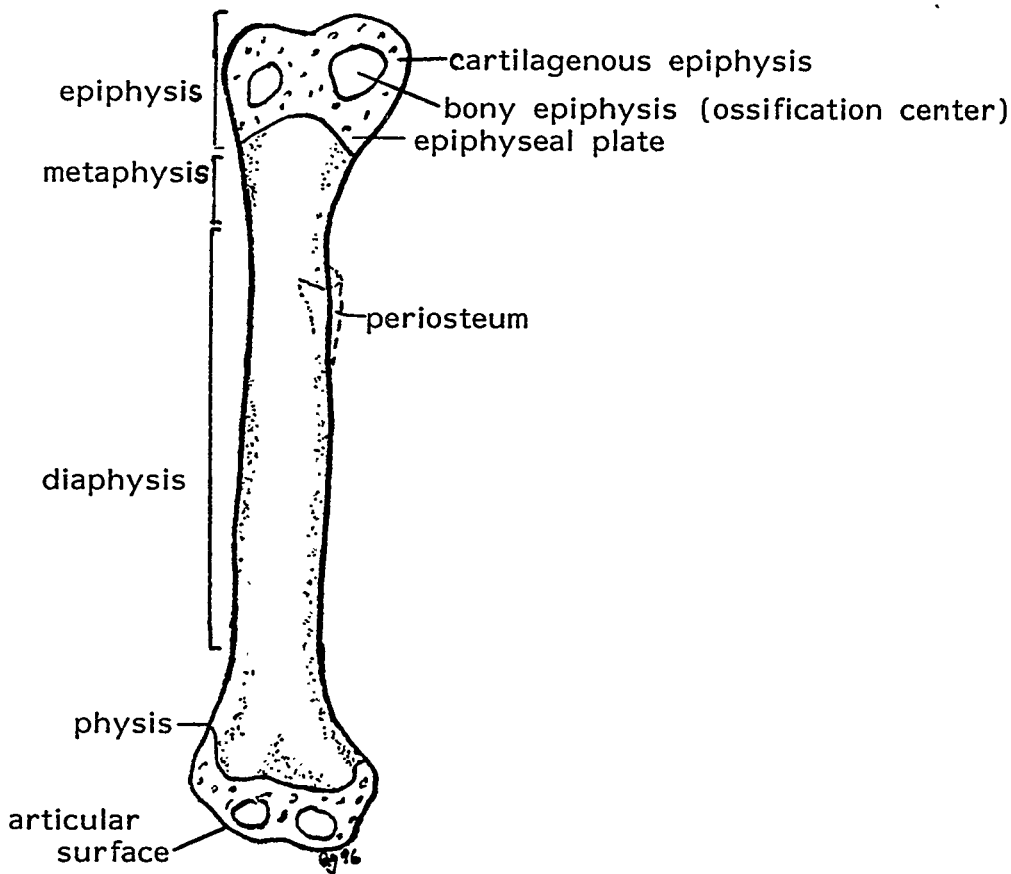
Periosteum (pair-ee-OST-ee-um): Specialized connective tissue that covers the bones. In the long bones, it is attached at both ends at the epiphysis. When used as an adjective, it is “periosteal.”

Physis (FIE-sis): Segment of bone between the epiphysis and diaphysis, important to the growth of the length of the bone. Also called the epiphyseal plate or growth plate.

Proximal: The part of the bone (or other structure) closest to the center of the body (for example, the part of the femur near the pelvis is the “proximal femur”).

Subperiosteal space: Potential space between the periosteum and the bone. “Potential space” means that under normal circumstances the space is nonexistent, but that it may fill with fluid, *e.g.*, blood.

Figure 2: Bone Anatomy



3. Fracture Types

Bowing: Bone deformed into a bowed shape. Attributed to forces exceeding the bone's elastic recoil (capacity to return to normal shape after bending). It is more common the younger the child, given the greater elasticity of immature bone.

Bucket-handle: A type of metaphyseal fracture. From a different angle, appears on x-ray as a corner fracture.

Corner: A type of metaphyseal fracture. From a different angle, appears on x-ray as a bucket-handle fracture.

Comminuted: Fracture in which bone is broken into multiple, variably sized pieces.

Displaced: Fracture in which the alignment of the bone is altered.

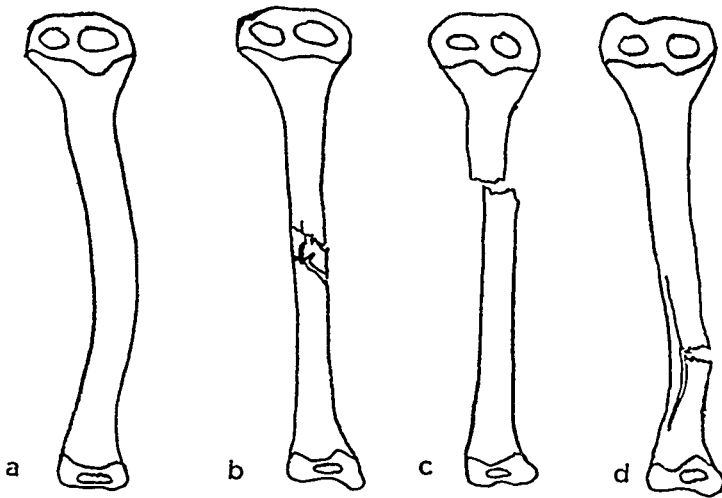
Greenstick: Fracture along the diaphysis (shaft) caused by bending past the point of bowing. A portion of the cortex and periosteum of the bone remain intact. Imagine the way in which an immature and elastic tree twig splinters when one attempts to break it. Common fracture type in children, given the greater elasticity of immature bone.

Oblique: Fracture in which the line of the fracture is both slanted (*i.e.*, not at a right angle) and varies when compared to the longitudinal axis (an imaginary line that runs through the diaphysis) of the bone. Often used interchangeably with "spiral" fracture.

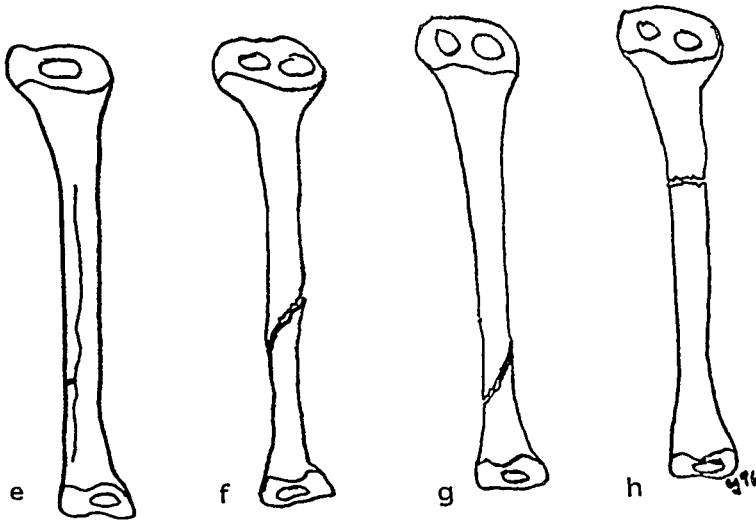
Spiral: Fracture in which the line of the fracture is oblique and encircles a portion of the diaphysis (shaft) of the bone, attributable to twisting and wrenching. Often used interchangeably with "oblique" fracture.

Transverse: Fracture in which the line of the fracture is at a right angle to the longitudinal axis (an imaginary line that runs through the diaphysis) of the bone.

Figure 3: Fracture Types



- a. bowing fracture
- b. comminuted fracture
- c. displaced fracture
- d. greenstick fracture



- e. longitudinal fracture
- f. oblique fracture
- g. spiral fracture
- h. transverse fracture

B. Nature of the Fracture and Abuse

1. Metaphyseal vs. Other Fracture Locations

Metaphyseal fractures constitute highly suggestive evidence of abuse in the infant or young child when they occur in the area of bone between the metaphysis and the epiphysis.³⁶ Metaphyseal fractures may be unintentional if they are located at the junction of the diaphysis and the metaphysis, known as the "metadiaphyseal" area.³⁷

Abusive metaphyseal fractures are often described as "bucket-handle" fractures or "corner" fractures.³⁸ Such fractures are generally observed in infants and children under two years of age, whose metaphysis is more fragile than that of older children.³⁹ Metaphyseal fractures can be caused by shaking as well as by pulling and twisting of the child's extremities.⁴⁰

Epiphyseal fractures or epiphyseal separation are supportive evidence of abuse.⁴¹ The history and other factors will be essential in determining if abuse has occurred.

Diaphyseal fractures are by themselves nonsupportive of abuse. "A variety of patterns of diaphyseal injury occur in infants. None possesses a specificity

36. H. Carty, *Brittle or Battered*, 63 ARCHIVES DISEASE CHILDHOOD 350, 350 (1988) [hereinafter Carty, *Brittle or Battered*]; Paul K. Kleinman, *Diagnostic Imaging in Infant Abuse*, 155 AJR 703 (1990) [hereinafter Kleinman, *Diagnostic Imaging in Infant Abuse*]; Paul K. Kleinman *Skeletal Trauma: General Considerations*, in DIAGNOSTIC IMAGING OF CHILD ABUSE 5, 10 (Timothy H. Grayson ed., 1987) [hereinafter Kleinman, *Skeletal Trauma*]; see C.J. Hobbs, *Fractures*, 298 BMJ 1015 (1989) [hereinafter Hobbs, *Fractures*].

37. Carty, *Fractures Caused by Child Abuse*, *supra* note 4, at 851.

38. Kleinman, *Skeletal Trauma*, *supra* note 36, at 15-17. The same fracture may appear as a bucket-handle fracture or as a corner fracture, depending upon the angle from which the fracture is x-rayed. If the bone is viewed tangentially (so that the bone is viewed at a right angle to the beam of the x-ray), the fracture is more likely to appear as a corner fracture, whereas if the bone is viewed obliquely (so that the bone is tilted toward or away from the beam of the x-ray), the fracture is more likely to be described as a bucket-handle fracture.

39. Interview with Ralph S. Lachman, M.D., Chief, Pediatric Radiology, Harbor-UCLA, in Los Angeles, Cal. (Oct. 27, 1994) [hereinafter Interview with Ralph S. Lachman, M.D.] (notes on file with authors).

40. 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, 396-97 (1974).

41. There is disagreement in the medical literature regarding the diagnosticity of epiphyseal injury. Compare Hobbs, *Fractures*, *supra* note 36, at 1016 (epiphyseal fractures high specificity for abuse); David F. Merten et al., *The Abused Child: A Radiological Reappraisal*, 146 RADIOLOGY 377, 380 (1983) [hereinafter Merten et al., *The Abused Child*] ("[M]any, if not most," of fractures involving metaphysis also involve epiphysis and are therefore Salter-Harris type fractures.); and Leonard E. Swischuk, *Radiographic Signs of Skeletal Trauma*, in CHILD ABUSE: A MEDICAL REFERENCE, *supra* note 7, at 151, 157 (separation of the epiphysis from the metaphysis "is the most pathognomonic of the battered child syndrome") with Kleinman, *Skeletal Trauma*, *supra* note 36, at 22 ("The vast majority of these [epiphyseal] fractures occur in patients beyond infancy and are accidental in nature. True Salter-Harris type fractures [a classification scheme for epiphyseal fractures] are unusual with child abuse.") and Kleinman, *Diagnostic Imaging in Infant Abuse*, *supra* note 36, at 708 tbl.1. (epiphyseal separation only moderately specific for abuse).

comparable to metaphyseal lesions for abuse, and factors such as the patient's age and an absent or inconsistent history provide clues to the diagnosis."⁴²

Subperiosteal hemorrhages occur with some fractures, and may be the product of abusive twisting and pulling. Repetitive injury may lead to multiple layers of subperiosteal hemorrhages and callus as new bone forms, as part of remodeling. However, periosteal new bone formation may also simply reflect normal bone growth. New bone formation and hemorrhages by themselves do not constitute evidence of abuse.⁴³

2. *Spiral vs. Other Types of Fracture*

Spiral fractures occur when torque (twisting force) is applied to the bone. They may occur unintentionally, as when a young child plants a foot, or gets it caught, and then falls. They may also occur following abuse in which the perpetrator violently twists a child's extremity. Long bone spiral fractures constitute highly suggestive evidence of abuse if a child is not yet walking well and there is no history of a fall involving a twisting force.⁴⁴ Furthermore, "there is no evidence that young infants can generate sufficient muscular effort to cause a self-induced spiral fracture."⁴⁵

Transverse fractures are a common fracture type among abused children, but are not more common among abused children than among nonabused children.⁴⁶ Transverse fractures may be the result of a direct blow.⁴⁷ It is important to consider the history and other factors in diagnosing an abusive injury; for example, a three-month-old child is not strong enough to give him or herself a transverse fracture.⁴⁸

C. *Specific Bones and Abuse*

1. *Rib Fractures*

Rib fractures are highly suggestive evidence of abuse in children under three years of age, particularly when located at the sides ("lateral") and back ("posterior")

42. Kleinman, *Skeletal Trauma*, *supra* note 36, at 22. The fact that a fracture involves the diaphysis does not of course mean that the fracture was accidental. In a review of 155 fractures of the extremities in abused children, a study found that 80% of the fractures were diaphyseal. Merten et al., *The Abused Child*, *supra* note 41.

43. Kleinman, *Skeletal Trauma*, *supra* note 36, at 6-8.

44. Swischuk, *supra* note 41, at 153.

45. Betty S. Spivack, *Biomechanics of Nonaccidental Trauma*, in CHILD ABUSE: A MEDICAL REFERENCE, *supra* note 7, at 61, 70.

46. John King et al., *Analysis of 429 Fractures in 189 Battered Children*, 8 J. PEDIATRIC ORTHOPAEDICS 585 (1988); Randall T. Loder & Christopher Bookout, *Fracture Patterns in Battered Children*, 5 J. ORTHOPAEDIC TRAUMA 428 (1991); Swischuk, *supra* note 41, at 153.

47. Swischuk, *supra* note 41, at 153.

48. *Id.*

of the rib next to the vertebral column,⁴⁹ and, more rarely, when they involve the rib ends in the front ("anterior costochondral").⁵⁰ Rib fractures in abused children are usually attributable to the squeezing of the chest.⁵¹ They can also be caused by direct blows.⁵²

Rib fractures are often difficult to visualize on x-rays when they first occur, and typically are not accompanied by external trauma,⁵³ although the child may cry when picked up. Signs of healing appear on x-rays within one to two weeks following the injury.⁵⁴

Minor trauma is unlikely to result in rib fractures, because of the compliance (flexibility) of the normal child's rib cage.⁵⁵ Falls (either free falls or falls down stairs) are not a reasonable alternative explanation for rib fractures.⁵⁶ Cardio-pulmonary resuscitation (CPR) does not lead to rib fractures.⁵⁷

2. Scapula, Sternum, and Vertebrae

Fractures of the scapula (shoulder blade) are highly suggestive evidence of abuse, especially when they involve the top of the scapula, known as the "acromion

49. Carty, *Fractures Caused by Child Abuse*, *supra* note 4, at 853 ("virtually diagnostic"); Hobbs, *Fractures*, *supra* note 36; Kleinman, *Diagnostic Imaging in Infant Abuse*, *supra* note 36, at 700; Paul K. Kleinman, *Bony Thoracic Trauma*, in *DIAGNOSTIC IMAGING OF CHILD ABUSE*, *supra* note 36, at 67. [hereinafter Kleinman, *Bony Thoracic Trauma*]; Leventhal et al., *supra* note 4, at 89 tbl.3 (12 of 12 rib fractures attributed to abuse); Swischuk, *supra* note 41, at 160 ("Infant rib fractures are very specific for child abuse."); Worlock et al., *supra* note 34, at 102 (rib fractures common in abused children; whereas only one of 826 nonabused children suffering from fractures had fractured ribs, and that was attributable to an auto accident).

50. Kleinman, *Bony Thoracic Trauma*, *supra* note 49, at 74; Marvin S. Kogutt et al., *Patterns of Injury and Significance of Uncommon Fractures in the Battered Child Syndrome*, 121 AM. J. ROENTGENOLOGY 143, 148 (1974); see Swischuk, *supra* note 41, at 160 (pathognomonic of abuse).

51. Kleinman, *Diagnostic Imaging in Infant Abuse*, *supra* note 36, at 706; Interview with Ralph S. Lachman, M.D., *supra* note 39.

52. Swischuk, *supra* note 41, at 153.

53. Carty, *Fractures Caused by Child Abuse*, *supra* note 4, at 854.

54. Interview of Ralph S. Lachman, M.D., *supra* note 39.

55. Merten et al., *Skeletal Manifestations*, *supra* note 34, at 39.

56. See Mark Joffe & Stephen Ludwig, *Stairway Injuries in Children*, 82 PEDIATRICS 457, 459 (1988) (no rib fractures in 363 falls down stairs); Melvin D. Smith et al., *Injuries in Children Sustained in Free Falls: An Analysis of 66 Cases*, 15 J. TRAUMA 987, 991 (1975) [hereinafter Smith et al., *Injuries in Children Sustained in Free Falls*] (no rib fractures in 66 free falls, including 39 falls 10 feet or greater; authors note that falls often do cause rib fractures in adults, and suggest that young children's ribs are more flexible). But see Victor F. Garcia et al., *Rib Fractures in Children: A Marker of Severe Trauma*, 30 J. TRAUMA 695, 696 tbl.1, 698 (1990) (three of 33 rib fractures in children attributed to falls; authors note, however, that "[r]ib fractures in an infant or toddler were usually a result of intentional injury" and the majority of fractures were attributed to auto accidents).

57. See Carty, *Brittle or Battered*, *supra* note 36, at 350 (the author has never encountered a rib fracture caused by cardiac massage); Kenneth W. Feldman & David K. Brewer, *Child Abuse, Cardiopulmonary Resuscitation, and Rib Fractures*, 73 PEDIATRICS 339 (1984) (in review of 41 abused children, 50 children administered CPR, and 22 children incidentally discovered to have rib fractures; no rib fracture attributable to CPR); Melissa R. Spevak et al., *Does Cardiopulmonary Resuscitation Cause Rib Fractures in Infants? Post-Mortem Radiologic-Pathologic Study*, 177 RADIOLOGY 162 (1990) (autopsies of 91 nonabused infants under one year of age who received CPR; no rib fractures).

process.”⁵⁸ Scapular fractures are attributable to shaking, or excessive twisting or pulling of the arms.⁵⁹ Fractures of the sternum are highly suggestive evidence of abuse.⁶⁰ They are attributable to direct blows to the chest or compression of the chest.⁶¹ Because of the great force required to cause fractures or dislocations (“subluxations”) of the vertebrae, such injuries are highly suggestive evidence of abuse, at least when there is not a clear history that adequately explains their occurrence.⁶²

3. Humerus

Fractures of the humerus in children under three are highly suggestive evidence of abuse, unless they are diaphyseal fractures just above the elbow (“supracondylar fractures”), in which case they are usually unintentional.⁶³ Humeral fractures are caused by a number of different types of abusive behavior, including lifting the child roughly from a lying position by the arm, swinging the child through the air by the arm, shaking the child while grabbing the upper arms, and/or shaking the child while grasping the child’s chest, thus causing the arms to flail about.⁶⁴

58. Carty, *Brittle or Battered*, *supra* note 36, at 350; Hobbs, *Fractures*, *supra* note 36; Kleinman, *Bony Thoracic Trauma*, *supra* note 49, at 83; Kogutt et al., *supra* note 50, at 147; *see* Kleinman, *Diagnostic Imaging in Infant Abuse*, *supra* note 36, at 706 (specific for abuse when fracture involves the top of the scapula, called the “acromion process”); Swischuk, *supra* note 41, at 160 (as diagnostic as metaphyseal fractures when involving “fragmentation of the acromial process of the scapula”).

59. *See* Kleinman, *Bony Thoracic Trauma*, *supra* note 49, at 86 (abusive scapular fractures due to “abnormal rotational or tractional forces applied to the shoulder” via the upper arm).

60. Hobbs, *Fractures*, *supra* note 36, at 1016; Kleinman, *Diagnostic Imaging in Infant Abuse*, *supra* note 36, at 706; Kogutt et al., *supra* note 50, at 146; Swischuk, *supra* note 41, at 153.

61. Kleinman, *Bony Thoracic Trauma*, *supra* note 49, at 80, 82.

62. Carty, *Brittle or Battered*, *supra* note 36, at 350; *see* Kleinman, *Diagnostic Imaging in Infant Abuse*, *supra* note 36, at 708 tbl.1 (“moderate specificity” which becomes high when explanation inadequate).

63. Carty, *Fractures Caused by Child Abuse*, *supra* note 4, at 850; *see* Leventhal et al., *supra* note 4 (13 of 16 humerus fractures in children under three due to abuse; three of three due to accident involved the elbow); Rosenberg & Bottenfield, *supra* note 5 (in fractures of infants under one year of age, oblique humerus fractures exclusively in abuse group, although half of humerus transverse fractures were nonabusive); Susan A. Thomas et al., *Long-Bone Fractures in Young Children: Distinguishing Accidental Injuries From Child Abuse*, 88 PEDIATRICS 471 (1991) (11 of 14 humerus fractures in children under three were due to abuse, three of three due to accident involved the elbow); Worlock et al., *supra* note 34 (in children under five, 13 or 14 abusive fractures were of shaft or metaphysis, whereas 13 of 15 nonabusive fractures were supracondylar).

64. Paul K. Kleinman, *Extremity Trauma*, in *DIAGNOSTIC IMAGING OF CHILD ABUSE*, *supra* note 36, at 46 [hereinafter Kleinman, *Extremity Trauma*].

4. Femur

If the child is under one year of age, a fractured femur is "more often than not attributable to abuse."⁶⁵ Spiral (or oblique) fractures of the femur are especially suspicious in infants who are not yet walking (preambulatory).⁶⁶ A twisting force is necessary to cause a spiral fracture. Children who are walking well and who have learned to run can stumble and fracture their femur, including a spiral fracture.⁶⁷

Femur fractures do not appear to result from falls down stairs,⁶⁸ but a greenstick fracture of the femur can occur if a person falls while holding the child on his or her hip.⁶⁹ An infant can suffer from a spiral femur fracture from a free (vertical) fall, but such would require "a severe torsional [twisting] force developed during a major fall."⁷⁰

65. Carty, *Fractures Caused by Child Abuse*, *supra* note 4, at 850; see Kleinman, *Extremity Trauma*, *supra* note 64, at 29 ("Femoral fractures in infants have a strong association with abuse, whereas similar fractures in older children are usually determined to be accidental."); Humberto Galleno & William L. Oppenheim, *The Battered Child Syndrome Revisited*, 162 CLINICAL ORTHOPAEDICS 11, 17 (1982) ("Lower extremity fractures in nonweight-bearing children are highly suggestive of the battered child syndrome."). Age differences in the association between femur fracture and abuse have been reported in a number of studies. See William A. Anderson, *The Significance of Femoral Fractures in Children*, 11 ANNALS EMERGENCY MED. 174 (1982) (20% of femur fractures in children up to 15 due to abuse; 79% of such fractures abusive in children under two); R. H. Gross & M. Stranger, *Causative Factors Responsible for Femoral Fractures in Infants and Young Children*, 3 J. PEDIATRIC ORTHOPAEDICS 341 (1983) (46% of femur fractures in children under six suspicious of abuse, 65% of such fractures suspicious of abuse in children under one); Leventhal et al., *supra* note 4 (0% of femur fractures in children two or older due to abuse; 60% of such fractures abusive in children under one); Thomas et al., *supra* note 63 (36% of femoral fractures in children under three due to abuse; 60% of such fractures abusive in children under one).

66. Kleinman, *Extremity Trauma*, *supra* note 64, at 37. Although most researchers have *not* found a relationship between the type of femur fracture (e.g., spiral, transverse) and the likelihood that the fracture was due to abuse, see Anderson, *supra* note 65; Rodney K. Beals & Emily Tufts, *Fractured Femur in Infancy: The Role of Child Abuse*, 3 J. PEDIATRIC ORTHOPAEDICS 583 (1983); Heidi J. Dalton et al., *Undiagnosed Abuse in Children Younger than Three Years with Femoral Fracture*, 144 AM. J. DISEASES CHILDREN 875 (1990), other researchers have found that spiral fractures among children younger than 15 months were suggestive of abuse, Dalton et al., *supra*, at 877; Thomas et al., *supra* note 63.

67. See Beals & Tufts, *supra* note 66, at 585 (Nine of 10 cases in which children under four have a femur fracture with a history of falling while walking or running were classified as nonabusive.); Carty, *Fractures Caused by Child Abuse*, *supra* note 4, at 850 (Children learning to walk can fracture a femur.); Leventhal et al., *supra* note 4, at 91 ("We believe that the association between abuse and fractures of [the femur] in toddlers has probably been overemphasized."); Thomas et al., *supra* note 63, at 476 ("Children who are old enough to run can trip or stumble and fracture a femur."); Interview with Ralph S. Lachman, M.D., *supra* note 39 (Children must be walking well, and not just learning, for a running fall to be a credible explanation of femur fracture.).

68. Joffe & Ludwig, *supra* note 56, at 459 (no femur fractures in 363 falls down stairs).

69. K. E. Daly & P. T. Calvert, *Accidental Femoral Fracture in Infants*, 22 INJURY 337 (1991).

70. Kleinman, *Extremity Trauma*, *supra* note 64, at 37.

5. Other Bones

A fracture of the distal (outer) end of the clavicle is highly suggestive evidence of abuse.⁷¹ Most clavicle fractures, however, are not attributable to abuse.⁷² If the child is preambulatory, finger fractures are highly suggestive evidence of abuse.⁷³ Fractures of the ulna and tibia are ordinarily the product of abuse if the child is under one year of age.⁷⁴ Children learning to walk often suffer from what is known as a "toddler's fracture," an accidental fracture of the diaphysis of the tibia.⁷⁵

D. Multiple Injuries and Abuse

Multiple fractures of different ages are highly suggestive evidence of abuse.⁷⁶ Bilateral fractures (two fractures of opposing extremities, such as fractures of the right and left femur) are also highly suggestive evidence of abuse.⁷⁷ Nevertheless, in most cases of identified abuse, the child presents only one fracture.⁷⁸

Moreover, fractures coexisting with soft tissue injury of the head and neck, including brain injuries, are highly suggestive evidence of abuse.⁷⁹ Since abused

71. Carty, *Brittle or Battered*, *supra* note 36, at 350; Kogutt et al., *supra* note 50, at 145; see Swischuk, *supra* note 41, at 157-60 (noting that "fragmentation fractures of the distal end of the clavicle" are as diagnostic as metaphyseal fractures, and are attributable to twisting or jiggling).

72. See Carty, *Fractures Caused by Child Abuse*, *supra* note 4, at 849 tbl.2 ("low specificity" when mid-clavicle fractured); Kleinman, *Bony Thoracic Trauma*, *supra* note 49, at 78 (midshaft fractures of the clavicle "commonly occur indirectly as a result of landing on an outstretched arm"); Kleinman, *Diagnostic Imaging in Infant Abuse*, *supra* note 36, at 6 (clavicle fractures "low specificity" for abuse); Leventhal et al., *supra* note 4, at 89 tbl.3 (25 of 25 clavicle fractures in children under three classified as nonabusive); Swischuk, *supra* note 41, at 153 (although midshaft fracture of the clavicle is common consequence of abuse, it is also common accidental fracture); Rosenberg & Bottenfield, *supra* note 5, at 179 (four of four clavicle fractures in children under one year of age classified as nonabusive).

73. Carty, *Fractures Caused by Child Abuse*, *supra* note 4, at 849 tbl.1.

74. Leventhal et al., *supra* note 4, at 89 (fractures of the ulna and tibia usually nonabusive, unless child is less than one year of age, in which case 82% due to abuse).

75. Kleinman, *Extremity Trauma*, *supra* note 64, at 41.

76. Carty, *Fractures Caused by Child Abuse*, *supra* note 4, at 849 tbl.1 ("high specificity"); Hobbs, *Fractures*, *supra* note 36, at 1016 ("high specificity"); Kleinman, *Diagnostic Imaging in Infant Abuse*, *supra* note 36, at 6 ("moderate specificity" but high if history missing or inconsistent).

77. Carty, *Brittle or Battered*, *supra* note 36, at 849 tbl.1; Galleno & Oppenheim, *supra* note 65, at 15; see Beals & Tufts, *supra* note 66, at 585 (three of three bilateral femur fractures were due to abuse).

78. See Anderson, *supra* note 65 (67% of abused children had only one fracture); King et al., *supra* note 46, at 589 (72% of abused children had one or two fractures); Loder & Bookout, *supra* note 46, at 429 (65% of abused children had only one fracture; only 13% had multiple fractures at various stages of healing); Charles Q. McClelland et al., *Fractures in the First Year of Life: A Diagnostic Dilemma?*, 136 AM. J. DISEASES CHILDREN 26, 27 (1982) (abused children no more likely to have multiple fractures than nonabused children); Rivara, *supra* note 5, at 95 (67% of abused children had only one fracture). But see Worlock et al., *supra* note 34, at 101 (26% of abused children had only one fracture).

79. Worlock et al., *supra* note 34, at 102 ("strongly suggestive"). The exception to this rule is, of course, when the accident that led to the fracture also plausibly caused the soft tissue injury, such as when bruising is evident around the site of a skull fracture, or when a child falls down the stairs and suffers from multiple injuries.

children are often presented without soft tissue injuries, however, their absence does not rule out an abusive etiology for other injuries.⁸⁰

Battered children frequently have multiple injuries, including patterned skin injuries such as bites, burns, and bruises; fractures; visceral injuries; and head injuries. Infants and children in whom recent, healing, or healed injuries of different sites are documented may be diagnosed as fulfilling criteria for the "battered child syndrome."⁸¹

E. Dating of Fractures

The amount of time that elapses between the circumstances leading to a fracture and the examination of the child by a physician is significant for several reasons. Dating of the fracture may help to determine whether the fracture could be attributable to birth trauma or which caretaker is responsible for injuring the child. If it is clear that the parents have delayed in bringing the child in for treatment, the delay itself is a factor suggesting that the injury was attributable to abuse.⁸² Claims that the fracture went unnoticed are implausible. The process of incurring a fracture is painful. Fractures are frequently associated with irritability, altered sleeping patterns, decreased feeds, pain with movement, and sometimes swelling of an extremity.

The following information is commonly used by physicians in dating injuries:⁸³

—The swelling from soft tissue injuries resolves (heals) in anywhere from two to ten days (most in four to ten days).

—Early periosteal new bone forms in four to twenty-one days (most likely ten to fourteen days). The younger the child, the more quickly new bone forms.

—Loss of fracture line in ten to twenty-one days (most likely fourteen to twenty-one days).⁸⁴

—Soft callus forms in ten to twenty-one days (peak fourteen to twenty-one days).

80. See Galleno & Oppenheim, *supra* note 65, at 11-12 (52% of abused children had bruises or other evidence of beatings); Loder & Bookout, *supra* note 46, at 430 (only 29% of abused children had associated soft tissue injury).

81. THE BATTERED CHILD, *supra* note 1, at 249-52; C. Henry Kempe et al., *The Battered-Child Syndrome*, 181 JAMA 105 (1962).

82. Carty, *Fractures Caused by Child Abuse*, *supra* note 4, at 854-57.

83. *Id.*; John F. O'Connor & Jonathon Cohen, *Dating Fractures*, in *DIAGNOSTIC IMAGING OF CHILD ABUSE*, *supra* note 36, at 112.

84. A fracture line is not always apparent, however. Swischuk, *supra* note 41, at 163-64.

—Hard callus forms in fourteen to ninety days (peak twenty-one to forty-two days).

—Remodeling of bone to normal shape in three months to two years.

Some types of fractures are difficult to date because of the absence of periosteal new bone formation. This includes many transverse fractures and metaphyseal fractures.⁸⁵ Skull fractures are virtually impossible to date.⁸⁶

F. Skull Fractures

In addition to having a terminology all their own, skull fractures present special problems in the diagnosis of child abuse, and therefore are treated separately here. As with all fractures, the history given by the caretakers is very important, as is the presence of other injuries that suggest abuse has occurred. Skull fractures are often associated with other injuries inside and/or outside the skull (*e.g.*, bleeding inside the membranes covering the brain and spinal cord, brain injury), and those injuries are described in Part V.

1. Glossary of Terms

a. Key Bones of the Skull

(See Figure 4)

Cranium: All of the bones of the head except the mandible and those forming the skeleton of the face.

Frontal bone: Forms the forehead.

Mandible: The jaw bone.

Occipital bone: Forms much of the back and base of the cranium. Encloses the large oval opening called the foramen magnum, connecting the cranial cavity (within which the brain sits) with the vertebral canal (where the spinal cord is situated).

85. Kleinman, *Diagnostic Imaging in Infant Abuse*, *supra* note 36, at 705.

86. Ralph S. Lachman, M.D., *Osteogenesis Imperfecta or Child Abuse?*, Address Before the Children's Services Division of Los Angeles County Counsel (Sept. 29, 1994) (videotape on file with the Children's Services Division of Los Angeles County Counsel) [hereinafter Address by Ralph S. Lachman, M.D.].

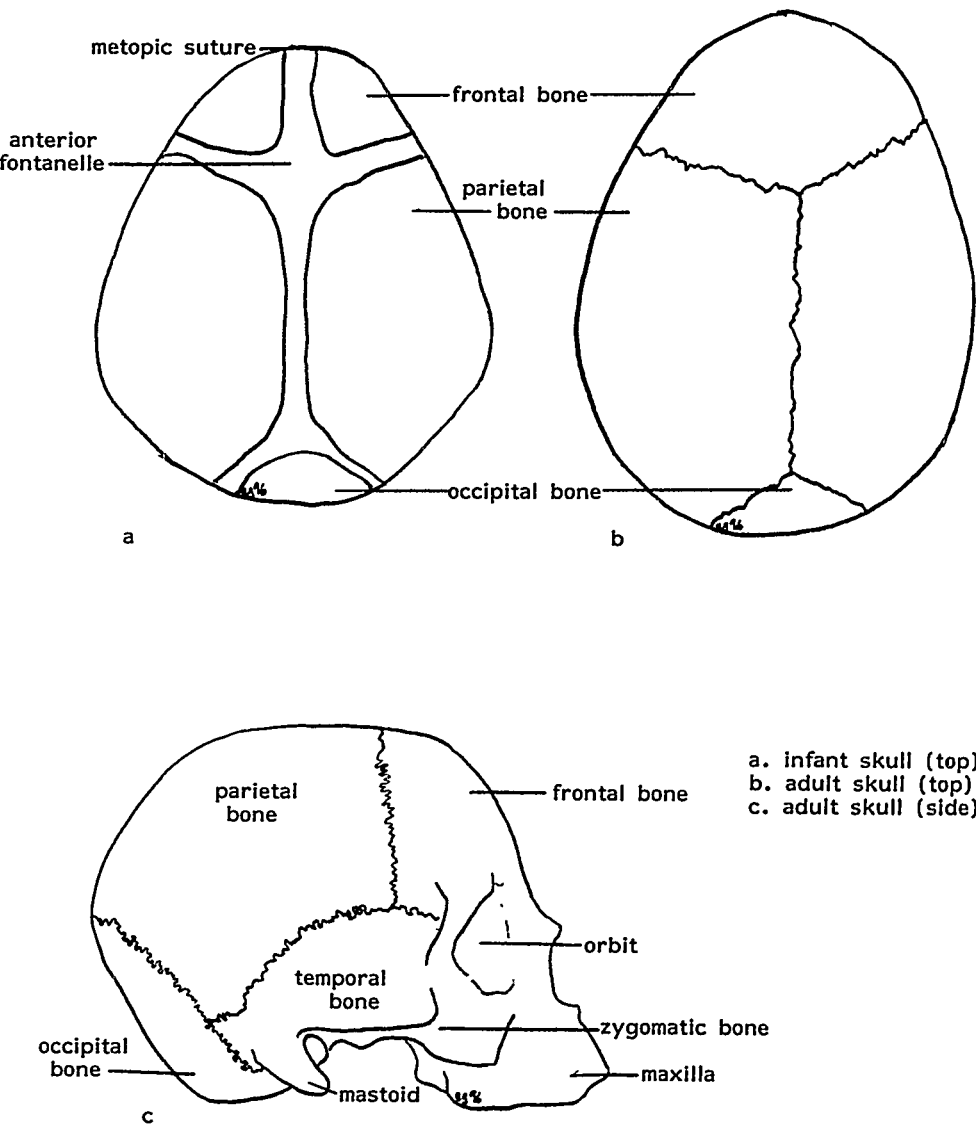
Other skull bones: ethmoid, hyoid, lacrimal, maxillae, nasal, palatine, sphenoid, zygomatic.

Parietal bones: Skull bones situated bilaterally (on opposite sides) which form the upper sides and roof of the cranium.

Suture lines: The junction between skull bones, where the bones of the skull meet.

Temporal bones: Skull bones situated bilaterally (on opposite sides) which form the lower sides and base of the skull.

Figure 4: Bones of the Skull



b. Specific Skull Fractures

Bilateral fracture: Fractures that are on both (right and left) sides of the skull.

Comminuted fracture: A fracture with multiple bone fragments.

Complicated fracture: A fracture that is not a simple linear fracture (*e.g.*, bilateral, diastatic, depressed).

Depressed fracture: A fracture that is sunken or indented inward from the normal surface of the skull. The majority of these fractures are comminuted.

Diastatic fracture: A fracture in which the edges of the fracture are split apart.

Linear fracture: A fracture that does not branch. Also known as a simple fracture.

Simple fracture: A linear fracture.

Stellate fracture: A fracture with numerous fracture lines radiating from a central point.

2. Skull Fractures and Abuse

The presence of a skull fracture confirms that trauma occurred to the cranial vault. Since skull fractures are frequently the result of unintentional falls, the history is particularly important in determining whether the trauma was abusive. One must consider the height and force of the fall, the surface onto which the child fell, the location and type of the fracture, and the degree and type(s) of brain injury. Also relevant is the child's age and gross motor development (*e.g.*, can the child crawl, stand, walk, run). The type of fracture depends in large part upon the amount of force directed at the skull and the site of impact. However, the mechanics of skull fracture are such that the site of the fracture does not always equate with the site of impact. Furthermore, since the skull of the young child is more compliant than that of an adult, a given impact may not result in any fracture.

The location of the fracture is not particularly helpful in diagnosing abuse. Both intentional and unintentional fractures tend to be parietal.⁸⁷ Although some experts feel that occipital fractures are suspicious of abuse, other researchers disagree.⁸⁸

Simple linear fractures are the most common skull fracture, regardless of etiology (accidental or nonaccidental). The determination that a linear fracture is attributable to abuse is made based on additional information, such as the presence of other injuries; the incidental identification of the fracture during a skeletal survey, with no history of injury; and other suspicious aspects of the injury.⁸⁹

Physicians are more likely to be suspicious if a young child presents with a complicated skull fracture (e.g., bilateral, multiple, or a fracture that crosses suture lines).⁹⁰ Bilateral or other multiple fractures indicate that impact occurred in more than one site. However, because they often occur accidentally, it is essential that the history offered by the caretakers be considered before determining whether the fracture is abuse.⁹¹

Falls of less than four feet onto flat surfaces sometimes result in skull fractures. However, the resulting skull fractures are almost always linear fractures.⁹² The only

87. Hobbs, *Fractures*, *supra* note 36, at 1017; Leventhal et al., *supra* note 4, at 90; Clifford J. Meservy et al., *Radiographic Characteristics of Skull Fractures, Resulting from Child Abuse*, 149 AJR 173, 174 (1987).

88. Compare Hobbs, *Fractures*, *supra* note 36 (occipital fractures most often abusive) with Leventhal et al., *supra* note 4, at 89-90 (occipital fractures not most often abusive, but parieto-occipital fractures exclusively abusive).

89. J.K. Brown & R.A. Minns, *Non-Accidental Head Injury, with Particular Reference to Whiplash Shaking Injury and Medico-Legal Aspects*, 35 DEV. MED. & CHILD NEUROLOGY 849, 853-54 (1993); Leventhal et al., *supra* note 4; O'Connor & Cohen, *supra* note 83, at 103. In Hobbs, *Fractures*, *supra* note 36, linear fractures were somewhat uncommon (20% of abusive fractures) in a series of 29 abusive skull fractures. But Hobbs's study has been criticized as limited to the more serious cases of abuse. See Leventhal et al., *supra* note 4, at 90; Meservy et al., *supra* note 87, at 174-75.

90. See Brown & Minns, *supra* note 89, at 853 (review of research); C.J. Hobbs, *Skull Fracture and the Diagnosis of Abuse*, 59 ARCHIVES DISEASE CHILDHOOD 246, 246-250 (1984) [hereinafter Hobbs, *Skull Fracture and the Diagnosis of Abuse*] (compared 29 abusive skull fractures to 60 accidental skull fractures in children under 2; 79% of abusive fractures were multiple complex compared to 5% of accidental skull fractures; 76% of abusive fractures involved more than one bone compared to 7% of accidental skull fractures); Meservy et al., *supra* note 87, at 175 (compared 39 abusive skull fractures to 95 accidental skull fractures in children under two; 31% of abusive fractures were multiple compared to 10% of accidental fractures; 18% of abusive fractures were bilateral compared to 3% of accidental skull fractures); Worlock et al., *supra* note 34, at 102 (compared 16 abusive skull fractures to 23 accidental fractures; abusive fractures more often multiple or complex than accidental fractures). A higher percentage of abused children than nonabused children had bilateral or multiple fractures in Leventhal et al., *supra* note 4, at 89-90 (bilateral 19% of abusive fractures versus 7% of accidental fractures, multiple 19% of abusive fractures versus 9% of accidental fractures), but the total numbers were small and the results were therefore not statistically significant.

91. Indeed, research by Leventhal et al., did not find that bilateral or multiple fractures were more common among abused children than among nonabused children, and as will be discussed below, found that relatively short falls sometimes resulted in such fractures. Leventhal et al., *supra* note 4, at 90-92.

92. See A.C. Duhaime et al., *Head Injury in Very Young Children: Mechanisms, Injury Types, and Ophthalmologic Findings in 100 Hospitalized Patients Younger Than 2 Years of Age*, 90 PEDIATRICS 179, 181 (1992) [hereinafter Duhaime et al., *Head Injury in Very Young Children*] (falls of less than four feet resulted in linear fractures, whereas basilar or depressed fractures were all due to falls of more than four feet or down stairs); Ray E. Helfer et al., *Injuries Resulting When Small Children Fall Out of Bed*, 60 PEDIATRICS 533, 534-35 (1977) (219 three-foot falls reported by parents of children under five resulted in two skull fractures; 85 three-foot falls

reported cases of complicated fractures in short falls occurred when young infants (under six months old) fell from two to four feet onto hard surfaces, such as pavement, cement, linoleum, and wood.⁹³ Even falls onto these surfaces do not ordinarily result in complicated fractures.⁹⁴ Typically, children do not sustain any significant brain injury with short falls. Falls down stairs often result in skull fractures,⁹⁵ and these fractures may be complicated.⁹⁶ Skull fractures are especially common if an infant falls while being carried down stairs.⁹⁷

The presence of a complicated fracture (multiple, comminuted, crosses suture lines, or diastatic without evidence of increased intracranial pressure), in conjunction with a history that the child fell from less than four feet onto a carpeted or padded surface, constitutes highly suggestive evidence of child abuse. If the caretaker claims that the child fell from less than four feet onto a hard surface, then a complicated fracture constitutes supportive evidence of abuse, since this fracture type does not ordinarily occur in unintentional injuries.

onto noncarpeted hospital floor resulted in one skull fracture; none of fractures were bilateral, diastatic, or more than one millimeter wide); Hobbs, *Skull Fracture and the Diagnosis of Abuse*, *supra* note 90 at 250 (60 accidental skull fractures in children under two years; only one case of skull fracture in fall less than three feet, and it was linear, narrow, and uncomplicated); Harvey Kravitz et al., *Accidental Falls from Elevated Surfaces in Infants from Birth to One Year of Age*, 44 PEDIATRICS 869 (1969) (336 20- to 60-inch falls in children under one reported by parents; three skull fractures; not described); Leventhal, *supra* note 4, at 91 (eight skull fractures from falls by children under three of less than two feet; all eight were simple linear fractures); Thomas J. Lyons & R. Kim Oates, *Falling out of Bed: A Relatively Benign Occurrence*, 92 PEDIATRICS 125 (1993) (208 25- to 54-inch falls in children under six onto vinyl floor in hospital resulted in one skull fracture (32-inch fall of 10-month-old), and it was a simple linear fracture with no neurological complications); Prasit Nimityongskul & Lewis D. Anderson, *The Likelihood of Injuries when Children Fall out of Bed*, 7 J. PEDIATRIC ORTHOPEDICS 184, 184-85 (1987) (761 one- to three-foot falls onto hard vinyl floor in the hospital in children under 16; one skull fracture, which was not depressed).

93. Leventhal et al., *supra* note 4, at 90 (six of 23 skull fractures in children under three from falls of two to four feet were complicated; five of six had histories of falling onto pavement, linoleum, or wood (the last unknown), and all six were under six months of age); *id.* at 90 ("Complicated fractures . . . also occurred when children fell from moderate heights (e.g., from a parent's arms or a changing table).").

94. Note that the falls reported in the hospital studies by Helfer et al., *supra* note 92, at 534, Lyon & Oates, *supra* note 92, and Nimityongskul & Anderson, *supra* note 92, at 185-86, were onto noncarpeted hospital floors, and never resulted in complicated fractures.

95. Leventhal et al., *supra* note 4, at 90 (18 of 26 accidental falls leading to fractures in children under three were from falls down stairs; most common fracture was of the skull); M.J. Rieder et al., *Patterns of Walker Use and Walker Injury*, 78 PEDIATRICS 488, 488-89 (1986) (139 injuries resulting from walkers; 19 skull fractures). Although Joffe & Ludwig, *supra* note 56, at 458, found that only 1.7% of the 363 children up to 18 years of age in their series who fell down stairs suffered a skull fracture, Christine T. Chiaviello et al., *Stairway-Related Injuries in Children*, 94 PEDIATRICS 679, 679-80 (1994), found a 7% rate of skull fracture among 69 children under five who fell down stairs, and suggested that the higher proportion of skull fractures might be attributable to the younger age of their subjects and to the greater frequency with which their hospital currently orders CT scans.

96. Leventhal et al., *supra* note 4, at 90-91 (35% of falls from four to seven feet that led to skull fractures were complicated; most of the four to seven-foot falls were down stairs).

97. See Chiaviello et al., *supra* note 95, at 680 (in a study of 69 children under five who fell down stairs, two of three infants fell while being held and suffered from a skull fracture); Joffe & Ludwig, *supra* note 56, at 458 (in study of 363 children who fell down stairs, four of 10 infants who fell while being held suffered from a skull fracture).

Some researchers believe that diastatic and depressed fractures are suspicious. These features are more common among abused children than among nonabused children, and therefore constitute supportive evidence of abuse.⁹⁸ However, special considerations apply to these fractures. The presence of a diastatic fracture does not necessarily imply greater force, but may also be the product of increasing intracranial pressure.

In order to incur a depressed fracture, the skull must either hit a protruding surface capable of causing an indentation in the skull, or be hit by an object traveling at high velocity. The fracture typically mimics the shape of the protruding surface or object. Such a history is necessary to rule out abuse even if the reported fall is up to ten feet.⁹⁹

G. Alternate Causes of Fractures

1. Falls

Caretakers will frequently explain that their child has a fracture because the child suffered a free (vertical) fall. Since falls are a leading cause of unintentional injury in infants and children,¹⁰⁰ it is essential to be able to discriminate between true and fabricated histories of falls. The history given by the caretaker must be carefully compared to the fracture sustained by the child to determine if the nature of the fracture is compatible with the mechanics of the fall. The likelihood that falls lead to specific types of fractures was discussed in the subparts specific to fracture types; this subpart will discuss the relation among falls, fractures, and serious injury more generally.

98. Although some researchers have argued that depressed fractures and diastatic fractures larger than three to five millimeters wide are highly suspicious, Brown & Minns, *supra* note 89, at 853; Hobbs, *Skull Fracture and the Diagnosis of Abuse*, *supra* note 90, other researchers have not found that a higher proportion of abused children suffer such fractures than accidentally injured children, *see* Leventhal et al., *supra* note 4 (comparing 26 abusive skull fractures to 57 accidental skull fractures in children under three); Meservy et al., *supra* note 87 (comparing 39 abusive skull fractures to 95 accidental skull fractures in children under two). If one sums across the three studies (Hobbs, Leventhal, and Meservy), one finds that the rate of depressed fractures and diastatic fractures is twice as high among abused children as among nonabused children, making these types of fractures nonspecific evidence of abuse.

99. *See* R.A. Williams, *Injuries in Infants and Small Children Resulting from Witnessed and Corroborated Free Falls*, 31 J. TRAUMA 1350 (1991) (50 witnessed falls of 10 feet or less among children up to three years of age; only two depressed skull fractures, and these resulted from falls of four to five feet onto edged surfaces). An exception occurs when infants experience short falls in which there is direct impact to the parietal tuberosity (an elevation of the parietal bone near its center), which may result in a simple, depressed fracture with no concomitant brain injury or other markers of injury. Such fractures are called "ping-pong fractures," since the effect is somewhat like the indentation one creates in a ping-pong ball by depressing the surface. In these cases, it is not necessary for the skull to impact on a protruding surface. This is a rare occurrence.

100. L.K. Garrettson & Susan S. Gallagher, *Falls in Children and Youth*, 32 PEDIATRIC CLINICS N. AM. 153, 154 (1985); Rivara et al., *supra* note 5.

The nature of the surface onto which the child allegedly fell can be as important as the height of the fall.¹⁰¹ Generally speaking, the harder the surface, the more dangerous the fall. Whereas a one-foot drop onto concrete creates a gravity force of approximately 500 g., a similar drop onto a rubber mat (1 1/8 inches thick) creates a force of only one to two g.¹⁰²

It is extremely rare for falls from distances of three feet or less to result in serious, life-threatening injury. Helfer and his associates questioned parents regarding 219 falls at home of 161 children under six years of age; none "suffered any serious, life-threatening injuries from their falls." The authors reached a similar conclusion when reviewing eighty-five reports of children under six falling approximately three feet onto a noncarpeted hospital floor.¹⁰³ Similarly, Nimityongskul and Anderson found no severe injuries in reviewing reports of seventy-six one to three foot falls by children under seventeen years of age onto a "hard vinyl tile" hospital floor.¹⁰⁴ Finally, Lyons and Oates reviewed 207 falls of children under six falling two to four and a half feet onto a vinyl hospital floor; none suffered life-threatening, multiple, or severe injuries.¹⁰⁵

Studies that find serious, life-threatening injury among children allegedly falling from short distances¹⁰⁶ have typically relied on uncorroborated histories given by caretakers.¹⁰⁷ One study compared injuries resulting from uncorroborated falls to corroborated falls found that the uncorroborated cases had a six times higher rate of serious injury.¹⁰⁸ The only cases of severe injury among the corroborated falls were falls from four to five feet onto protruding surfaces, which led to depressed skull fractures, but with no loss of consciousness.¹⁰⁹

Although it is highly unlikely that a short fall will lead to a fracture, it is possible for it to do so. Researchers at the Yale University of Medicine examining fractures

101. Rivara et al., *supra* note 5.

102. The threshold for serious injury to the head is 50 g., Thomas E. Reichelderfer et al., *Unsafe Playgrounds*, 64 PEDIATRICS 962, 963 (1979); however, death may occur at 160 g., Theodora B. Sweeney, *X-Rated Playgrounds?*, 64 PEDIATRICS 961 (1979).

103. Helfer et al., *supra* note 92, at 534.

104. Nimityongskul & Anderson, *supra* note 92, at 185.

105. Lyons & Oates, *supra* note 92.

106. Smith et al, *Injuries in Children Sustained in Free Falls*, *supra* note 56.

107. JAMES T. WESTON, *THE CHILDHOOD MALTREATMENT SYNDROME* (Werner U. Spitz & Russell S. Fister eds., 1973)); David L. Chadwick et al., *Deaths from Falls in Children: How Far is Fatal?*, 31 J. TRAUMA 1353 (1991); Elizabeth E. Gilles, *Abusive Head Injury in Children: A Review*, 20 W. ST. L. REV. 335, 351 (1993) [hereinafter Gilles, *Abusive Head Injury in Children*]; Williams, *supra* note 99. Chadwick et al., reviewed 317 cases of infants and children who died with a history of having fallen. They found that if "the histories of short falls are accepted as correct, the conclusion would be reached that the risk of death is eight times greater in children who fall from one to four feet than for those who fall from 10 to 45 feet." Chadwick et al., *supra*, at 1355. They concluded that when a seriously injured child is presented with a history of a short fall, it is probable that the child was in fact abused.

108. Williams, *supra* note 99. A fall was classified as corroborated if the fall was witnessed either by two or more people, or by a nonrelative not involved in the child's care.

109. *Id.*

in children under three years of age have concluded that "[e]ven falls . . . from relatively low objects, such as beds, chairs, tables, or parents' arms, resulted in fractures."¹¹⁰ Clavicle fractures were most common when falls were less than two feet, whereas skull fractures predominated among children falling two to four feet. However, research that surveys parents regarding their children's experience at home or that reviews falls while in the hospital suggests that fractures are quite rare. In four studies, including a total of 917 short falls (ranging from one to five feet), there were seven skull fractures, four clavicle fractures, and one humerus fracture.¹¹¹ In falls down stairs, "[s]ingle injuries of the head or distal extremity [the lower leg or arm] predominates. A fall down a flight of stairs seldom causes injury to the trunk [the body excluding the head and extremities] or proximal extremity."¹¹² Stairway falls are less serious than free falls of the same vertical distance.¹¹³ Injuries sustained from falls from shopping carts or the tipping over of such carts, although frequently involving the head, rarely lead to serious intracranial pathology.¹¹⁴ In order for death to occur, the research on free (vertical) falls suggests that it is necessary for the child to fall twenty feet or more.¹¹⁵

110. Leventhal et al., *supra* note 4, at 91; see Thomas et al., *supra* note 63 (reporting on femur and humerus fractures). Leventhal et al., found 24 cases in which falls of less than 60 centimeters (about two feet) which were judged by both the directors of the child abuse team and pediatric radiologists as accidental, eight suffered short, linear fractures of the parietal bone. The authors found 23 cases in which falls from 60 to 120 centimeters led to fractures, including three multiple parietal fractures and one had fractures of the frontal bone and femur, 23 skull fractures, two radius/ulna fractures, two humerus fractures, and four femur fractures. Overall, 81% of the cases in which a fall was described were classified as accidental. Leventhal et al., *supra*.

111. See Helfer et al., *supra* note 92, at 534 (219 three-foot falls in children under five reported by parents: three clavicle fractures, one humerus fracture, two skull fractures); *id.* (85 three-foot falls in the hospital in children under five: one skull fracture); Kravitz et al., *supra* note 92, at 873 (330 20- to 60-inch falls in children under one: three skull fractures); Lyons & Oates, *supra* note 92 (207 2- to 4½-foot falls in the hospital in children under six: one skull fracture and one clavicle fracture); Nimityongskul & Anderson, *supra* note 92 (76 one- to three-foot falls in the hospital in children under 16: one skull fracture).

112. Joffe & Ludwig, *supra* note 56, at 458. Among 363 falls of children (up to 18 years of age) down stairs, there were six skull fractures, two humerus fractures, 13 fractures of the ulna/radius or tibia/fibular (the "distal extremities"). Notably, there were no femur fractures or rib fractures. Chiaviello et al. examined the records of 69 children under five years of age falling down stairs; five suffered a skull fracture, four experienced "extremity injury," and three experienced "truncal injury." Chiaviello et al., *supra* note 95. Whether the extremity and truncal injuries were fractures is unclear. The authors noted, consistent with the Joffe and Ludwig study, that "[i]njury to more than one body region did not occur in this series." *Id.* at 679.

113. See Joffe & Ludwig, *supra* note 56, at 460.

114. Gary A. Smith et al., *Injuries to Children Related to Shopping Carts*, 97 PEDIATRICS 161, 161-65 (1996).

115. Christopher Swalwell wrote:

[N]umerous . . . publications have addressed the issue of injuries sustained when children fall from a wide variety of heights. These clinical studies include more than 1,500 cases of falls from heights up to 17 stories. A total of 25 deaths have been reported. Excluding the seven deaths in Chadwick's study that were suspected to be due to child abuse and not to the alleged fall, there were no fatalities due to falls from heights of less than two stories.

Christopher Swalwell, *Head Injuries from Short Distance Falls*, 14 AM. J. FORENSIC MED. & PATHOLOGY 171, 172 (1993) (citing David L. Chadwick et al., *supra* note 107); see, e.g., Barbara Barlow et al., *Ten Years of Experience with Falls from a Height in Children*, 18 J. PEDIATRIC SURGERY 509 (1983) (61 children falling 10 feet or more; all fatal falls were more than three stories); Janet L. Meller & Dennis W. Shermata, *Falls in Urban Children*, 141

In sum, the research supports the conclusion that short falls rarely if ever lead to serious injury. When a parent claims that a child's injuries are due to a fall, it is important to carefully question the parent regarding the height of the fall, the surface onto which the child fell, and upon which part of the body the child fell. It is also important to determine whether entrapment of a limb occurred (e.g., was an extremity caught in the slats of the crib?). Finally, one should determine whether there were other witnesses to the fall as a means of corroborating or questioning the caretakers' story.

2. Diseases Including Osteogenesis Imperfecta

If a young child has sustained multiple fractures, it is important to consider whether the child suffers from osteogenesis imperfecta, infantile cortical hyperostosis, metabolic bone disease (scurvy, rickets, hypophosphatasia, and copper abnormalities), syphilis, osteoid osteoma, neoplasms, and osteomyelitis (bone infection). These conditions can be "ruled out by the appearance of the bone on the radiograph [x-ray], and by the levels of calcium, phosphorous, alkaline phosphatase [and other metabolites] in the serum."¹¹⁶ To the extent that forms of these conditions mimic injuries typically attributable to abuse, they are far less common than abuse.¹¹⁷

A frequently litigated claim is that an abused child suffers from osteogenesis imperfecta ("OI," colloquially known as "brittle bone disease"). OI can be differentiated from fractures attributable to abuse by an examination of the radiographs, a clinical examination of the child, and a history of the existence of OI or symptoms of OI in the child's family. When necessary, a definitive collagen test can be performed. (Collagen is a substance found within connective tissue, including the bones.)

Most individuals with OI have blue sclerae (sklair-AY).¹¹⁸ The sclerae are the whites of the eyes, and when they are thinner, they are blue. Many normal infants up to three to four months have blue sclerae as well; so although the absence of blue sclerae makes OI unlikely, the presence of blue sclerae is not definitive.¹¹⁹

Almost 90% of individuals with nonlethal forms of OI have an excessive number of wormian bones detectable by x-ray. Wormian bones are little bones in the sutures of the skull. The sutures are the spaces between the various bones in the skull which

AM. J. DISEASES CHILDREN 1271 (1987) (48 children falling an average of 2.7 stories; only fatal fall was from 13 stories); Catherine A. Musemeche et al., *Pediatric Falls from Heights*, 31 J. TRAUMA 1347 (1991) (70 children falling 10 feet or more; all survived).

116. Carty, *Brittle or Battered*, *supra* note 36, at 350 (copper deficiency leads to osteoporosis which is evident in x-rays); Stephen Ludwig, *Psychosocial Emergencies*, in TEXTBOOK OF PEDIATRIC EMERGENCY MEDICINE 1135 (Gary Fleisher & Stephen Ludwig eds., 1988); Interview with Ralph S. Lachman, M.D., *supra* note 39.

117. Ludwig, *supra* note 116, at 1135.

118. Deborah S. Ablin et al., *Differentiation of Child Abuse from Osteogenesis Imperfecta*, 154 AJR 1035, 1042 (1990); Carty, *Brittle or Battered*, *supra* note 36, at 351.

119. Ablin et al., *supra* note 118, at 1040.

grow together as the infant matures. In newborns with OI and in less severe cases of OI, however, excessive wormian bones may not be visible.¹²⁰ Conversely, many normal children have a small number of wormian bones, so it is important to determine if the number noted is abnormally large.¹²¹

All or virtually all individuals with OI show signs of osteoporosis (osteopenia, reduced bone density). Osteoporosis is usually detectable by x-ray,¹²² except in some less severe cases of OI.

A claim that a child has a "mild" form of OI is implausible if the child suffers fractures and is not yet walking,¹²³ or if the child suffers from metaphyseal corner fractures and/or rib fractures.¹²⁴ Furthermore, subdural hematoma and retinal hemorrhages are not features of osteogenesis imperfecta.¹²⁵

Some purported signs of OI are not in fact useful in diagnosing the disorder. "The presence or absence of bruising over a fracture site gives no indication as to the cause of a fracture."¹²⁶ Laxity of the joints (hypermobility) and a triangular facial appearance are often seen in patients with OI, but are too nonspecific (they occur too frequently among those without OI) to be of much diagnostic value.¹²⁷

In court, it is often asserted that a child suffers from a Type IV form of OI, in which the child may be presented without blue sclerae, wormian bones, osteoporosis, or a family history of OI. It has been estimated that the chance that fractures in a child under one are attributable to such a case is only three in a million.¹²⁸ Most of the reported cases of OI with such features are contained in a work by Colin Paterson and his colleagues, and this work has been sharply criticized.¹²⁹ Moreover, even if

120. *Id.* at 1042; Sheila Gahagan & Mary Ellen Rimsza, *Child Abuse or Osteogenesis Imperfecta: How Can We Tell?*, 88 PEDIATRICS 987, 990 (1991).

121. Address by Ralph S. Lachman, M.D., *supra* note 86.

122. Ablin et al., *supra* note 118, at 1037; Carty, *Fractures Caused by Child Abuse*, *supra* note 4, at 857; Interview with Ralph S. Lachman, M.D., *supra* note 39.

123. Carty, *Fractures Caused by Child Abuse*, *supra* note 4, at 850.

124. Ablin et al., *supra* note 118, at 1042; Carty, *Fractures Caused by Child Abuse*, *supra* note 4, at 857; Interview with Ralph S. Lachman, M.D., *supra* note 39. *But see* Gahagan & Rimsza, *supra* note 120, at 990 (noting a metaphyseal fracture in a child whose OI was confirmed by collagen testing, and whose x-rays did not show other signs of OI).

125. Carty, *Fractures Caused by Child Abuse*, *supra* note 4, at 857; *see* Gahagan & Rimsza, *supra* note 120, at 991 (retinal hemorrhages or intracranial injuries make OI an unlikely cause of the observed fractures). Intracranial injuries are discussed at greater length in Part V.

126. Carty, *Brittle or Battered*, *supra* note 36, at 351; *see* L.S. Taitz, *Child Abuse and Metabolic Bone Disease: Are They Often Confused?*, 302 BMJ 1244 (1991) [hereinafter Taitz, *Child Abuse and Metabolic Bone Disease*].

127. Taitz, *Child Abuse and Metabolic Bone Disease*, *supra* note 126, at 1244.

128. Carty, *Fractures Caused by Child Abuse*, *supra* note 4, at 857 (citing L.S. Taitz, *Child Abuse and Osteogenesis Imperfecta*, 296 BRIT. MED. J. 292 (1988)).

129. *See* Colin R. Paterson & Susan J. McAllion, *Child Abuse and Osteogenesis Imperfecta*, 295 BRIT. MED. J. 1561 (1987). *But see* Ablin et al., *supra* note 118, at 1041 ("Paterson and colleagues did not explain how they diagnose OI subtype IVA with neither radiologic confirmation nor progressive deformity (except fractures), without family history, with normal sclera and teeth, and without fracture recurrence in protective environment."). Taitz reported on his personal experience with Dr. Paterson's diagnostic method:

such a case occurs, a collagen test ought to produce evidence of OI more than 85% of the time.¹³⁰ "[T]he frequency of child abuse is orders of magnitude greater than [the nonlethal forms of] OI. Providing initial protection to likely victims of child abuse should not be significantly delayed by a prolonged search for a rare disease."¹³¹

3. Birth Trauma

Birth trauma, particularly following breech delivery (when the child is born buttocks first) may lead to a number of fractures that may be difficult to distinguish from abuse, especially fractures of the clavicle, including the end of the clavicle; the humerus; and the femur.¹³² Moreover, birth trauma may lead to metaphyseal and epiphyseal fractures and to periosteal hemorrhaging.¹³³ However, fractures should show signs of callus (new bone) formation before the child is two weeks old, so dating of the fracture can determine if it could have been the result of birth trauma.¹³⁴

V. INTRACRANIAL INJURIES

Injuries to the head and intracranial contents (within the skull) often occur with physical abuse.¹³⁵ Unintentional injuries and a large number of diseases may also result in intracranial pathology. Although there are some cases of intracranial injury that are clearly abusive, many cases will not be so clear, especially if the injuries are less severe. Following the definitions of key terms, we will provide an overview of abusive intracranial injuries and how they occur. We will discuss the term "shaken

Dr. Paterson needs to explain how he diagnoses osteogenesis imperfecta without a family history, no radiological features, and no repeated fractures. In this case he based it on absence of bruises, a single fracture in a cousin, and joint hyperflexibility in *both* parents—a curious finding in an autosomal dominant condition.

Taitz, *Child Abuse and Osteogenesis Imperfecta*, 296 BRIT. MED. J. 292, 292 (1988). In a different case, "Dr. Paterson first gave evidence at the trial for the defense diagnosing osteogenesis imperfecta. Later after further research he changed his diagnosis to copper deficiency." *Id.*

130. Gahagan & Rimsza, *supra* note 120, at 990.

131. Swischuk, *supra* note 41, at 171.

132. Paula W. Brill & Patricia Winchester, *Differential Diagnosis of Child Abuse*, in DIAGNOSTIC IMAGING OF CHILD ABUSE, *supra* note 36, at 221, 221-22 (humerus and clavicle); Hobbs, *Fractures*, *supra* note 36 (clavicle and humerus); Kogutt et al., *supra* note 50 (end of clavicle).

133. Brill & Winchester, *supra* note 132, at 221 (epiphyseal separations and periosteal hemorrhaging); Carty, *Fractures Caused by Child Abuse*, *supra* note 4, at 850 (metaphyseal fractures); Swischuk, *supra* note 41, at 169 (epiphyseal-metaphyseal fractures).

134. Hobbs, *Fractures*, *supra* note 36; O'Connor & Cohen, *supra* note 83, at 107; Swischuk, *supra* note 41, at 169. For more on the dating of fractures, see subpart IV.E.

135. Although these injuries often involve the face and facial bones, ears, and teeth, the focus of this discussion will be on intracranial pathology. Moreover, injuries to the scalp are often also due to abuse. Such injuries include traumatic alopecia, in which hair loss and soft tissue swelling may occur after violent hair pulling, scalp bruising, cephalhematoma (a collection of blood under the periosteum of the skull), and subgaleal hematoma (a collection of blood in the galeal space within the scalp between the scalp and the periosteum of the skull). For a discussion of scalp injuries and abuse, see generally Schmitt, *supra* note 12, at 188-89.

infant syndrome” and how it is misused, and advocate the use of the term “shake-impact syndrome.” The overview will conclude with a discussion of how abusive injuries can be distinguished from nonabusive injuries. We will then discuss each type of injury in turn, noting the extent to which it suggests abuse and the possibility of etiologies other than abuse.

The literature on intracranial and intraocular injury is filled with inaccuracies. Consider how many cases were included in the study, the criteria for classifying children as abused or nonabused (frequently, inclusion criteria are omitted entirely), and whether the discussion confines itself to the cases analyzed. Be especially wary of incidental cases included in the discussion that were not rigorously scrutinized according to the study protocol. Also, beware of references in the literature to theories presented as fact, without critical analysis. As an example, consider the oft-repeated statement “retinal hemorrhages result from cardiopulmonary resuscitation.” As discussed below, the rare case reports offered in support of this proposition are flawed, and the proposition is not supported either by surveys of children with retinal hemorrhages or by the only experimental animal model in existence. Much remains unknown about the accurate diagnosis of abusive head injury in the young child. Every child deserves complete evaluation of all case data.

A. Glossary of Terms

1. Normal Anatomy

Arachnoid: One of the three layers of the meninges, the coverings of the brain and spinal cord. Three membranes lie between the brain and the skull. The pia lies closest to the brain, the arachnoid lies between the pia and the dura, and the dura lies closest to the skull.

Bridging veins: Veins that originate in the brain, cross the subdural space, and drain into the venous sinuses.

Cerebellum: Portion of the brain situated in the posterior fossa (lower back of the skull), lying below the cerebrum.

Cerebrospinal fluid (CSF): Fluid that is made within the ventricles (normal cavities) of the brain, and which circulates into the subarachnoid space, thence is reabsorbed into the bloodstream.

Cerebrum: The main portion of the brain, occupying the upper part of the cranial (skull) cavity. Has two large hemispheres connected in the middle by a large bundle of fibers called the corpus callosum.

Convexity: In this context, the outer surface of the cerebral hemispheres, next to the skull. The “hemispheric convexity.”

Dura: Also known as dura mater. The outermost, toughest layer of the meninges covering the brain. Has two main infoldings: the falx (or falx cerebri), which lies between the two hemispheres of the cerebrum, and the tentorium (or tentorium cerebelli), which lies between the cerebrum and the cerebellum.

Falx cerebri: Also known as the falx. Infolding of the dura between the two hemispheres of the cerebrum.

Fontanelle: Soft spot in the head where the skull bones do not meet; in infants, typically used in reference to the anterior fontanelle.

Fossa: Depression in the floor of the skull.

Frontal lobe: The anterior (front) portion of the brain. Technically speaking, it is anterior to the central sulcus and above the lateral fissure. See Figure 5.

Gray matter: The areas of the brain which appear gray on visual inspection. Occurs at the surface of the brain (cortex) and deep within the brain (*e.g.*, basal ganglia and thalamus). Composed of nerve cell bodies. Compare white matter defined *infra*.

Gyrus (pl. gyri): Bumps of the cerebrum formed by the normal folding of the cortex.

Leptomeninges: The arachnoid and pia.

Meninges: The three membranes covering the brain and spinal cord: dura mater, arachnoid, and pia.

Myelin: An insulating fatty sheath around nerve fibers.

Occipital lobe: The area of the cerebrum located at the back of the brain. Technically speaking, it lies behind an imaginary line drawn from the parieto occipital fissure to the preoccipital notch. See Figure 5.

Optic nerve: The second cranial nerve which travels from the back of the eye to the brain.

Optic nerve sheath: The three layers of membrane (or meninges) covering the optic nerve.

Parenchymal: Within the brain substance.

Parietal lobe: The portion of the brain that separates the frontal from the occipital lobes and lies above the temporal lobe. Technically speaking, it extends from the central sulcus to the parieto occipital fissure, and is separated from the temporal lobe by an imaginary line from the horizontal portion of the lateral fissure to the middle of the line demarcating the occipital lobe. (See Figure 5).

Pia: Also known as pia mater. The innermost part of the meninges, closely approximating the surface of the brain.

Retina: The innermost part of the eyeball, consisting of ten layers containing a variety of nerve and connective cells, as well as extensive capillary beds.

Subarachnoid space: The space between the arachnoid and the pia in which the CSF flows.

Subdural space: A potential space within the dura.

Sulcus (pl. sulci): A general term for the grooves on the surface of the brain which separate the gyri.

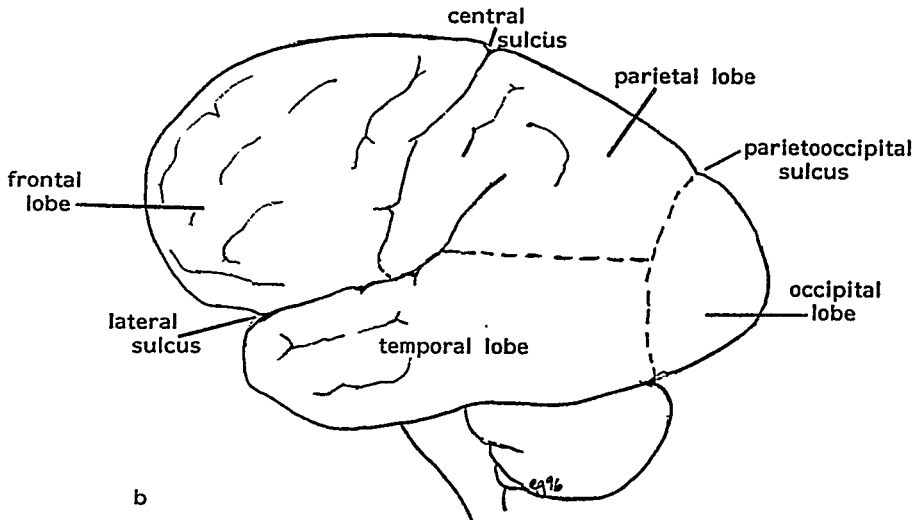
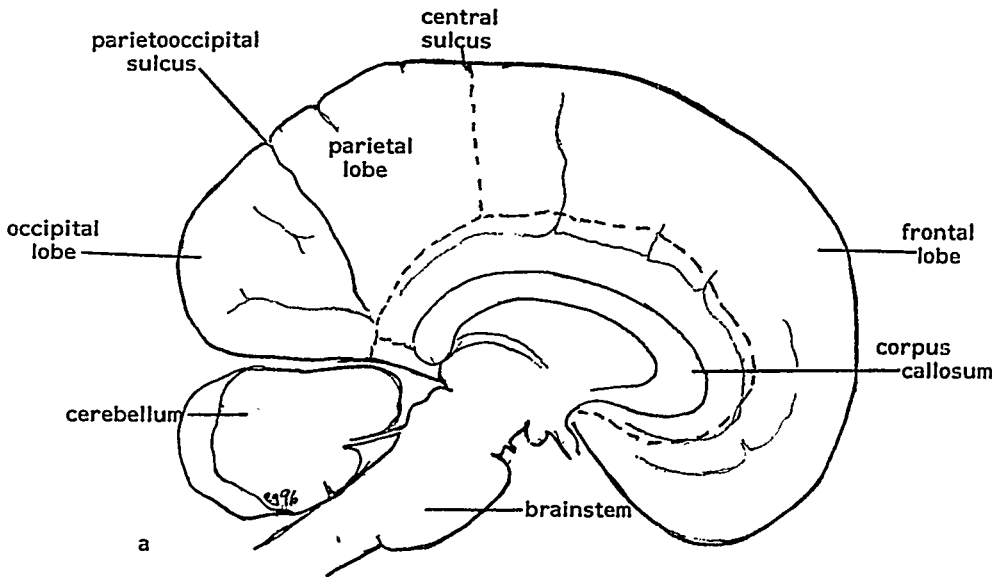
Temporal lobe: Portion of the brain behind the frontal lobe and below the parietal lobe. (See Figure 5).

Tentorium cerebelli: Also known as the tentorium. Infolding of the dura overlying the posterior fossa, separating the occipital and inferior temporal lobes from the cerebellum (which lies underneath). The tentorium cerebelli separates the cerebrum and the cerebellum, which sits below the cerebrum in the portion of the back of the skull called the posterior fossa.

Venous sinuses: Also known as the dural sinuses. Veined structures that drain blood from the brain. Some, such as the superior sagittal sinus, are relatively fixed in their location.

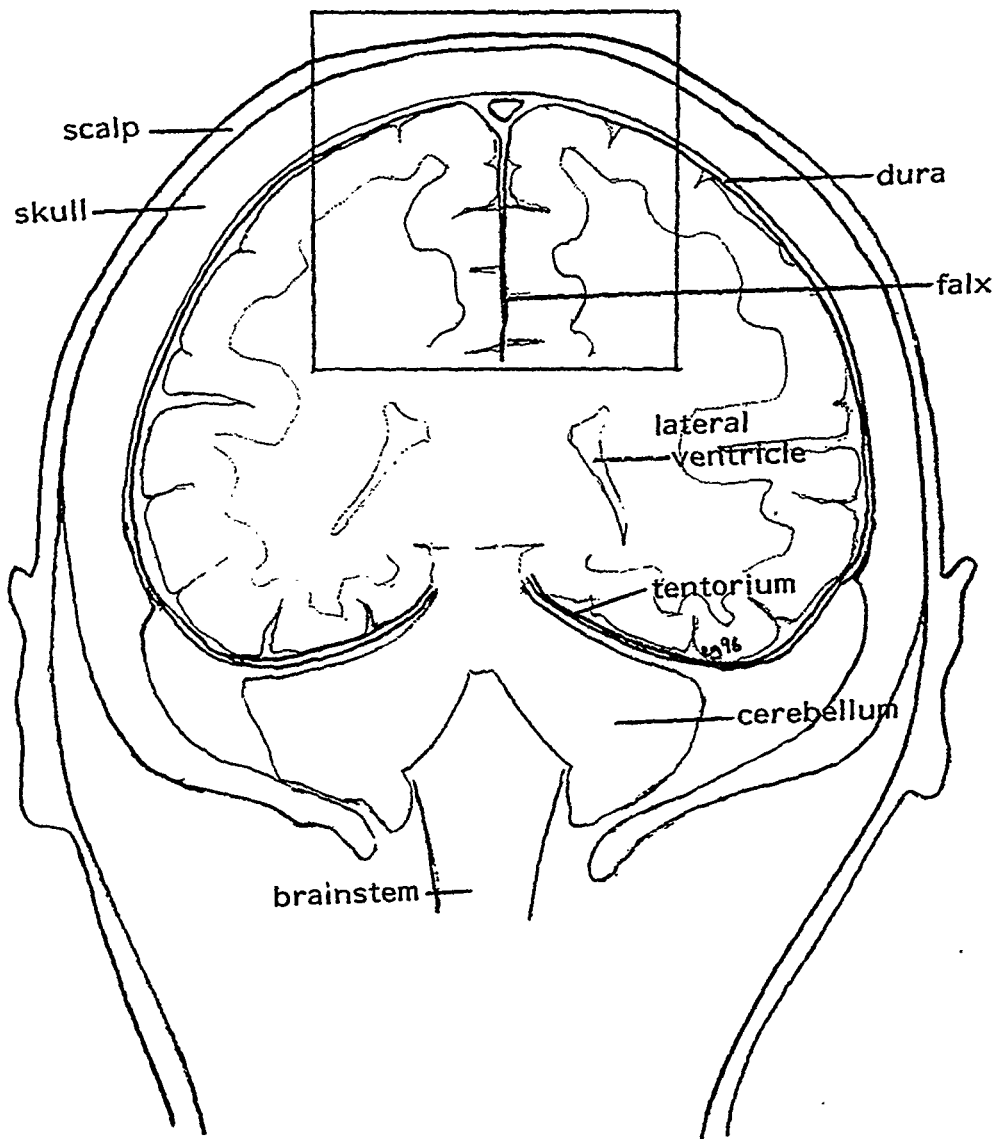
Ventricles: Normal cavities within the brain, in which cerebrospinal fluid is made.

White matter: The connecting nerve fibers which in the mature human are covered with myelin, a substance that acts like the covering on insulated electrical wire. Infant brains are not completely myelinated. Compare gray matter defined *supra*.



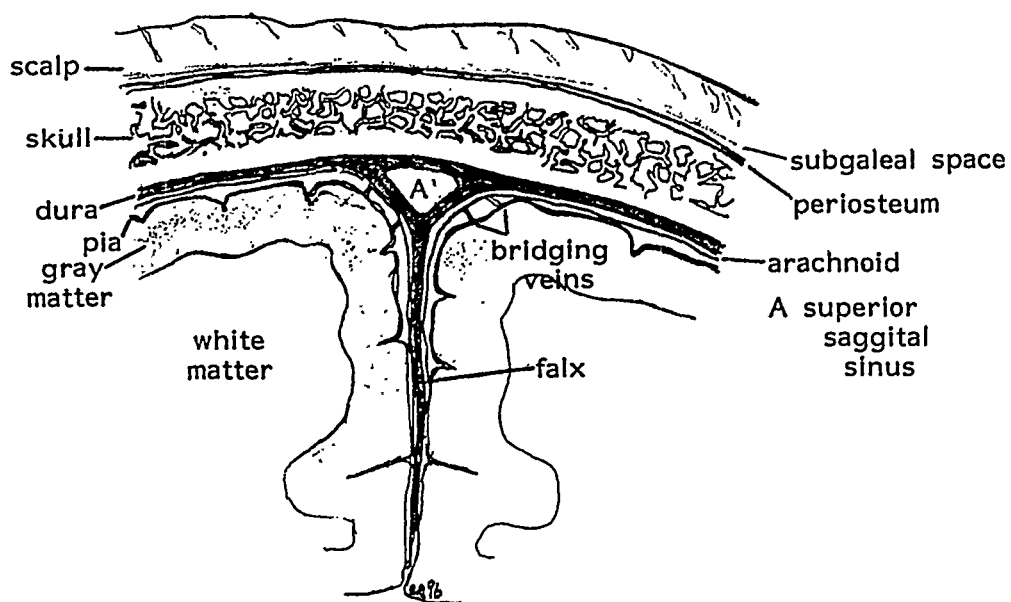
a. medial view of cerebral hemisphere (left), cerebellum & brainstem
b. lateral view of cerebral hemisphere (left), cerebellum & brainstem

Figure 6: Surrounding the Brain



coronal section - head

Figure 7: Surrounding the Brain: Detailed View



coronal view of scalp, skull, meninges and cerebrum

2. Abnormal Conditions

Aneurysm: A blood-filled sac formed by a localized dilatation (an abnormal outpouching), usually of an artery.

Angiopathy: Any disease of the blood vessels.

Arteriovenous malformation: A developmental deformity of arteries and veins.

Blood dyscrasia: Any abnormal or pathologic condition of the blood.

Brain swelling: Refers to an increase in brain volume. As used here, the term can refer to an increase in brain tissue water (edema) or to an increase in intravascular blood (cerebral hyperemia).

Cerebral infarction: A lack of blood (ischemia) to a portion of the brain due to occlusion of the arterial supply or venous drainage, which leads to tissue death.

Coagulopathy: Abnormality affecting the coagulation of blood.

Coagulation: The process by which bleeding stops.

Contusion (of the brain): In a sense, a bruise of the brain substance, although not the same as soft tissue bruises. These are usually bloody (hemorrhagic) but may be nonhemorrhagic. In infants, particularly younger than six months, typically refers to contusional white matter tears, which are slit-like tears of the brain that occur at the junction of the white and gray matter or within the white matter.

Diffuse axonal injury: Also known as white matter shearing injury. Injury to axons (part of the nerve cells) in the brain, typically without bleeding.

Edema: An increase in brain volume due to an increase in brain water content. Compare brain swelling defined *supra*.

Encephalitis: Inflammation of the brain. Often used in reference to brain infection.

Epidural hematoma: Also known as extradural hematoma. A blood collection outside of the dura and inside the skull. Usually due to the rupture

of the middle meningeal artery, which courses along the inside of the skull table.

Extra-axial collection: Collections of fluid outside of the brain, including epidural and subdural hematomas and subdural effusions.

Extra-cerebral hemorrhage: Subdural or subarachnoid bleeding. Also known as extra-axial hemorrhage.

Hydrocephalus: Abnormal accumulation of cerebrospinal fluid (CSF) within the skull. Two main types: communicating, in which there is free movement of fluid from the ventricles to the subarachnoid space, and noncommunicating, in which the flow of CSF through the ventricular system is blocked.

Hygroma: A controversial term referring to the accumulation of fluid in the subdural space. Some believe they are resolving subdural hematomas, others believe they are abnormal collections of CSF in the subdural space. See also extra-axial collection defined *supra*.

Infarction: A localized area of tissue death produced by occlusion of the arterial supply or the venous drainage.

Interhemispheric hematoma: Also called a falcine hematoma. A collection of blood between the hemispheres. May be subarachnoid, subdural, or interdural (between the leaves of the falx cerebri).

Ischemia: Deficiency of blood, attributable to functional constriction or actual obstruction of a blood vessel.

Mass effect: The effect caused by a space-occupying lesion (such as a large subdural hematoma) exerting pressure on surrounding or subjacent structures (including the brain).

Meningitis: Inflammation of the meninges. Usually due to an infection.

Seizure: A paroxysmal and transient (sudden and temporary) disturbance of brain function that may be manifested as a loss of consciousness or abnormal motor, sensory, or psychic phenomena. Attributable to disturbance of the electrical activity of the brain.

Subarachnoid hemorrhage: Bleeding within the subarachnoid space, which mixes with the cerebrospinal fluid.

Subdural hematoma: A localized collection of blood beneath the dura mater. Collects in the potential space that exists between the dura and arachnoid.

Thrombocytopenia: Deficiency of blood platelets, which aid in coagulation.

3. Diagnostic Tools and Terms

Axial view: (See Figure 8).

Cerebral angiography: Imaging of the brain's blood vessels by peripheral injection of a radio-opaque dye.

Computerized tomographic scan (CT scan): Imaging through radiographic analysis of sections of the brain, which are then summed by a computer. Different substances appear different based on their density.

Coronal view: (See Figure 8).

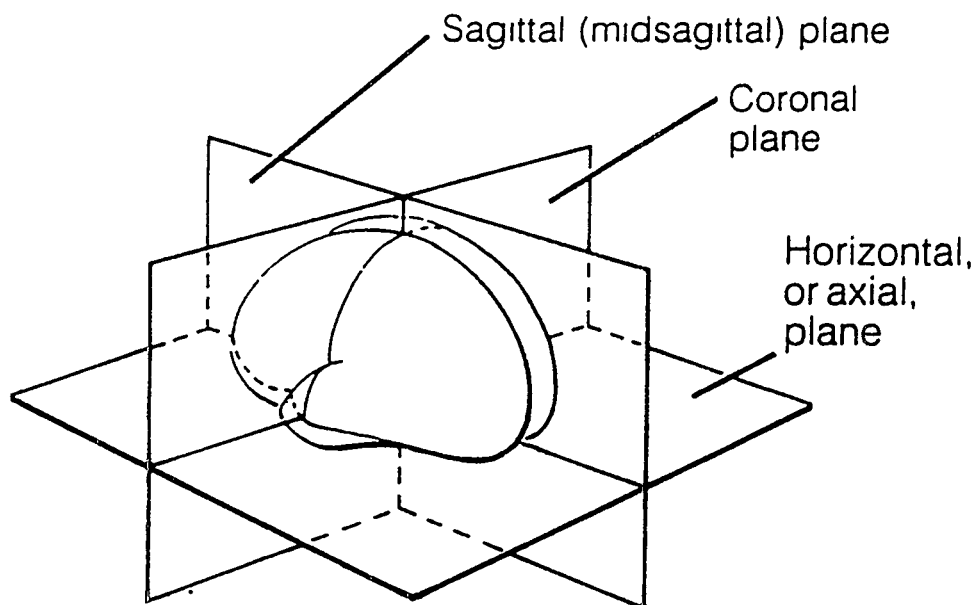
Lateral view: (See Figure 8).

Magnetic resonance imaging (MRI): Imaging through electromagnetic analysis of sections of the brain, which are then summed by a computer. Different substances appear different based on their intensity.

Magnetic resonance angiography (MRA): Like cerebral angiography, but much less invasive, only needing peripheral injection of a paramagnetic material to illuminate blood vessels. Lacks some of the resolution of traditional cerebral angiography.

Sagittal view: Image of the middle of the brain, as viewed laterally.

Figure 8: Orientations from Which the Brain Can Be Viewed[†]



[†] This figure is reprinted by permission of the author, Stephen G. Waxman and the copyright holder, Appleton & Lange. Stephen G. Waxman, *CORRELATIVE NEUROANATOMY* 223 (23d ed. 1996).

B. Overview of Abusive Intracranial and Intraocular Injuries

1. Types of Injuries

A number of findings are associated with infants who have sustained head injury due to abuse, including soft tissue injuries, ocular findings (such as retinal hemorrhages and ocular nerve sheath hemorrhages), extra-axial blood collections (subdural hematomas and subarachnoid hemorrhage), diffuse brain injury (such as diffuse axonal injury), focal brain injury (such as white matter tears), and skull fractures.¹³⁶ Although many injuries occur because of the initially applied forces, many evolve secondarily. These include brain swelling, often with increased intracranial pressure, and brain infarctions.¹³⁷ Morbidity and mortality are high. Survivors often exhibit significant behavioral, cognitive, and motor consequences, as well as visual abnormalities and seizures.¹³⁸ Repetitive injury is also common.¹³⁹

2. How the Injuries Occur

The vast majority of head injuries due to abuse occur in children less than three years of age, and the younger the child, the more vulnerable to serious injury. Infant neck muscles are weak, and their heads are proportionally larger in relation to their

136. Stephen Ludwig & Matt Warman, *Shaken Baby Syndrome: A Review of 20 Cases*, 13 ANNALS EMERGENCY MED. 104, 105 (1984) (20 shaken babies; 10 had subdural hematomas, 5 subarachnoid hemorrhages, 10 retinal hemorrhages, and 8 cerebral contusions). Soft tissue injuries are discussed in Part III (Bruises), and skull fractures are discussed in subpart IV.F.

137. Derek A. Bruce, *Head Injuries in the Pediatric Population*, [Feb.] CURRENT PROBS. PEDIATRICS 66, 72 (1990) (increased intracranial pressure results from injury); Ronald A. Cohen et al., *Cranial Computed Tomography in the Abused Child with Head Injury*, 146 AJR 97 (1986) (37 abused children with serious head injury, 95% less than one year old; 24 or 65% exhibited cerebral edema); Y. Frank et al., *Neurological Manifestations in Abused Children Who Have Been Shaken*, 27 DEV. MED. & CHILD NEUROLOGY 312, 314 (1985) (4 shaken babies under 4 months old; edema and brain infarction); Joseph Giangiacomo et al., *Sequential Cranial Computed Tomography in Infants with Retinal Hemorrhages*, 95 OPHTHALMOLOGY 295, 298 (1988) (common findings include cerebral edema, brain atrophy, and massive brain infarction); Gregory D. Launius et al., *Radiology of Child Abuse, in CHILD MALTREATMENT: A CLINICAL GUIDE AND REFERENCE*, *supra* note 1, at 27, 42 (65% of abused infants exhibited cerebral edema); Ludwig & Warman, *supra* note 136 (20 shaken babies; 55% with full or bulging fontanelle, 56% exhibited head circumference greater than 90% for age).

138. Derek A. Bruce & Robert A. Zimmerman, *Shaken Impact Syndrome*, 18 PEDIATRIC ANNALS 482, 492 (1989); see Lucinda J. Dykes, *The Whiplash Shaken Infant Syndrome: What Has Been Learned?*, 10 CHILD ABUSE & NEGLECT 211, 217 (1984) (reviewing research; very high incidence of neurologic morbidity and death); Launius, *supra* note 137, at 39 ("[R]esidual changes can be seen in nearly 80% of those surviving intracranial injury. It is suspected that many cases of mental retardation can be ascribed to child abuse."); Ludwig & Warman, *supra* note 136 (20 shaken infants, mean age six months; 15% died, and of those who survived, 50% had significant morbidity, including "blindness, visual impairment, motor impairment, seizures, and developmental delay").

139. See Randall Alexander et al., *Serial Abuse in Children Who Are Shaken*, 144 AM. J. DISEASES CHILDREN 58 (1990) (24 abused children with injuries from shaking; 71% had evidence of prior abuse, neglect, or both); Susan Elner et al., *Ocular and Associated Systemic Findings in Suspected Child Abuse*, 108 ARCHIVES OPHTHALMOLOGY 1094, 1098 (1990) (7 abused infants who died from child abuse with ocular injuries; "[i]n 3 cases, hemosiderin indicating old hemorrhage was found in the retina").

body. As a result, shaking of an infant can result in violent rotation of the head and its contents.¹⁴⁰ Compared to an adult's brain, the infant's brain is especially vulnerable to injuries from shaking: the infant's brain has less myelin¹⁴¹ and a higher water content, making it less rigid.¹⁴²

The contents of the skull are most susceptible to rotational acceleration, which occurs when the head moves through an arc. The intracranial contents move variably depending on their composition and location. When rotation occurs, shear forces are greatest at the junctures between tissues that differ in their rigidity.¹⁴³ For instance, the dura is attached to the skull at a number of points, and being relatively rigid, moves less than the substance of the brain, leading to the stretching of the bridging veins that run between the brain and the dura.¹⁴⁴ Most subdural hematomas are thought to develop from the tearing of these vessels. The brain itself is composed of gray matter and white matter, which are of different densities. Rotation leads to shearing forces at the interface between gray and white matter.¹⁴⁵ These forces can lead to diffuse axonal injury and white matter contusional tears.

Impact by itself is unlikely to result in serious intracranial injury if there is no rotational component. However, impact in combination with rotation enhances the magnitude of injury, since the softer brain and more malleable skull of the infant facilitates significant deformation during impact.¹⁴⁶

140. Caffey, *supra* note 40, at 401.

141. Brown & Minns, *supra* note 89, at 860; Gilles, *Abusive Head Injury in Children*, *supra* note 107, at 348; G. H. Vowles et al., *Diffuse Axonal Injury in Early Infancy*, 40 J. CLINICAL PATHOLOGY 185, 189 (1987).

142. A.C. Duhaime et al., *The Shaken Baby Syndrome*, 66 J. NEUROSURGERY 409, 414 (1987) [hereinafter Duhaime et al., *The Shaken Baby Syndrome*]; Dykes, *supra* note 138, at 216; Gilles, *Abusive Head Injury in Children*, *supra* note 107; David F. Merten & Becky L.M. Carpenter, *Radiologic Imaging of Inflicted Injury in the Child Abuse Syndrome*, 37 PEDIATRIC CLINICS N. AM. 815, 825 (1990).

143. Lindell R. Gentry et al., *MR Imaging of Head Trauma: Review of the Distribution and Radiopathologic Features of Traumatic Lesions*, 150 AJR 663, 670 (1988) ("Injuries from rotational forces are expected to correspond closely in location to the sites of maximum shear-strain. Shear-strain will be greatest at the junction of tissues of different rigidity (gray/white matter interface, brain/CSF interface, skull/brain interface, dura matter/brain interface."); Ayub K. Ommaya & Thomas A. Gennarelli, *Cerebral Concussion and Traumatic Unconsciousness*, 97 BRAIN 633, 652 (1974) ("Rotational components of accelerative trauma to the head produce a graded centripetal progression of diffuse cortical-subcortical disconnection phenomena which is always maximal at the periphery and enhanced at sites of structural inhomogeneity.").

144. Thomas A. Gennarelli & Lawrence E. Thibault, *Biomechanics of Acute Subdural Hematoma*, 22 J. TRAUMA 680, 682 fig.2 (1982) ("Parasagittal bridging veins, being the sole connection between the skull and the brain, are subjected to tensile strain (stretching) when the skull and the brain move differentially when subjected to an angular acceleration.").

Since the dura is comparatively firmly attached to the skull, the motion of the dura relative to the skull will usually be negligible. Hence the main motion must be the sliding of the pia relative to the arachnoid and of the arachnoid relative to the dura—the latter being presumably greater. In this process the vessels which drain the cortical veins into the venous sinuses will be stretched, and may break anywhere along their length, causing subdural or subarachnoid hemorrhages.

A.H.S. Holbourn, *Mechanics of Head Injuries*, 2 LANCET 438, 440 (1943).

145. See *supra* note 143 and accompanying text.

146. See Duhaime et al., *Head Injury in Very Young Children*, *supra* note 92, at 183 (young children's skulls are more malleable than adults' skulls).

3. "Shaken Infant Syndrome" and "Shake-Impact Syndrome"

Infants with intracranial injuries believed to be attributable to abuse have been described as having "shaken infant syndrome," or, more recently, "shake-impact syndrome." Use of the terms is confusing and potentially misleading. The term "shaken infant syndrome" can easily be misunderstood, because it implies a single cause (shaking) for injuries that may be attributable to other forces (e.g., striking or throwing the child, strangulation, or smothering), or to a combination of shaking and other forces.¹⁴⁷ Although more recent abuse literature has recognized the potential importance of impact in the evolution of these injuries, and has adopted the term "shake-impact syndrome," the original term continues to be used widely.¹⁴⁸

Researchers disagree whether young children can die from shaking alone, without some sort of impact to the head.¹⁴⁹ Up to 50% of fatally abused infants exhibit no signs of blunt trauma,¹⁵⁰ leading some to conclude that shaking alone must be responsible. However, it is possible for a child to be thrown against an object that would diffuse the force of impact, such as a mattress, yet the throw still results in severe brain injury.¹⁵¹ In the child who survives abuse, signs of blunt trauma can easily be missed, because many of those signs are only detectable at the time of

147. See, e.g., Bruce & Zimmerman, *supra* note 138, at 492, 494; Caffey, *supra* note 40; Gilles, *Abusive Head Injury in Children*, *supra* note 107, at 359. The term "shaken infant syndrome" had its origins in the work of Caffey, a radiologist, who noted long bone injuries and subdural hematoma in a group of infants, and conjectured that the mode of injury was one of violent to and fro shaking. What Caffey's hypothesis failed to take into account was the work of Ambroise Tardieu, Weston, and others, who were examining these cases from a clinical and postmortem perspective, which clearly indicated that a large number of these young children sustained impact as part of their injuries. This impact was documented to be both impact of the head against an object (such as a wall), and objects against the head (such as hands).

148. Even the term "shake-impact syndrome" neglects the importance of other contributing factors, such as associated strangulation, smothering, multiple events on the same day, or clinical issues such as concurrent dehydration.

149. Compare Duhaime et al., *The Shaken Baby Syndrome*, *supra* note 142, at 411 (of 13 abused infants who died, all had evidence of blunt head trauma at autopsy) with Donald L. Budenz et al., *Ocular and Optic Nerve Hemorrhages in Abused Infants with Intracranial Injuries*, 101 *OPHTHALMOLOGY* 559, 560 (1994) (of 13 abused infants who died, 4 showed no evidence of blunt head trauma at autopsy; "pure shaking injury may indeed lead to fatal intracranial injury").

150. See Edward C. Benzel & Theresa A. Hadden, *Neurologic Manifestations of Child Abuse*, 82 *S. MED. J.* 1347, 1349 (1989) (of 12 abused children with intracranial hemorrhages, 6 did not show any evidence of external head trauma); Duhaime et al., *The Shaken Baby Syndrome*, *supra* note 142, at 410 (of 48 abused infants who were shaken, 25% had "no findings of associated blunt trauma to the head and no extra-cranial trauma"); Mark N. Hadley et al., *The Infant Whiplash-Shake Injury Syndrome: A Clinical and Pathological Study*, 24 *NEUROSURGERY* 536 (1989) (of 36 abused infants with nonaccidental head injuries, 13 had no evidence of external trauma and no skull fractures).

151. See Gilles, *Abusive Head Injury in Children*, *supra* note 107, at 363 ("Even if no signs of impact are found at necropsy, impact cannot be excluded. The head may have been impacted against a soft object such as a pillow covering a hard surface, diffusing the transnational force without damaging extra-cranial or cranial structures."); Spivack, *supra* note 45, at 76 ("Impact against a yielding surface will not alter the force delivered to the intracranial structures but will decrease the damage done to superficial soft tissues, just as a boxer may suffer a knockout from a blow delivered by a gloved hand, without any apparent facial bruise.").

autopsy.¹⁵² Lacking evidence of blunt trauma therefore does not mean that blunt trauma did not occur. Other mechanisms of injury to be considered include smothering and associated strangulation.¹⁵³

4. Distinguishing Between Abusive and Unintentional Injury

Intracranial injuries may be due to a number of causes other than abuse, including birth trauma, accidental injury, bleeding disorders (coagulopathies), blood vessel abnormalities (aneurysms and arteriovenous malformations), and infections.¹⁵⁴ Clinically significant intracranial injuries due to birth trauma or unintentional injury are not common.¹⁵⁵ "Clinically significant" refers to injuries that are associated with significant neurologic symptoms. Birth trauma can usually be excluded by the dating of the injuries through an examination of the clinical course and through imaging;¹⁵⁶ imaging techniques are discussed below. Unintentional trauma is an unlikely cause of severe head injury without a history of a witnessed accident involving significant applied forces, such as a high fall (greater than ten feet) or a high speed motor vehicle accident.¹⁵⁷

A general word of caution is in order, however. One should be careful not to over interpret research suggesting that falls of less than ten feet rarely if ever lead to intracranial injury, because much of the research examining the effects of falls in childhood did not routinely include brain imaging of all children who had fallen. Children who did not exhibit clinical signs of intracranial injury were not imaged,

152. See Duhaime et al., *The Shaken Baby Syndrome*, *supra* note 142, at 410-11 (although 13 of 13 infants who died suffered from blunt trauma, for 7 of the 13, that evidence "had not been apparent prior to death"); Elner et al., *supra* note 139, at 1100 (7 infants who died due to abusive head injury; "signs of direct head trauma may not be readily identified in all cases. These signs are often subtle and are frequently recognized only at autopsy in the form of subcutaneous hematomas, skull fractures, and skin bruises.").

153. See Roger Bird et al., *Strangulation in Child Abuse: CT Diagnosis?*, 163 *RADIOLOGY* 373 (1987); Gilles, *Abusive Head Injury in Children*, *supra* note 107, at 362; Elizabeth E. Gilles, *Infant Syndromes in Abusive Head Injury*, 34 *ANNALS NEUROLOGY* 475 (1993).

154. See Giangiacomo et al., *supra* note 137, at 297 ("The combination of intracranial and intraocular hemorrhage occurs in numerous disorders including head trauma, cerebrovascular accidents, coagulopathies, embolic disorders, vasculitides, hypertension, diabetes, strangulation, and the whiplash-shaken infant syndrome."); Gilles, *Abusive Head Injury in Children*, *supra* note 107, at 349, 349-50 ("Epidural and subdural hematomas, subarachnoid hemorrhage and intracerebral hemorrhage may all be a consequence of the birth process by a variety of mechanisms."); Merten & Carpenter, *supra* note 142, at 831 ("No specific diagnostic patterns exist for inflicted craniocerebral trauma, and accidental head trauma may produce any or all cranial and intracranial lesions found in abused infants and children.").

155. See Gilles, *Abusive Head Injury in Children*, *supra* note 107, at 349 ("Most infants do not sustain significant injury as a result of the birth process.").

156. See Israel F. Abrams & Beth A. Rosen, *Neurologic Trauma in Newborn Infants*, 13 *SEMINARS NEUROLOGY* 100 (1991); Gail Stewart et al., *Trauma in Infants Less than Three Months of Age*, 9 *PEDIATRIC EMERGENCY CARE* 199 (1993).

157. The research discussing the relation between falls and serious injury is discussed *supra* notes 103-09 and accompanying text. The relation between falls and specific types of intracranial injury is discussed in the parts specifically discussing those injuries.

making it possible that short falls may have resulted in intracranial injury that was simply never detected.¹⁵⁸ For that reason, it is appropriate to state that the research supports the conclusion that *clinically* significant intracranial injury is not likely to occur after short falls.¹⁵⁹

Bleeding disorders are also known as blood dyscrasias, hemorrhagic diatheses or tendencies, and coagulopathies. They include various types of hemophilia and thrombocytopenia (a deficiency of platelets in the blood, which aid in clotting). Subarachnoid hemorrhages, subdural hematoma, intracerebral hemorrhages, and retinal hemorrhages have all been reported in patients with coagulopathies.¹⁶⁰ A child with a bleeding disorder will often (but not always) have a history of excessive bleeding and of easy bruisability.¹⁶¹ A standard battery of blood tests rules out most bleeding disorders.¹⁶²

If abnormalities of blood vessels such as aneurysms and arteriovenous malformations rupture, subarachnoid and intracerebral hemorrhages may develop.¹⁶³ More rarely, their rupture may result in a subdural hematoma.¹⁶⁴ Blood vessel

158. See David Doezeema et al., *Magnetic Resonance Imaging in Minor Head Injury*, 20 ANNALS EMERGENCY MED. 1281 (1991). This article reviews 58 patients with an age range of three years to 66 years (mean age 24 years) who sustained mild head injury. Six out of 58 had either small subdural hematomas or small contusions identified by MRI screening. *Id.* at 1281; see also A. Jenkins et al., *Brain Lesions Detected by Magnetic Resonance Imaging in Mild and Severe Head Injuries*, 2 LANCET 445 (1986). Jenkins evaluated 50 patients with a range of head trauma severity. While they do not define the age of these patients, 6/8 patients with full level consciousness at the time of admission had cortical lesions present on MRI.

159. An unresolved issue is whether young children's bridging veins are susceptible to greater stress such that the application of even minor force (such as a short fall) might cause intracranial injury.

160. See Gordon L. Bray & Naomi Luban, *Hemophilia Presenting with Intracranial Hemorrhage: An Approach to the Infant with Intracranial Bleeding and Coagulopathy*, 141 AM. J. DISEASES CHILDREN 1215 (1987); Ann Dietrich et al., *Head Trauma in Children with Congenital Coagulation Disorders*, 29 J. PEDIATRIC SURGERY 28 (1994); George H. Rodman, Jr., *Bleeding and Clotting Disorders*, in TEXTBOOK OF CRITICAL CARE 722, 726 (William C. Shoemaker et al. eds., 1984) (hemophilia can lead to spontaneous hemorrhage in the cranium); see also Tetsuo Matsuzaka et al., *Incidence and Causes of Intracranial Hemorrhage in Infancy: A Prospective Surveillance Study after Vitamin K Prophylaxis*, 11 BRAIN & DEV. 384 (1989) (late hemorrhagic disease can lead to subdural hematomas and retinal bleeding).

161. See Rodman, *supra* note 160, at 725 (history of excessive bleeding or multiple bruises suggests bleeding disorder).

162. See *id.* (battery includes tests for bleeding time, platelet count, peripheral blood smear, prothrombin time, and partial thromboplastin time).

163. Julio H. Garcia & Michael L. Anderson, *Circulatory Disorders and Their Effects on the Brain*, in TEXTBOOK OF NEUROPATHOLOGY 621, 622 (Richard L. Davis & David M. Robertson eds., 2d ed. 1991); Nakamasa Hayaski et al., *Intracranial Hemorrhage Due to Rupture of an Arteriovenous Malformation in a Full-Term Neonate*, 10 CHILD'S NERVOUS SYS. 344 (1994) [hereinafter Hayaski et al., *Intracranial Hemorrhage*]; K. Kuchelmeister et al., *A Probably Familiar Saccular Aneurysm of the Anterior Communicating Artery in a Neonate*, 9 CHILD'S NERVOUS SYS. 302 (1993).

164. N. J. McLellan et al., *Spontaneous Subhyaloid and Retinal Haemorrhages in an Infant*, 61 ARCHIVES DISEASE CHILDHOOD 1130, 1132 (1986).

Any intracranial haemorrhage may leak into the subarachnoid space and a subdural haematoma can develop if an aneurysm or arteriovenous malformation ruptures through the arachnoid mater. Such a finding has been described in a 3-month-old child with an intracerebral aneurysm. Arteriovenous malformations may also occur within the meninges and so bleed directly into the subdural space.

abnormalities are a possible but exceedingly rare cause of intracranial bleeding in young children. Three large series reporting on over 10,000 patients with subarachnoid hemorrhages (SAHs) identified no children one year or younger.¹⁶⁵ In these rare instances, arteriovenous malformation is a more common cause of intracranial bleeding than aneurysm.¹⁶⁶ Bleeding in these cases is more likely to be parenchymal (within the substance of the brain) or into the subarachnoid space, a different pattern of injury than that of nonaccidental head injury. Arteriovenous malformations and aneurysms can be detected by CT scan with contrast enhancement, magnetic resonance angiography, or cerebral angiography.¹⁶⁷

Infections, such as meningitis and encephalitis, can lead to intracranial pathology, including subdural fluid collections and brain infarction.¹⁶⁸ The definitive diagnosis of meningitis is made by examination of the cerebrospinal fluid obtained with a lumbar puncture.¹⁶⁹ Markers of encephalitis are also sometimes seen in the

Id.

165. R. W. Newton, *Intracranial Haemorrhage and Non-Accidental Injury*, 64 ARCHIVES DISEASE CHILDHOOD 188, 188 (1989).

166. Although there are case reports of aneurysms occurring in young children, *see, e.g.*, Edgardo A. Crisostomo et al., *Features of Intracranial Aneurysms in Infants and Report of a Case*, 28 DEV. MED. & CHILD NEUROLOGY 62 (1986) (11-month-old child); Hayaski et al., *Intracranial Hemorrhage*, *supra* note 163; Fariss D. Kimbell et al., *Surgical Treatment of Ruptured Aneurysm with Intracerebral and Subarachnoid Hemorrhage in a 16-Month-Old Infant*, 17 J. NEUROSURGERY 331 (1960) (16-month-old child); Kuchelmeister, *supra* note 163, at 305 (1993); McLellan et al., *supra* note 164 (six-week-old child); A. Nishio et al., *Anterior Communicating Artery Aneurysm in Early Childhood: Report of a Case*, 35 SURGICAL NEUROLOGY 224, 224 (1991) (13-month-old child); P.M. Vapalahti et al., *Intracranial Arterial Aneurysm in a Three-Month-Old Infant*, 30 J. NEUROSURGERY 169 (1969) (3-month-old child), large-scale surveys of patients with aneurysms find that the vast majority occur in adults, probably because of the role that hypertension and arteriosclerosis play in their etiology, *see id.* at 169 (reviewing studies; 1% in children); *see also* Donald D. Matson, *Intracranial Arterial Aneurysms in Childhood*, 23 J. NEUROSURGERY 578, 579 (1965) (citing a study which found 2 of 1125 verified aneurysm occurred in children under five years of age). *But see* Nishio et al., *supra*, at 224 (notes surveys showing small percentage of aneurysms in infants, but "with the increasing use of cerebral angiography and computed tomography (CT) intracranial aneurysms have been discovered more often in recent years, even in neonates with subarachnoid or intracranial hemorrhage").

167. Gilles, *Abusive Head Injury in Children*, *supra* note 107; David M. Klein, *Central Nervous System Injuries*, in CHILD ABUSE AND NEGLECT 86 (Norman S. Ellerstein ed., 1981); *see* Crisostomo et al., *supra* note 166 (11-month-old is presented with seizures and retinal hemorrhages; CT revealed subarachnoid bleeding and a large intracerebral hematoma; angiography revealed an aneurysm); John J. Kelly et al., *Intracranial Arteriovenous Malformations in Childhood*, 3 ANNALS NEUROLOGY 338, 339 (1978) (37 patients under 20 with arteriovenous malformation, four under one year of age; 13/15 CT scans taken were abnormal, 12 revealing intracerebral hematoma; 34/34 angiograms abnormal); McLellan et al., *supra* note 164, at 1131 (six-week-old is presented with seizures, full fontanelle, and retinal hemorrhages; CT revealed a large intracerebral hematoma suggesting spontaneous bleeding; angiography revealed an aneurysm).

168. Gilles, *Abusive Head Injury in Children*, *supra* note 107, at 351; Newton, *supra* note 165, at 189.

169. Larry E. Davis, *Central Nervous System Infections*, in NEUROLOGY FOR THE NON-NEUROLOGIST 350 (William J. Wiener & Christopher G. Goetz eds., 3d ed. 1994).

The CSF exam is the key to the diagnosis of meningitis . . . cultures for bacteria take one to three days, for tuberculosis and fungi one to six weeks, and for viruses days to three weeks. Rapid diagnosis of bacteria can be made by Gram stain of CSF sediment and by testing CSF for common bacterial antigens. The Gram stain will detect bacteria in CSF sediment in over three-fourths of patients with acute bacterial meningitis. . . . Antigen tests have about the same sensitivity as the Gram stain.

cerebrospinal fluid,¹⁷⁰ and can sometimes be diagnosed by serologic tests.¹⁷¹ Viral meningitis and encephalitis are unlikely to result in subdural fluid collections.

Frequently, it is a combination of findings that suggests a diagnosis of abuse. For example, a child with multiple skull fractures, acute subdural hematoma, brain swelling, and retinal hemorrhages did not develop the subdural hematoma from a rare arteriovenous malformation. Furthermore, infants are presented with injuries to other parts of the body sometimes, including burns, bruises, and fractures. Some fracture patterns, such as associated rib fractures, or metaphyseal injuries of the long bones, may be the associated consequences of vigorous, violent shaking;¹⁷² other types of injury (e.g., burns) may simply suggest that the child has been subjected to different types of abuse. These types of injury help to rule out accident or disease as alternate causes of the intracranial pathology.¹⁷³ Conversely, the fewer markers of injury, the more difficult it is to distinguish abuse from unintentional injury.

C. Parenchymal Brain Injuries

Injuries to the brain are very common in shake-impact events. They can occur both directly (from applied forces such as violent movement of the head) or indirectly (from swelling and ischemia). Brain injuries due to shearing include diffuse axonal injury, white matter tears, and other injuries such as corpus callosal tears.¹⁷⁴ Diffuse axonal injury refers to the microscopically detectable injury of brain and brainstem axons from stretching and disruption of nerve fiber tracts following sudden forceful rotation of the head.¹⁷⁵ White matter tears are tears in the white matter of the brain, and also the result of sudden and severe rotational forces.¹⁷⁶ Consequences of the initial injury include brain swelling and brain infarction, with

Id.

170. See *id.* at 353-54 (noting changes in CSF white blood cell count and protein levels).

171. See *id.* at 354 ("The diagnosis of viral encephalitis is usually made by serologic tests.").

172. See Yoon S. Hahn et al., *Traumatic Mechanisms of Head Injury in Child Abuse*, 10 CHILD'S BRAIN 229, 238 (1983) (shaken babies often present with metaphyseal fractures).

The pathogenesis of long bone changes, metaphyseal avulsion and subperiosteal hemorrhages was thought to be caused by traction-stretching stresses on the periosteum, induced by grabbing the infants by the extremities or by the thorax, and shaking them, which in turn resulted in the whiplashing of the head onto the thorax.

Id.

173. See Brown & Minns, *supra* note 89 (combination of rib fractures and intracranial injuries is a reliable pointer to abuse); Gilles, *Abusive Head Injury in Children*, *supra* note 107, at 363 (external trauma helps to rule out disease as alternate cause of intracranial injury).

174. Corpus callosal tears are tears of the corpus collosum, which connects the two hemispheres of the brain.

175. Holbourn, *supra* note 144; Sabina J. Strich, *Shearing of Nerve Fibers as a Cause of Brain Damage Due to Head Injury*, 1 LANCET 443 (1961); see John M. Hardman, *Cerebrospinal Trauma*, in TEXTBOOK OF NEUROPATHOLOGY, *supra* note 163, at 969 (diffuse axonal injury due to sudden angular rotation of the head).

176. Brown & Minns, *supra* note 89; Thomas A. Gennarelli, *Cerebral Concussion and Diffuse Brain Injuries*, in HEAD INJURY 137, 145 (Paul R. Cooper ed., 3d ed. 1993).

secondary necrosis (tissue death).¹⁷⁷ Typically, severe abusive head injury leads to immediate unconsciousness and apnea (cessation of breathing).¹⁷⁸ It is the brain injury that causes the infant's unconsciousness, limpness, and seizures.¹⁷⁹

White matter tears are found most often at the juncture of the white and gray matter, which differ in density, and are therefore most susceptible to shear strain.¹⁸⁰ They are one to three centimeters in length, and tend to be in the frontal and temporal lobes, although they may be seen throughout the brain. They are found most typically in an infant less than six to eight months of age.¹⁸¹

White matter tears are sometimes referred to as gliding contusions, or as white matter contusional tears, which is somewhat confusing, as these injuries are different from the contusions found in older children and adults after blunt force trauma.¹⁸² In the mature brain, parenchymal contusions typically occur under the site of cranial impact (coup) or on the side opposite to the impact (contre-coup).¹⁸³ They are typically found at the tips of the frontal and temporal lobes, and probably relate to movement of the brain over the adult's rough inner skull table. Infants are less likely than adults to sustain coup and contre-coup contusions.¹⁸⁴ The infant's skull is more compliant, and has a smoother inner table.¹⁸⁵ On the other hand, infants are more

177. Cohen et al., *supra* note 137, at 101.

178. Gilles, *Abusive Head Injury in Children*, *supra* note 107, at 351.

179. Although a great deal of attention is paid to the presence of even small subdural hematomas, those cause brain symptoms only in their mass effects on the underlying brain or in the development of ipsilateral (same side) brain swelling.

180. Strich, *supra* note 175; Robert A. Zimmerman et al., *Computed Tomography of Shearing Injuries of the Cerebral White Matter*, 127 *RADIOLOGY* 393, 395 (1978) [hereinafter Zimmerman et al., *Computed Tomography of Shearing Injuries*]; see Brown & Minns, *supra* note 89, at 859 (grey and white matter are of different densities, so move at different velocities when rotation occurs); T. Jaspan et al., *Cerebral Contusional Tears as a Marker of Child Abuse—Detection by Cranial Sonography*, 22 *PEDIATRIC RADIOLOGY* 237, 244 (1992) ("Due to the different physical properties of the grey [sic] matter and the semi-liquid underlying white matter, gliding of the relatively fixed gray matter over the more gelatinous white matter leads to shear strains at their interface."); Kleinman, *Skeletal Trauma*, *supra* note 36, at 185 (different densities of gray and white matter leads to shear strains at their interface).

181. See Jaspan et al., *supra* note 180 (white matter tears in six abused infants; all under six months of age); see also sources cited *infra* note 184.

182. Benzel & Hadden, *supra* note 150, at 1351.

183. Brown & Minns, *supra* note 89, at 857.

184. See Ian M. Calder et al., *Primary Brain Trauma in Non-Accidental Injury*, 37 *J. CLINICAL PATHOLOGY* 1095 (1984) (autopsies of 12 infants fatally abused; none of 12 exhibited contusions, seven of nine infants under five months exhibited contusional tears of the white matter); Richard Lindenberg & Ella Freytag, *Morphology of Brain Lesions From Blunt Trauma in Early Infancy*, 87 *ARCHIVES PATHOLOGY* 298, 298-305 (1969) (16 infants injured at five months of age or younger with white matter tears; review of 52 infants up to two years of age showed that children injured before five months of age showed tears, whereas older children showed contusions); Vowles et al., *supra* note 141 (autopsies of 10 infants under five months old fatally abused; of eight for whom information was available, six had a diffuse axonal injury).

185. Mary S. Case, *Head Injury in Child Abuse*, in *CHILD MALTREATMENT: A CLINICAL GUIDE AND REFERENCE*, *supra* note 1, at 75, 81; Calder et al., *supra* note 184, at 1099-1100; Cyril B. Courville, *Contrecoup Injuries of the Brain in Infancy*, 90 *ARCHIVES SURGERY* 157, 160 (1965). When contusions do occur in young children, they frequently accompany skull fracture. See Case, *supra*, at 81.

likely than adults to sustain white matter tears and diffuse axonal injuries. Their brains are less dense and incompletely myelinated.¹⁸⁶

The presence of white matter tears or other contusions indicate that trauma occurred.¹⁸⁷ In the absence of a history of major trauma—such as a high speed motor vehicle accident—they are highly suggestive of shake-impact abuse.¹⁸⁸ Falls of less than four feet have not been found to lead to such injuries,¹⁸⁹ and only rarely following falls down stairs;¹⁹⁰ contusions and brain lacerations (typically accompanied by skull fracture) have been reported in falls greater than ten feet.¹⁹¹

Contusions and other parenchymal injuries in abused children frequently occur with subdural hematomas, SAH, and skull fractures,¹⁹² which contributes to a diagnosis of abuse. As white matter tears heal and brain swelling resolves, the tears may appear as slit-like or oval holes in the brain substance. This appearance may assist in confirming a diagnosis of abuse after the initial presentation.

When white matter tears are filled with fresh blood, they are often mistaken for parenchymal bleeds, which are actually quite rare in abusive or accidental head injury. When a primary parenchymal bleed is observed, other diagnoses, such as ruptured arteriovenous malformation, aneurysm, or blood dyscrasia, are far more likely.¹⁹³

186. See Lindenberg & Freytag, *supra* note 184, at 303 (greater frequency of white matter tears among younger infants attributed to "the soft, gelatin-like consistency of the poorly myelinated cerebrum of the young infant and the pliancy of the skull" which leads to greater deformation of the skull and brain when trauma occurs); Vowles et al., *supra* note 141, at 188 ("In children less than five months of age myelination is at an early stage, and it may be that the axons are less cushioned and more vulnerable to injury than those of adults.").

187. See Hardman, *supra* note 175, at 965 ("A cerebral contusion is distinctive, if not pathognomonic, of mechanical injury.").

188. See Ommaya & Gennarelli, *supra* note 143, at 244 (the presence of white matter tears "should alert clinicians to the very strong possibility of child abuse in a young infant").

189. See Duhaime et al., *Head Injury in Very Young Children*, *supra* note 92 (76 children with accidental head injuries, 24 months or younger; no contusions among 34 children falling less than four feet, four contusions among 39 children falling more than four feet, either down stairs or down stairs in a walker); Williams, *supra* note 99, at 1351 (50 witnessed falls of 10 feet or less in children under three years of age; no contusions).

190. See Chiavello et al., *supra* note 95 (69 children under five years of age with stairway-related injuries; two with contusions, one of whom also suffered skull fracture and subdural hematoma after falling while being carried); Joffe & Ludwig, *supra* note 56, at 458 (363 children falling down stairs; no intracranial contusions). *But see* Chiavello et al., *supra*, at 680 (suggesting that Joffe and Ludwig may have underestimated the number of children suffering from intracranial injuries because of a failure to obtain CT scans in many patients).

191. See Barlow et al., *supra* note 115, at 510 (61 children up to 15 years of age falling 10 feet or more; 13 brain contusions, 11 of whom fell four stories or more); R.L. Sieben et al., *Falls as Childhood Accidents: An Increasing Urban Risk*, 47 PEDIATRICS 886 (1971) (55 falls by children up to 14 years of age; five brain contusions, one brain laceration).

192. See Kleinman, *Skeletal Trauma*, *supra* note 36, at 159, 193 (contusions often accompanied by subdural hematomas); *id.* at 188 ("Cortical contusion or white matter laceration extending to the surface of the brain will be associated with subarachnoid bleeding."); Lindenberg & Freytag, *supra* note 184, at 298 (contusional tears in 16 infants suffering blunt trauma to head; 12 had subdural hematomas, 16 had subarachnoid hemorrhage, and seven had skull fractures).

193. See Garcia & Anderson, *supra* note 163, at 695 (intracerebral hemorrhage may be due to leukemia, sickle cell anemia, bacterial endocarditis, and saccular aneurysms; also mentions effects of methamphetamine, phenylpropanolamine, cocaine, phencyclidine, and tumors).

Post-traumatic cerebral infarction (stroke) is a well recognized complication of head injuries in the adult population.¹⁹⁴ Although known to occur as part of the spectrum of injury in nonaccidental injuries to infants and young children, post-traumatic cerebral infarction has received relatively little attention in this context.

D. Retinal Hemorrhages and Other Ocular Injuries

A variety of injuries to the eye and its associated cranial nerve (the optic nerve) have been reported following abusive injuries. Among these are retinal hemorrhages, optic nerve sheath hemorrhages, retinoschisis, retinal folds, and retinal detachment. This discussion will focus on the two most common findings, retinal hemorrhages and optic nerve sheath hemorrhages.

The retina lines the inner surface of the globe of the eye. It consists of a number of layers of nerve cells, supportive cells, and their connections. Between the layers of the retina, there is an extensive network of capillaries (tiny blood vessels). The axons of the optic nerve, the central retinal artery, and the central retinal vein enter and exit through the optic nerve, located at the back of the globe. Retinal hemorrhages are hemorrhages occurring between these cell layers within the retina.¹⁹⁵

The retina is visualized with the use of direct ophthalmoscopy (with an ophthalmoscope) and indirect ophthalmoscopy (with the examiner wearing a binocular scope and using a free lens to magnify). Documentation that the pupil was dilated with mydriatic agents is very helpful, as retinal hemorrhages and retinal folds may be missed with an undilated examination alone.

Retinal hemorrhages in young infants and children, especially when found with other markers of injury, are highly suggestive of abuse.¹⁹⁶ Retinal hemorrhages are commonly associated with shake-impact events, and only rarely occur following

194. Stuart E. Mirvis et al., *Posttraumatic Cerebral Infarction Diagnosed by CT: Prevalence, Origin, and Outcome*, 11 AJNR 355 (1996) (an incident of 1.9% of post-traumatic cerebral infarction in a population of 1332 patients (mean age 38.1) admitted with significant craniocerebral trauma); see Bird et al., *supra* note 153; Joseph P. Evans & I. Mark Scheinker, *Histologic Studies of the Brain Following Head Trauma III. Post Traumatic Infarction of the Cerebral Arteries, with Consideration of the Associated Clinical Picture*, 50 ARCHIVES NEUROLOGY & PSYCHIATRY 258 (1943); Gilles, *Abusive Head Injury in Children*, *supra* note 107; Charles M. Glasier et al., *Cerebral Infarction in Child Abuse: Diagnosis by Technetium-99m Methylene Diphosphonate Skeletal Scintigraphy*, 12 CLINICAL NUCLEAR MED. 807 (1987). The role of this type of injury in clinical course and outcome is still being investigated. See Elizabeth E. Gilles & Marvin D. Nelson, Jr., *Cerebral Complications of Nonaccidental Head Injury in Childhood* (unpublished manuscript, on file with author).

195. Hemorrhages under the innermost layer (the inner limiting membrane) next to the inside of the globe are often called sublaminal hemorrhages, whereas hemorrhages in which blood extends into the jelly, or vitreous, within the globe are called vitreous hemorrhages. To date, there is no correlation between the type and extent of retinal hemorrhages and their pathogenesis.

196. See Duhaime et al., *Head Injury in Very Young Children*, *supra* note 92, at 184 (retinal hemorrhages are "highly associated" with abuse).

accidental injuries.¹⁹⁷ Although some authors have gone so far as to suggest that they are pathognomonic (definitely diagnostic) for abuse,¹⁹⁸ this is an overstatement, because many other conditions, including the birth process, have been associated with retinal hemorrhages. These alternate causes are discussed below.

Retinoschisis refers to the splitting of retinal nerve fibers; retinal folds to the folding of the retina; retinal detachment to the tearing and separation of the retina from its epithelium (one of the outer layers close to the vitreous). All are possible consequences of abuse.¹⁹⁹ The discussion focuses on the two most common findings, retinal hemorrhages, and optic nerve sheath hemorrhages.

197. See M. Elaine Billmire & Patricia A. Myers, *Serious Head Injury in Infants: Accident or Abuse?*, 75 PEDIATRICS 340, 341 (1985) (84 children under one year with head injury and/or abnormal CT findings, 28 abused and 54 nonabused; "89% of the shaken infants had retinal hemorrhages. . . . Retinal hemorrhages were not seen in any accidentally injured infants."); Budenz et al., *supra* note 149, at 560 (autopsies of 19 infants, 13 abused and 6 sudden infant death syndrome; 11 of abused and none of nonabused had retinal hemorrhages); Yvonne M. Buys et al., *Retinal Findings After Head Trauma in Infants and Young Children*, 99 OPHTHALMOLOGY 1718, 1722 (1992) (78 children 36 months or younger with head injury; 3 abused and 75 nonabused; none of accidentally injured children and all 3 abused children had retinal hemorrhages); Duhaime et al., *Head Injury in Very Young Children*, *supra* note 92, at 182 (100 children 24 months or younger with head trauma, 24 abused and 76 nonabused; 9 or 38% of abused children had retinal hemorrhages; 1 nonabused child with retinal hemorrhages involved in a fatal auto accident); Arthur B. Eisenbrey, *Retinal Hemorrhage in the Battered Child*, 5 CHILD'S BRAIN 40, 42 (1979) (50 children, 26 abused and 24 nonabused; 16 or 62% of abused children had retinal hemorrhages, 1 nonabused child with retinal hemorrhages was a newborn suffering a traumatic delivery); J.E. Elder et al., *Retinal Hemorrhage in Accidental Head Trauma in Childhood*, 27 PEDIATRIC CHILD HEALTH 286, 287 (1991) (25 children with head trauma, average age 5.6 years; none had retinal hemorrhages); Elner et al., *supra* note 139, at 1098 (autopsies of 10 abused children; 7 had retinal hemorrhages); Brahm Goldstein et al., *Inflicted Versus Accidental Head Injury in Critically Injured Children*, 21 CRITICAL CARE MED. 1328, 1330 (1993) (40 children with head injury, 14 abused and 26 nonabused; 8 or 57% of abused children had retinal hemorrhages, none in nonabused children); Hahn et al., *supra* note 172, at 232 (77 children with head injuries due to abuse, 85% under two years of age; 26 or 34% had retinal hemorrhages); Dennis L. Johnson et al., *Accidental Head Trauma and Retinal Hemorrhage*, 33 NEUROSURGERY 231 (1993) (140 children with head injury, median age 4.5 years; 2 children had retinal hemorrhages, both of them attributed to auto accidents); Ralph S. Riffenburgh & Lakshmanan Sathyavagiswaran, *Ocular Findings at Autopsy of Child Abuse Victims*, 98 OPHTHALMOLOGY 1519, 1521, 1523 (1991) (autopsies of 190 children; 55 definitely abused, 43 suspected abused, and 92 nonabused; retinal hemorrhages in 27 or 49% of definitely abused, 24 or 56% of suspected abused, and 4 or 4% of nonabused, most of whom died of sudden infant death syndrome).

198. See Bruce & Zimmerman, *supra* note 138, at 484 ("The presence of retinal hemorrhages in the absence of a history of severe trauma is diagnostic of some type of shaken impact injury."); Duhaime et al., *Head Injury in Very Young Children*, *supra* note 92, at 184 (retinal hemorrhages are "highly associated" with abuse); Eisenbrey, *supra* note 197, at 42 ("We believe that retinal hemorrhage in children under 3 with or without other evidence of injury is pathognomonic of the battered child syndrome.").

199. See Elner et al., *supra* note 139, at 1098 (10 fatally abused children, 7 had eye damage: 7 had vitreous hemorrhages, 4 had retinoschisis, and 3 had retinal folds); J. Allen Gammon, *Ophthalmic Manifestations of Child Abuse*, in CHILD ABUSE & NEGLECT, *supra* note 167, at 121, 124 (retinal detachment possible consequence of child abuse); Michael W. Gaynon et al., *Retinal Folds in the Shaken Baby Syndrome*, 106 AM. J. OPHTHALMOLOGY 423 (1988) (2 shaken infants with retinal folds); Mark J. Greenwald et al., *Traumatic Retinoschisis in Battered Babies*, 93 OPHTHALMOLOGY 618 (1986) (5 abused infants; 3 had vitreous hemorrhages; and other findings suggesting retinoschisis); Stephen J. Massicotte et al., *Vitreoretinal Traction and Perimacular Retinal Folds in the Eyes of Deliberately Traumatized Children*, 98 OPHTHALMOLOGY 1124 (1991) (3 fatally abused infants with retinal folds).

The optic nerve, unlike most of the other cranial nerves, communicates directly with the cerebrum. It is covered with meninges throughout its extent from the globe to the brain. Subdural and subarachnoid bleeding most likely occurs within the optic nerve sheath from rupture of dural or bridging vessels, transmission of intracranial pressure to within the optic nerve sheath, either by extension from bleeding elsewhere or because of injury to bridging vessels along the nerve.²⁰⁰ Nonaccidental injury is not the only etiology of optic nerve sheath hemorrhages.²⁰¹ However, if these alternate causes are not found at necropsy, and if other markers of injury are present, optic nerve sheath hemorrhages contribute to a diagnosis of abuse.²⁰² Optic nerve sheath hemorrhages cannot be identified accurately in the living child.

Although abused infants with retinal hemorrhages often have associated intracranial pathology such as subdural hematoma, this is not always the case.²⁰³ Researchers have reported cases in which CT findings of intracranial injury have lagged behind the clinical detection of retinal hemorrhages, although retinal hemorrhages appear subsequent to other CT findings in the typical case.²⁰⁴

A number of theories have been proposed to explain the development of retinal hemorrhages and other ocular injuries in shake-impact events. One theory with a

200. Paul J. Muller & John H. N. Deck, *Intraocular and Optic Nerve Sheath Hemorrhage in Cases of Sudden Intracranial Hypertension*, 41 J. NEUROSURGERY 160 (1974).

201. As with retinal hemorrhages, optic nerve sheath hemorrhages may develop as a secondary consequence of disorders which result in sudden or prolonged increase in intracranial pressure. Muller & Deck, *supra* note 200. These include nonaccidental and accidental head trauma, as well as causes of extensive subarachnoid hemorrhage, such as ruptured aneurysms and arteriovenous malformations. Elner, *supra* note 139; Crain E. Munger et al., *Ocular and Associated Neuropathologic Observations in Suspected Whiplash Shaken Infant Syndrome*, 14 AM. J. FORENSIC MED. & PATHOLOGY 193 (1993).

202. See Budenz et al., *supra* note 149, at 559 (13 fatally abused infants with intracranial injury; all had optic nerve sheath hemorrhages); Elner et al., *supra* note 139, at 1098 (10 fatally abused children, seven with eye damage; seven had subdural optic nerve hemorrhages).

203. Brown & Minns, *supra* note 89, at 860; Elder et al., *supra* note 197, at 288 (quoting Levin et al. 1989 case study); Ludwig & Warman, *supra* note 136, at 106 (quoting R.R. Ober, *Hemorrhagic Retinopathy in Infancy: A Clinicopathologic Report*, 17 J. PEDIATRIC OPHTHALMOLOGY STRABISMUS 17 (1980)); Lawrence G. Tomasi & N. Paul Rosman, *Purtscher Retinopathy in the Battered Child Syndrome*, 129 AM. J. DISEASES CHILDREN 1335 (1975) (two cases).

204. Elner et al., *supra* note 139, at 1099 (six fatally abused children with retinal hemorrhages who received CT scans before death; in three cases, autopsies revealed subdural hematomas not visible on CT scans); Giangiacomo et al., *supra* note 137, at 298 (five shaken infants with retinal hemorrhages; two initially presented with normal CT scans, one with subtle abnormalities, all three eventually developed subdural hematomas). Giangiacomo et al., also suggests several reasons why retinal hemorrhages might be detected before subdural hematomas in some cases. First, "[b]ecause the bridging cerebral vessels are venous in origin and perfused at low pressure, the initial degree of subdural hemorrhage may be self-limited because of the elevated intracranial pressure." Second, "acute subdural hematoma in a few patients may be isodense with brain parenchyma by CT scan. Over the course of several days or weeks as cerebral edema declines, infantile subdural effusion enlarges and is no longer isodense with brain parenchyma, thus facilitating the CT recognition of this disorder." Third, in more than 80% of cases the subdural hematomas are bilateral in the shaken infant and thus, no midline shift may be evident. Thus, when subdural fluid first accumulates, the bilateral frontoparietal distribution masks the midline shift normally associated with subdural hematoma and may simulate the appearance of the normally increased subarachnoid space sometimes seen in infancy.

Id. at 298.

great deal of support attributes the development of retinal hemorrhages to increases in intracranial pressure.²⁰⁵ Intracranial pressure may increase temporarily as a direct result of impact, or in a more sustained fashion as a consequence of growing intracranial mass lesions, such as subdural hematomas and brain swelling.²⁰⁶ When pressure is communicated to the space behind the globe of the eye (the retrobulbar space) and the optic nerve, backflow of venous blood to the eye and retina may occur, because the retinal venous drainage system does not have valves.²⁰⁷ Other factors that may contribute to the formation of retinal hemorrhages include apnea (lack of breathing) after a significant injury with resultant hypoxia (lack of oxygen to the blood cells), ischemia related to low blood flow states in the brain, and compression of the central retinal vein due to optic nerve sheath subdural hemorrhage.²⁰⁸

One, as yet unproven, hypothesis is that shake-impact forces result in shearing injuries due to hydraulic forces within the vitreous of the globe, leading to retinal tearing.²⁰⁹ Retinal folds and retinal detachment might occur from this mechanism. Rotational forces, incurred during shaking, may be especially damaging in the infant, whose vitreous is more solid than in adults.²¹⁰ Although shearing forces among vitreous, lens, and retina have also been postulated as a cause of retinal hemorrhages, this mechanism would not explain the presence of small retinal hemorrhages between retinal layers.

As with other markers of injury, other possible etiologies associated with retinal hemorrhage development must be considered, including birth trauma, vascular malformation, unintentional trauma, blood disorders, hypertension, infections, and papilledema. Nonabusive causes of intracranial bleeding must be considered when retinal hemorrhages are presented with acute subdural hematomas, and especially extensive subarachnoid hemorrhages.²¹¹

205. James R. Keane, *Retinal Hemorrhages: Its Significance in 100 Patients with Acute Encephalopathy of Unknown Cause*, 36 ARCHIVES NEUROLOGY 691 (1979); Scott R. Lambert et al., *Optic Nerve Sheath and Retinal Hemorrhages Associated With the Shaken Baby Syndrome*, 104 ARCHIVES OPHTHALMOLOGY 1509 (1986); Muller & Deck, *supra* note 200.

206. Lambert et al., *supra* note 205, at 1512. Adults with subarachnoid hemorrhages and associated intracranial pressure frequently develop retinal hemorrhages. See Keane, *supra* note 205 (100 adults with retinal hemorrhages; 31 also had subarachnoid hemorrhages).

207. Giangiacomo et al., *supra* note 137, at 297; Keane, *supra* note 205, at 693 ("A sudden increase in intracranial pressure is transmitted to the distal optic nerve sheath where it causes a temporary obstruction of retinal venous outflowing. The subsequent rise in retinal venous pressure results in local venous-capillary bleeding.").

208. Ayub K. Ommaya et al., *Whiplash Injury and Brain Damage: An Experimental Study*, 204 JAMA 285 (1968); William T. Shults & K.C. Swan, *High Altitude Retinopathy in Mountain Climbers*, 93 ARCHIVES OPHTHALMOLOGY 404 (1975); Michael Wiedman & G. Tabin, *High-Altitude Retinal Hemorrhage as a Prognostic Indicator in Altitude Illness*, 26 OPHTHALMOLOGY CLINICS 175 (1986).

209. Greenwald et al., *supra* note 199, at 623.

210. Duhaime et al., *Head Injury in Very Young Children*, *supra* note 92, at 182; Greenwald et al., *supra* note 199, at 623; Massicotte et al., *supra* note 199, at 1125.

211. See Oscar A. Cruz & Joseph Giangiacomo, *Ophthalmic Manifestations of Child Abuse*, in CHILD MALTREATMENT: A CLINICAL GUIDE AND REFERENCE, *supra* note 1, at 67-68 (retinal hemorrhages can be caused by subdural or subarachnoid hemorrhages secondary to accidental trauma); Duhaime et al., *Head Injury in Very*

A significant number of newborns have retinal hemorrhages.²¹² These appear to be a consequence of pressure and molding effects, but exactly how they develop is unknown. Retinal hemorrhages are observed more often following traumatic births (e.g., with vacuum extraction), but are also found commonly following nontraumatic deliveries.²¹³ Most birth-related retinal hemorrhages are small,²¹⁴ and disappear within the first few days of life,²¹⁵ though many can be observed several weeks after delivery. In infants over one month old, causes other than the birth process must be considered if fresh retinal hemorrhages are detected.²¹⁶ Neonates with retinal hemorrhages related to the birth process will rarely have other intracranial injuries of clinical significance (such as subdural or subarachnoid hemorrhages, or an increase in intracranial pressure).²¹⁷ They are typically healthy with no neurological symptomatology.

Simple, short falls are exceedingly unlikely to result in retinal hemorrhages; even falls of more than ten feet only very rarely result in retinal hemorrhages.²¹⁸ Retinal

Young Children, *supra* note 92, at 183 (retinal hemorrhages can be caused by spontaneous subarachnoid hemorrhage); Eisenbrey, *supra* note 197, at 40 (retinal hemorrhages can be caused by hydrocephalus); Hardman, *supra* note 175, at 991 ("Retinal hemorrhages can also occur in response to increased intracranial pressure and not be related to traumatic injury."); Keane, *supra* note 205. Keane reviewed 100 adults with retinal hemorrhages of whom 94% had intracranial bleeding from a number of causes. The most common was aneurysmal rupture. He also reviewed previous studies of subarachnoid hemorrhage with associated retinal hemorrhage. Paul O'Brien et al., *Acute Subdural Hematomas of Arterial Origin*, 41 J. NEUROSURGERY 435 (1974); Henry A. Shenkin, *Acute Subdural Hematoma*, 57 J. NEUROSURGERY 254 (1982).

212. Cruz & Giangiacomo, *supra* note 211, at 68 (8%-50%); Gilles, *Abusive Head Injury in Children*, *supra* note 107, at 368 (11%-40%).

213. See Caffey, *supra* note 40, at 400; F. Sezen, *Retinal Hemorrhages in Newborn Infants*, 55 BRIT. J. OPHTHALMOLOGY 248, 250 (1970) (vacuum extraction increases likelihood of retinal hemorrhages; over 40% of infants delivered by vacuum extraction, less than 1% of infants born by caesarian section). *But see* J.D. Baum & C.J. Bulpitt, *Retinal and Conjunctival Hemorrhage in the Newborn*, 45 ARCHIVES DISEASE CHILDHOOD 344, 348 (1970) ("one must bear in mind the high incidence [of retinal hemorrhages] after entirely normal parturition"); Sezen, *supra*, at 250 (14.2% of children delivered spontaneously had retinal hemorrhages).

214. Baum & Bulpitt, *supra* note 213, at 248.

215. Gammon, *supra* note 199, at 130; Newton, *supra* note 165, at 188; *see, e.g.*, Baum & Bulpitt, *supra* note 213, at 346 (230 infants; 31% of those seen within 12 hours of birth had retinal hemorrhages; 15.5% when all infants seen within first week of life were included); Sezen, *supra* note 213, at 192 (1238 infants; 19% of those seen within 24 hours of birth had retinal hemorrhages; 2.6% of those seen three to five days after birth). Retinal hemorrhages due to abuse, on the other hand, may take much longer to clear. *See* Joseph Giangiacomo & Kenneth J. Barkett, *Ophthalmoscopic Findings in Occult Child Abuse*, 22 J. PEDIATRIC OPHTHALMOLOGY & STRABISMUS 234, 235-36 (1985) (two cases of abused infants with retinal and vitreous hemorrhages; took nine and 11 weeks to clear); Newton, *supra* note 165, at 188 (retinal hemorrhages in abused infants could last for several years).

216. Cruz & Giangiacomo, *supra* note 211, at 68; Dykes, *supra* note 138, at 212.

217. *See* Baum & Bulpitt, *supra* note 213, at 349 (230 newborns; "none of the infants with retinal hemorrhages had any evidence of associated intracranial bleeding"); I.O. Skälpe et al., *Cerebral Computed Tomography in Newborn Children With Large Retinal Hemorrhages*, 23 NEURORADIOLOGY 213, 214 (1982) (10 newborns with large retinal hemorrhages; none had intracranial hemorrhages).

218. *See* Buys et al., *supra* note 197, at 1720 (67 children under three suffering from falls and taken to emergency room; none had retinal hemorrhages, five fell ten feet or more, eight fell down more than 10 stairs); Duhaime et al., *Head Injury in Very Young Children*, *supra* note 92, at 181 (71 children two years of age or younger suffering from head trauma due to falls; none had retinal hemorrhage; 21 fell more than four feet, 10 fell down stairs, and eight fell down stairs in a walker); Johnson et al., *supra* note 197, at 233 ("None of the 70 children in

hemorrhages associated with unintentional head injury tend to be limited to victims of motor vehicle accidents.²¹⁹

Some have argued that CPR can cause retinal hemorrhages,²²⁰ due to the transmission of pressure from the chest to the head and thence to the retina.²²¹ This hypothesis appears to have evolved by influence from case reports earlier in the century in which some adults who sustained major accidental chest compression developed retinal hemorrhages, so-called Purtscher retinopathy.²²² In children, there is weak support for the role of CPR in retinal hemorrhage formation, based largely on

our study who suffered falls significant enough to cause skull fracture and/or intracranial hemorrhage sustained a retinal hemorrhage"; 11 fell five to 10 feet, 17 fell down stairs, 12 fell one story or more off building.).

219. See Duhaime et al., *Head Injury in Very Young Children*, *supra* note 92, at 182 (76 accidentally injured children with head injury, 24 months or younger; one had a retinal hemorrhage associated with fatal injuries from auto accident); Johnson et al., *supra* note 197, at 233 (140 accidentally injured children with skull fractures and/or intracranial hemorrhages; two had retinal hemorrhages, both of them due to side impact auto accident); Riffenburgh & Sathyavagiswaran, *supra* note 197, at 1523 (autopsies of 92 accidental infant fatalities; four retinal hemorrhages, two in auto accidents, one birth trauma, one respiratory failure followed by resuscitation attempts). *But see* Duhaime et al., *Head Injury in Very Young Children*, *supra*, at 184 (reporting case in which well-witnessed fall down stairs resulted in retinal hemorrhage).

220. Compare Cruz & Giangiacomo, *supra* note 211, at 68 ("Retinal hemorrhages can occur rarely when cardiopulmonary resuscitation is administered.") and Dykes, *supra* note 138, at 213 (retinal hemorrhages may be attributable to "resuscitation efforts") with Gilles, *Abusive Head Injury in Children*, *supra* note 107, at 373 ("No definitive evidence exists which proves the theory that retinal hemorrhages occur as the direct result of cardiopulmonary resuscitation.").

221. See Mark G. Goetting & Bonnie Sowa, *Retinal Hemorrhage After Cardiopulmonary Resuscitation in Children: An Etiologic Reevaluation*, 85 PEDIATRICS 585, 587 (1990).

During intracranial hypertension, venous drainage through the central retinal vein is impeded. In addition, increased cerebrospinal pressure fluid in the subarachnoid space, which extends anteriorly along the optic nerve, will occlude the retinochoroidal anastomoses, thereby blocking all retinal venous drainage.

Id.

222. Examination of the term "Purtscher retinopathy" provides a good example of how the use of citation in medical literature may alter the initial report of an author. The term "Purtscher retinopathy" or "angiopathie retinae traumatica" derived from the original observations made by Purtscher in two papers which he wrote in 1910 and 1912. In the first paper, he reported a case of head trauma in an adult sustained during a fall, who subsequently developed retinal hemorrhages and "white spots of the retina" (exudates), and briefly described another adult who developed retinal hemorrhages after a fall off of a roof. In the second paper, the first two cases were the same as in the 1910 paper. The other three patients had skull fractures, and presumably, head injury was the major antecedent factor. Purtscher postulated that two types of events might result in retinopathy from sudden elevation of venous pressure, those resulting from chest compression and those resulting from head injury, even though his cases had head injury as a major component of their injuries, not compressive chest injury. The transition to the concept that "Purtscher retinopathy" meant a retinopathy developing only subsequent to a sudden violent thoracic compression occurred more recently in the literature. W.G. Marr & E.G. Marr, *Some Observations on Purtscher's Disease: Traumatic Retinal Angiopathy*, 54 AM. J. OPHTHALMOLOGY 693 (1962). The authors reviewed many of the case reports extant to date, which included both avenues of pathogenesis, and added one of their own, a 48 year old man who fell on a conveyor-belt and experienced a severe chest compression in a brick-cutting machine. As part of their discussion, the topic of traumatic asphyxia due to chest compression was reviewed. It is after this publication that "Purtscher retinopathy" is used synonymously with compressive chest-related retinopathy. Tomasi & Rosman, *supra* note 203.

isolated case reports.²²³ Almost without exception, these cases involve children for whom alternative causes of the retinal hemorrhages are actually more likely.²²⁴ Larger studies have not found an association between CPR and retinal hemorrhages; only two cases of retinal hemorrhages have been reported in 118 children who were administered CPR,²²⁵ and in these two cases, alternative explanations for the retinal hemorrhages have been offered.²²⁶ The best experimental data is based on piglets, whose retinal anatomy is similar to ours. Ventricular fibrillation (a heart rhythm abnormality) was induced in a series of six piglets. Sustained CPR was performed, with documentation of intra cardiac and intrathoracic pressures equivalent to those reached during CPR in the human. No retinal hemorrhages were found in any of the twelve eyes from the six piglets tested.²²⁷ In summary, it is exceedingly unlikely that CPR alone will result in retinal hemorrhages.

Blood disorders must be excluded as a cause of retinal hemorrhages. Bleeding disorders, anemia, and leukemia have all been associated with retinal hemorrhage

223. M.G.F. Gilliland & Martha W. Luckenbach, *Are Retinal Hemorrhages Found After Resuscitation Attempts?*, 14 AM. J. FORENSIC MED. & PATHOLOGY 187, 192 (1993); R.H. Kirschner & R.J. Stein, *The Mistaken Diagnosis of Child Abuse: A Form of Medical Abuse*, 139 AM. J. DISEASES CHILDREN 873 (1985); V.W. Weed et al., *Retinal Hemorrhage in an Infant After Cardiopulmonary Resuscitation*, 11 AM. J. FORENSIC MED. & PATHOLOGY 79 (1990).

224. These cases almost without exception consist of children with severe underlying pathology or with a significant hypoxic/ischemic component. Without adequate prearrest documentation that the retinas were normal, "assigning responsibility to the actual cardiopulmonary resuscitation is impossible." Gilles, *Abusive Head Injury in Children*, *supra* note 107, at 373-74.

225. See Budenz et al., *supra* note 149, at 560-61, 563 (autopsies of four infants dying of SIDS who received CPR; none had retinal hemorrhages); Gilliland & Luckenbach, *supra* note 223, at 190 (autopsies of 169 child deaths, mean age 17 months; "A total of 49 children in our series received prolonged vigorous resuscitative attempts and died of comparable causes to those in Goetting and Sowa's series . . . None of these children had retinal hemorrhages."); Goetting & Sowa, *supra* note 221, at 585 (20 children with no history of trauma or conditions known to be associated with retinal hemorrhages undergoing CPR; two had retinal hemorrhages); R.K. Kanter, *Retinal Hemorrhages After Cardiopulmonary Resuscitation or Child Abuse*, 108 J. PEDIATRICS 430 (1986) (45 children with no history of trauma undergoing CPR; one had retinal hemorrhages, but also suffered from seizures and severe arterial hypertension); cf. Riffenburgh & Sathyavagiswaran, *supra* note 197, at 1521 (autopsies of 92 accidental child deaths; one case of retinal hemorrhages attributed to CPR). It should be noted, however, that Kanter's study has been criticized as underestimating the number of retinal hemorrhages due to his failure to use mydriatics (which dilate the pupils, facilitating detection of retinal hemorrhages). Alex V. Levin, *Retinal Hemorrhages After Cardiopulmonary Resuscitation or Child Abuse*, in TEXTBOOK OF PEDIATRIC EMERGENCY CARE, *supra* note 116, at 269.

226. See Gilles, *Abusive Head Injury in Children*, *supra* note 107, at 373.

Of the two cases with identified retinal hemorrhages, one died of diffuse cerebral edema and severe bilateral pneumonia while the other was an infant found apneic and pulseless while hospitalized for suspected sepsis. In the latter case, the length of time between arrest and the onset of cardiopulmonary resuscitation and a documented lack of retinal hemorrhages before arrest are not known. Whether the infant was smothered or not is also unknown.

Id.

227. James C. Fackler, *Retinal Hemorrhages in Newborn Piglets Following Cardiopulmonary Resuscitation*, 146 AM. J. DISEASES CHILDREN 1294 (1992).

development.²²⁸ The rupture of blood vessel abnormalities such as aneurysms, (though rare), may lead to the secondary development of retinal hemorrhages.²²⁹ Other possible causes include hypertension²³⁰ and infectious diseases, such as meningitis²³¹ and bacterial endocarditis.²³²

Papilledema refers to swelling of the optic nerve due to increases in intracranial pressure. In the presence of papilledema, retinal hemorrhages should not be attributed to abuse.

E. Subdural Hematomas

A subdural hematoma is a collection of blood within the dural membranes. Subdural hematomas are most commonly thought to occur when sudden and violent movement of the head leads to rupturing of the bridging veins (sometimes arteries) that travel from the brain's surface, through the pia, arachnoid, and dura, into the superior sagittal sinus (see Figure 7).²³³ The superior sagittal sinus is one of the dural

228. Budenz et al., *supra* note 149, at 562-63; Dykes, *supra* note 138, at 213; Eisenbrey, *supra* note 197, at 40; Gilles, *Abusive Head Injury in Children*, *supra* note 107, at 371; cf. J.M. Holt & E.C. Gordon-Smith, *Retinal Abnormalities in Diseases of the Blood*, 53 BRIT. J. OPHTHAMOLOGICAL 145, 146, 159 (1969) (152 patients with blood disease, including anemia, leukemia, and thrombocytopenia; one-third had retinal hemorrhages, exudates, or papilledema; anemia associated with abnormal findings but "[r]etinal hemorrhages were not seen when the sole abnormality in the peripheral blood was thrombocytopenia"); *id.* at 158 (suggesting that severe anemia leads to retinal hemorrhages both because anemia can cause anemic hypoxia (lack of oxygen), which damages a protective layer of the blood vessels (the endothelium) and makes them vulnerable to bursting, and because anemia leads to an increase in intracranial pressure, which increases retinal vein pressure). Techniques for diagnosing bleeding disorders are discussed *supra*, in subpart V.B.4.

229. Budenz et al., *supra* note 149, at 543; Gilles, *Abusive Head Injury in Children*, *supra* note 107, at 371. Bruce and Zimmerman argue that retinal hemorrhages in the absence of a history of severe trauma is nevertheless "diagnostic of some type of shaken impact injury," noting that "although [retinal hemorrhages] can occur with severe acute subarachnoid hemorrhage resulting from a ruptured arteriovenous malformation or aneurysm, such underlying factors are rare in small babies." Bruce & Zimmerman, *supra* note 138, at 484.

230. Duhaime et al., *Head Injury in Very Young Children*, *supra* note 92, at 183; Gilles, *Abusive Head Injury in Children*, *supra* note 107, at 371.

231. Gilles, *Abusive Head Injury in Children*, *supra* note 107, at 371. Ludwig and Warman argue that in "meningitis, retinal hemorrhage is rare. Retinal hemorrhage might be seen only if the meningitis were complicated by a subdural empyema, brain abscess, or venous sinus thrombosis." Ludwig & Warman, *supra* note 136, at 106.

232. Dykes, *supra* note 138, at 213; Gilles, *Abusive Head Injury in Children*, *supra* note 107, at 371.

233. Gilles, *Abusive Head Injury in Children*, *supra* note 107, at 361; Kleinman, *Skeletal Trauma*, *supra* note 36, at 177; O'Brien, *supra* note 211. This observation is based in part upon experiments in which whiplash forces (sudden forward or backwards rotations of the head) were applied to monkeys. See Gennarelli & Thibault, *supra* note 144, at 681 (sagittal rotational acceleration of the head led to rupture of parasagittal bridging veins, frontally predominant and frequently extending into the interhemispheric fissure); Ommaya et al., *supra* note 208, at 288-89 ("The cortical draining veins enter the sagittal sinus in the parasagittal zone and thus are particularly liable to rupture. We found that subdural bleeding in these areas was by far the commonest visible lesion" following whiplash."). It is also consistent with Holbourn's pioneering work with two-dimensional gelatin models of the brain. Holbourn, *supra* note 144, at 440. Finally, Zimmerman et al., noted that in 10 of the 15 cases of abuse-related interhemispheric subdural hematomas, the caretakers ultimately admitted to having shaken the child. Robert A. Zimmerman et al., *Computed Tomography of Craniocerebral Injury in the Abused Child*, 130 RADIOLOGY 687, 689 (1979) [hereinafter Zimmerman et al., *Computed Tomography of Craniocerebral Injury*].

sinuses (also known as venous sinuses), through which blood drains from the brain. It is located within the dura between the hemispheres of the brain. (see Figure 7). Bridging veins to the superior sagittal sinus are thought to be the most prone to injury. The portion of the bridging veins crossing the potential subdural space at this site is structurally weaker than the bridging veins at other areas.²³⁴ In addition, the dura is relatively adherent to the skull along the length of the superior sagittal sinus. Rupturing of the veins or arteries typically leads to bleeding within the potential subdural space, and a subdural hematoma forms.

Typically, infants with subdural hematomas attributable to abuse are presented with other markers of intracranial and intraocular injury, including brain swelling and edema, brain infarction, retinal and optic nerve sheath hemorrhages, and subarachnoid hemorrhages.²³⁵ Obstructive hydrocephalus may be observed early in the course of the postinjury period due to the blockage of cerebrospinal fluid flow from brain swelling and shift, or later due to impaired cerebrospinal fluid absorption.²³⁶

A subdural hematoma in a previously healthy infant with no history of trauma, or with a history of minor trauma, is highly suggestive of abuse.²³⁷ Subdural hematomas are both a common pathologic consequence of shake-impact events²³⁸ and a relatively uncommon consequence of other types of trauma and disease. Subdural hematomas that are located between the two hemispheres are most

234. See Hardman, *supra* note 175, at 976 (noting that the subdural portions of the bridging veins "have thin irregular collagenous walls and do not have covering arachnoid trabeculae"); Tetsumori Yamashima & Reinhard L. Friede, *Why Do Bridging Veins Rupture into the Virtual Subdural Space?*, 47 J. NEUROLOGY NEUROSURGERY & PSYCHIATRY 121, 127 (1984).

235. See, e.g., Duhaime et al., *The Shaken Baby Syndrome*, *supra* note 142, at 410 (in a 1987 article, associated findings include subarachnoid and retinal hemorrhages); Zimmerman et al., *Computed Tomography of Craniocerebral Injury*, *supra* note 233 (associated findings in abused children with interhemispheric subdural hematomas included edema and infarction; 80% also had retinal hemorrhages).

236. Hardman, *supra* note 175.

237. Case, *supra* note 185, at 81.

238. See Brown & Minns, *supra* note 89, at 858 (30 abused children with brain involvement; 16 or 53% with subdural hematomas); Cohen et al., *supra* note 137, at 98 (37 abused children with serious head injury, mean age nine months; 9 or 24% had subdural hematomas); Duhaime et al., *Head Injury in Very Young Children*, *supra* note 92, at 181 (100 children 24 months old or younger with head injury, 24 abused, 76 nonabused; 16/24 or 67% had subdural hematomas, 3/76 or 4% of nonabused had subdural hematomas); Dykes, *supra* note 138 (review of 7 studies of whiplash shake injury with total of 99 children; 56 or 57% had subdural hematomas); Goldstein et al., *supra* note 197, at 1330 (40 children with head injury, 14 abused and 26 nonabused; 8/14 or 57% had subdural hematomas, 3/26 or 12% of nonabused had subdural hematoma); Hahn et al., *supra* note 172 (77 children with head injury due to abuse, 85% under two years of age; 30 or 39% had subdural hematomas); Hobbs, *Skull Fracture and the Diagnosis of Abuse*, *supra* note 90, at 250 (89 children with skull fractures, 29 due to abuse and 60 accidental; 15 of 15 children with subdural hematomas were abused); Launius et al., *supra* note 137, at 40 (Subdural hematomas are the most common intracranial consequence of abuse.); Ludwig & Warman, *supra* note 136 (20 shaken infants without external evidence of head trauma, average age 5.8 months; 10 or 50% had subdural hematomas); Charles Q. McClelland et al., *Cerebral Injury in Child Abuse: A Changing Profile*, 7 CHILD'S BRAIN 225, 227 (1980) (21 children with head injury due to abuse, 57% under one year of age, 4 with subdural hematomas, 6 characterized as shaken babies); Zimmerman et al., *Computed Tomography of Craniocerebral Injury*, *supra* note 233, at 688 (26 abused children with head injury, 80% under two years of age; 17 or 65% had subdural hematomas).

suspicious for an abusive etiology.²³⁹ These are known as interhemispheric or falxine hematomas, or, if between the two infolding layers of dura that comprise the falx, as interdural hematomas. The next most common location is over the convexities of the hemispheres.

Subdural hematomas are not pathognomonic (definitely diagnostic) for abusive injury. Alternative etiologies must be considered, including blood disorders,²⁴⁰ meningitis,²⁴¹ blood vessel abnormalities,²⁴² neurodegenerative disease,²⁴³ birth trauma,²⁴⁴ and unintentional trauma.

Subdural hematomas are only rarely caused by the birth process,²⁴⁵ and when they do occur, are often, though not invariably, associated with a difficult delivery.²⁴⁶

239. Brown & Minns, *supra* note 89, at 862; Gilles, *Abusive Head Injury in Children*, *supra* note 107, at 361; Klein, *supra* note 167, at 80; *see* Dykes, *supra* note 138, at 215-16 (three of three shaken babies described in J.E. Carter & A.Q. McCormick, *Whiplash Shaking Syndrome: Retinal Hemorrhages and Computerized Axial Tomography of the Brain*, 7 CHILD ABUSE & NEGLECT 279 (1983), had interhemispheric subdural hematomas); Goldstein et al., *supra* note 197, at 1331 (eight abused children with subdural hematomas; four or 50% of subdural hematomas were located in the posterior intra hemispheric region); Sara A. Sinal & Marshall A. Ball, *Head Trauma Due to Child Abuse: Serial Computerized Tomography in Diagnosis and Management*, 80 S. MED. J. 1505, 1511 (1987) (seven of 17 infants with shaken infant syndrome had interhemispheric subdural hematomas); Zimmerman et al., *Computed Tomography of Craniocerebral Injury*, *supra* note 233, at 688 (17 abused children with subdural hematomas; 15 or 88% of subdural hematomas were located in the posterior interhemispheric region).

240. *See* Hardman, *supra* note 175, at 975 (blood dyscrasias can lead to subdural hematoma); Klein, *supra* note 167, at 82 (same); Newton, *supra* note 165, at 188 ("Hemorrhagic disease of the newborn may present with subdural hematoma emphasizing the importance of a platelet count and clotting screen at any age."). Techniques for diagnosing blood disorders are discussed *supra*, in subpart V.B.4.

241. Kleinman, *Skeletal Trauma*, *supra* note 36, at 182; *see* Eisenbrey, *supra* note 197 (meningitis can lead to subdural hematoma). Techniques for diagnosing meningitis are discussed *supra*, in subpart V.B.4.

242. *See* Gilles, *Abusive Head Injury in Children*, *supra* note 107, at 342 (arteriovenous malformation can lead to subdural hematoma); Hardman, *supra* note 175 (ruptured aneurysm can lead to subdural hematoma). Techniques for diagnosing blood vessel abnormalities are discussed *supra*, in subpart V.B.4.

243. Hitoshi Osaka et al., *Chronic Subdural Hematoma as an Initial Manifestation of Glutaric Aciduria Type-I*, 15 BRAIN & DEV. 125, 127 (1993).

244. Newton, *supra* note 165, at 188.

245. Akira Ikeda et al., *Infantile Acute Subdural Hematoma*, 3 CHILD'S NERVOUS SYS. 19, 20 (1987); Takashi Hayashi et al., *Neonatal Subdural Hematoma Secondary to Birth Injury: Clinical Analysis of 48 Survivors*, 3 CHILD'S NERVOUS SYS. 23 (1987) [hereinafter Hayashi et al., *Neonatal Subdural Hematoma*]; *see* Mauricio Castillo & Lynn A. Fordham, *MR of Neurologically Symptomatic Newborns After Vacuum Extraction Delivery*, 16 AJNR 816, 817-18 (1995) (case reports of three clinically significant intracranial injuries following vacuum extraction delivery; a "review of the international literature showed that the overall prevalence of intracranial hemorrhage was .72% of 14,276 vacuum extraction deliveries"); Hardman, *supra* note 175, at 989 ("With modern obstetrical care, such hemorrhages are quite rare.").

246. *See* Israel F. Abroms et al., *Acute Neonatal Subdural Hematoma Following Breech Delivery*, 131 AM. J. DISEASES CHILDREN 192, 194 (1977) (noting relationship between breech deliveries and intracranial injury, including subdural hematomas); Hayashi et al., *Neonatal Subdural Hematoma*, *supra* note 245 (48 neonatal subdural hematomas; 22 of 48 or 46% involved suction, 11 of 48 or 23% were breech deliveries); Stephan E. Natelson & Martin P. Sayers, *The Fate of Children Sustaining Severe Head Trauma During Birth*, 51 PEDIATRICS 169, 172 (1973) (10 acute subdural hematomas and 13 chronic subdural hematomas attributed to birth process; "in all cases there was a history of traumatic delivery, manifestation of injury from birth, or a thick neomembrane"). *But see* Newton, *supra* note 165, at 188.

It must be remembered that subdural hemorrhage may occur after apparently atraumatic deliveries including caesarean section. Regardless of the birth route taken shearing forces are applied to the head.

Birth trauma is an exceedingly unlikely cause if a sudden onset of neurologic symptoms occurs in a previously healthy infant more than three to four weeks of age.²⁴⁷

Subdural hematomas "are coincident far more often with nonaccidental than with accidental head injury."²⁴⁸ Falls, even of great height, are not likely to result in clinically significant subdural hematomas.²⁴⁹ ("Clinically significant" refers to those which are associated with significant neurologic symptoms.) Falls down stairs and baby walker accidents will likewise rarely result in subdural hematomas, and then only in exceptional circumstances.²⁵⁰

Although occasionally reported in the context of abusive injuries, epidural hematomas are far more frequently a result of unintentional injury.²⁵¹ They often

Even at caesarean section the head often needs disengaging and further pressure changes occur as it moves through a uterine incision that may be tight into atmospheric pressure at the moment when venous pressure is also rising.

Id.; Hayaski et al., *Neonatal Subdural Hematoma*, *supra*, at 27 (31% of neonatal subdural hematomas followed simple spontaneous delivery; "tentorial tearing may be produced by the molding process, even in normal labor").

247. See Newton, *supra* note 165, at 188 ("Symptoms can be insidious in onset and within the first three or four weeks of life birth trauma remains a likely culprit.").

248. Gilles, *Abusive Head Injury in Children*, *supra* note 107, at 361; see Billmire & Myers, *supra* note 197, at 341 (84 children less than one year of age with head injury and/or abnormal CT findings; 30 abused, 54 nonabused; intracranial bleeding without skull fracture in 11 of 30 of abused children and 1 of 8 of nonabused children; nonabused child with intracranial bleeding was an unrestrained passenger in auto accident); Duhaime et al., *Head Injury in Very Young Children*, *supra* note 92, at 181 (100 children 24 months or younger with head injury, 24 abused, 76 nonabused; 13 of 24 abused infants had subdural hematomas; 3 of 76 nonabused children, and all 3 were attributable to serious auto accidents).

249. For falls less than ten feet, see Helfer et al., *supra* note 92, at 219 (three-foot falls in children under five reported by parents with no serious injuries and 85 three-foot falls in hospitals occurred to children under five; no serious injuries); Kravitz et al., *supra* note 92, at 873 (536 children under one year of age falling 20 to 60 inches; 1 subdural hematoma); Lyon & Oates, *supra* note 92, at 126 (207 25- to 54-inch falls in the hospital in children six years old or younger; no serious injuries); Nimityongskul & Anderson, *supra* note 92, at 185 (76 one- to three-foot falls in children under 17; no serious injuries); Williams, *supra* note 99, at 1351 (50 witnessed falls of less than 10 feet among children less than three years of age; only serious injuries were three depressed skull fractures with no loss of consciousness). For falls greater than ten feet, see Barlow et al., *supra* note 115, at 510 (1 subdural among 61 children falling greater than 10 feet; child with subdural fell more than three stories); Meller & Shermata, *supra* note 115, at 1275 (no subdural hematomas in 48 children falling up to 13 stories; average 2.7 stories); Musemeche et al., *supra* note 115, at 1348 (2 subdural hematomas among 70 children up to 15 years of age falling 10 feet or more); Smith et al., *Injuries in Children Sustained in Free Falls*, *supra* note 56, at 989 (2 subdural hematomas among 66 children falling up to eight stories; most of falls greater than 10 feet).

250. See Chiavello et al., *supra* note 95 (1 subdural hematoma among 69 children under five falling down stairs; child was an infant being carried by an adult who fell); Francis J. DiMario, Jr., *Chronic Subdural Hematoma: Another Babywalker-Stairs Related Injury*, 29 CLINICAL PEDIATRICS 405, 405-06 (1990) (six-month-old in baby walker fell down a full flight of wooden stairs to a cement floor; presented at 23 months with chronic subdural hematoma); Joffe & Ludwig, *supra* note 56, at 458 (no central nervous system contusions or bleeding among 363 children falling down stairs); Rieder et al., *supra* note 95, at 489 (139 children with walker-related injuries seen at hospital, average age nine months; no intracranial injuries). *But see* Chiavello et al., *supra* (criticizing Joffe and Ludwig and suggesting that the failure to obtain CT scans may have resulted in intracranial injury being missed in some of the subjects).

251. See M. Meuli et al., *Characteristics and Prognosis of Extradural Hematomas in Children*, 1 EUR. J. PEDIATRIC SURGERY 196, 198 (1991) (31 children with epidural hematomas, average age eight years; none attributed to abuse); Richard P. Shugarman et al., *Epidural Hemorrhage: Is it Abuse?*, 97 PEDIATRICS 664 (1996)

occur after relatively minor trauma. While their initial clinical presentation may be dramatic, children almost always do well clinically. This makes sense given that the biomechanics of epidural formation are quite different from subdural. Brain substance is often not damaged by the local deforming forces which result in epidural hematomas when applied forces are small.

F. Subarachnoid Hemorrhages

A subarachnoid hemorrhage (SAH) refers to blood in the subarachnoid space mixed with cerebrospinal fluid. SAH may develop following direct surface trauma to the brain from contact with the inner portion of the skull, or leakage of blood from ruptured blood vessels crossing the subarachnoid space.²⁵² If extensive, SAHs may affect cerebrospinal fluid flow, leading to hydrocephalus. Brain ischemia and infarction have been reported as secondary consequences of SAH also.²⁵³

Given the limitations of current imaging techniques, subarachnoid hemorrhages are often difficult to identify or to distinguish from other conditions, such as subdural hematomas²⁵⁴ and subjacent brain swelling.²⁵⁵ SAH over the cerebral convexities are particularly difficult to identify. Smaller hemorrhages may be difficult to distinguish from the normal appearance of the falx.²⁵⁶ Definitive diagnosis is possible by examining the cerebrospinal fluid, usually obtained by a lumbar puncture (also

(93 children three years old and under with epidural or subdural hematomas, median age 15 months; abuse diagnosed in 28 of 59 or 47% of children with subdural hematomas and 2 of 34 or 6% of children with epidural hematomas).

252. David A. Ramsay & David M. Robertson, *Meninges and Their Reaction to Injury*, in TEXTBOOK OF NEUROPATHOLOGY, *supra* note 163, at 170.

253. Garcia & Anderson, *supra* note 163, at 702.

254. Cohen et al., *supra* note 137, at 101; Kleinman, *Skeletal Trauma*, *supra* note 36, at 190; Merten & Carpenter, *supra* note 142, at 828. *But see* Robert D. Zimmerman et al., *Falx and Interhemispheric Fissure on Axial CT: II. Recognition and Differentiation of Interhemispheric Subarachnoid and Subdural Hemorrhage*, 3 AJNR 635, 640 (1982) [hereinafter Zimmerman et al., *Falx and Interhemispheric Fissure*] (reviewed CT scans of 50 subarachnoid hemorrhages, three patients with acute cerebral edema, and 22 patients with interhemispheric subdural hematoma; interhemispheric subdural hematomas always appeared in posterior part of the interhemispheric fissure, whereas subarachnoid hemorrhages did not appear in posterior part on CT scans).

255. Cohen et al., *supra* note 137, at 101; Kleinman, *Skeletal Trauma*, *supra* note 36, at 190; Zimmerman et al., *Falx and Interhemispheric Fissure*, *supra* note 254, at 637.

256. Cohen et al., *supra* note 137, at 101; Kleinman, *Skeletal Trauma*, *supra* note 36, at 190; Zimmerman et al., *Falx and Interhemispheric Fissure*, *supra* note 254. Subarachnoid hemorrhages may be confused with the normal falx on CT scans because the CT differentiates between substances based on their relative density; substances with greater density appear lighter on the CT. Subarachnoid hemorrhages appear as hyperdense, since blood is more dense than cerebrospinal fluid. However, the normal falx also appears hyperdense. In adults, a subarachnoid hemorrhage may be distinguished from normal falx by a zig-zag appearance, which reflects the fact that the subarachnoid space (and therefore the subarachnoid hemorrhage) extends into the sulci (furrows) of the brain, whereas the falx appears as a straight line. In children, however, there is less space between the hemispheres, and therefore a subarachnoid hemorrhage often appears as a straight line rather than as a zig-zag, making it extremely difficult to differentiate from normal falx. *Id.*

known as a spinal tap),²⁵⁷ or at necropsy. However, because a lumbar puncture is performed in only a small percentage of infants suspected of having been abused, definitive diagnosis of SAH in the live child may be difficult.

Although SAH are thought to occur commonly as a consequence of shake-impact events,²⁵⁸ alternate causes must be ruled out, such as birth trauma,²⁵⁹ bleeding disorders,²⁶⁰ meningitis,²⁶¹ blood vessel abnormalities,²⁶² and unintentional trauma.

SAHs are found in at least 10% of newborns, according to one 1925 study, but the vast majority of these hemorrhages do not cause any significant symptoms.²⁶³ These SAHs are often small and patchy in their distribution. Dating by examination of cerebrospinal fluid analysis is very helpful in excluding birth trauma as the cause. By looking at the cell count, cell morphology, glucose and protein content of the spinal fluid, and spinal fluid appearance, clinicians may estimate the age of bleeding with some degree of accuracy (especially if the bleeding is recent). For instance, children over a few weeks old with a sudden onset of neurologic symptoms (such as seizures and unconsciousness) and multiple markers of injury were not injured

257. See Cohen et al., *supra* note 137, at 98 (of eight abused children with subarachnoid hemorrhages who received lumbar puncture, all eight had bloody cerebrospinal fluid). The process of obtaining spinal fluid can injure the venous plexus which surrounds the spinal meninges, leading to bloody cerebrospinal fluid, the "traumatic tap." Clinicians sometimes miss the diagnosis of SAH because they assume bloody fluid is the consequence of the technique. Conversely, they sometimes overcall SAH if they do not take appropriate steps in analyzing the fluid. However, there are several means by which one can distinguish between a traumatic tap and signs of subarachnoid bleeding. See Julio O. Apolo, *Bloody Cerebrospinal Fluid: Traumatic Tap or Child Abuse?*, 3 PEDIATRIC EMERGENCY CARE 93 (1987) (in traumatic tap, the specimens contain less blood as more CSF is drawn, and if the specimens are centrifuged, the resulting liquid (supernatant) is clear, whereas if blood has been present in the CSF for one to two hours, the supernatant will be yellowish in color (xanthochromic)); Brown & Minns, *supra* note 89, at 863 (same); Robert M. Spear et al., *Fatalities Associated With Misinterpretation of Bloody Cerebrospinal Fluid in the 'Shaken Baby Syndrome,'* 146 AM. J. DISEASES CHILDREN 1415, 1416 (1992) (same).

258. See Brown & Minns, *supra* note 89, at 858 (30 abused children with head trauma; three or 10% with subarachnoid hemorrhage alone; percentage with subarachnoid hemorrhage and other findings not reported); Cohen et al., *supra* note 137, at 98 (37 abused children with head trauma, 95% less than one year of age; 27 or 73% with subarachnoid hemorrhage); Duhaime et al., *Head Injury in Very Young Children*, *supra* note 92, at 181 (24 abused children with head trauma two years of age or younger; 13 with subdural hemorrhage); Goldstein et al., *supra* note 197, at 1330 (14 abused children with head trauma, average age 19 months; four or 28% with subarachnoid hemorrhage); Kleinman, *Skeletal Trauma*, *supra* note 36, at 190 (citing D.F. Merten & D.R.S. Osborne, *Cranio-cerebral Trauma in the Child Abuse Syndrome: Radiological Observations*, 14 PEDIATRIC RADIOLOGY 272 (1984)) (36 abused children with head trauma; nine or 25% with subarachnoid hemorrhage); Ludwig & Warman, *supra* note 136, at 105 (20 shaken infants without external evidence of head trauma, average age 5.8 months; five or 25% with subarachnoid hemorrhage); Sinal & Ball, *supra* note 239, at 1508, 1511 (24 abused children with head trauma, 96% under one year of age; nine of 23 or 39% with subarachnoid hemorrhage on initial CT scan).

259. P. Govaert et al., *CT Diagnosis of Neonatal Subarachnoid Hemorrhage*, 20 PEDIATRIC RADIOLOGY 139 (1990); Hardman, *supra* note 175, at 989.

260. Garcia & Anderson, *supra* note 163, at 701. Techniques for diagnosing bleeding disorders are discussed *supra*, in subpart V.B.4.

261. Apolo, *supra* note 257, at 94. Techniques for diagnosing meningitis are discussed *supra*, in subpart V.B.4.

262. Bruce, *supra* note 137, at 87. Techniques for diagnosing blood vessel abnormalities are discussed *supra*, in subpart V.B.4.

263. Hardman, *supra* note 175, at 989.

during the birth process. Obtaining the birth medical record of both mother and child is helpful in documenting any perinatal (around the time of birth) complications.

Unintentional head injury is an uncommon cause of clinically significant SAHs. Surveys of free falls (of less than four feet)²⁶⁴ and falls down stairs²⁶⁵ do not report any cases of SAH. However, as noted above, this fact must be qualified by the warning that unless clinical signs of neurologic injury are present, a child is unlikely to receive a scan that could detect SAH.²⁶⁶ In the rare case report of SAH resulting from short falls, falls down stairs, or falls down stairs in a baby walker, the bleeding is usually focal (limited) rather than diffuse, with no neurologic complications.²⁶⁷

G. Extra-Axial Collections Including Subdural Effusions

Sometimes, collections of fluid within the subdural or subarachnoid space over the convexities of the brain can be confused with chronic subdural hematomas on a CT scan.²⁶⁸ The fluid appears slightly less dense than surrounding brain tissue (hypodense), which gives rise to its possible misinterpretation as a chronic subdural hematoma. One such condition is known as a benign subdural effusion, in which the infant is presented with an abnormally enlarged head, but typically exhibits normal psychomotor development, and shows no other signs of brain injury or clinical symptoms of brain irritability (*i.e.*, vomiting, decreased feeds, fussiness, or lethargy).²⁶⁹ These effusions typically resolve as the child ages. CT scans will reveal enlarged ventricles and large subarachnoid cisternal spaces, which are attributable to the increase in cerebrospinal fluid volume, but which may be misinterpreted by

264. See Helfer et al., *supra* note 92 (219 three-foot falls in children under five reported by parents; no serious injuries and 85 three-foot falls in the hospital in children under five; no serious injuries); Lyon & Oates, *supra* note 92, at 126 (207 25- to 54-inch falls in the hospital in children six or younger; no serious injuries); Nimityongsukul & Anderson, *supra* note 92 (76 one- to three-foot falls in children under 17 years of age; no serious injuries); Williams, *supra* note 99, at 1351 (50 witnessed falls of less than 10 feet among children less than three years of age; only serious injuries were three depressed skull fractures with no loss of consciousness).

265. See Chiavello et al., *supra* note 95, at 680 (no subarachnoid hemorrhages among 69 children under five falling down stairs); Joffe & Ludwig, *supra* note 56, at 458 (no central nervous system contusion or bleeding among 363 children falling down stairs); Rieder, *supra* note 95, at 489 (139 children with walker-related injuries seen at hospital, average age nine months; no intracranial injuries).

266. Cf. Chiavello et al., *supra* note 95, at 680 (noting that absence of intracranial injury in Joffe and Ludwig's study of falls down stairs might be attributable to failure to obtain CT scans).

267. Duhaime et al., *Head Injury in Very Young Children*, *supra* note 92, at 181 (39 children 24 months or younger with head injury attributed to fall of more than four feet, down stairs, or down stairs in a walker; two subarachnoid hemorrhages).

268. Bruce, *supra* note 137, at 84; Dykes, *supra* note 138, at 216; Gilles, *Abusive Head Injury in Children*, *supra* note 107; Giuseppe Scotti et al., *Evaluation of the Age of Subdural Hematomas by Computerized Tomography*, 47 J. NEUROSURGERY 311, 314 (1977). CT and MRI as diagnostic tools are discussed *infra*. See *infra* notes 287-304 and accompanying text.

269. Dykes, *supra* note 138, at 216; Laura R. Ment et al., *Benign Enlargement of the Subarachnoid Spaces in the Infant*, 54 J. NEUROSURGERY 504 (1981); William C. Robertson et al., *Benign Subdural Collections of Infancy*, 94 J. PEDIATRICS 382, 384 (1979).

radiologists as evidence of brain shrinkage (atrophy).²⁷⁰

Another condition is referred to as a hygroma, which simply means a collection of fluid. There is considerable confusion regarding the significance of hygromas. Some believe they are chronic subdural hematomas; others believe they are collections of cerebrospinal fluid. An MRI, CT with a contrast agent, or CT with careful comparison of relative densities of fluid and surrounding brain tissue can be useful in differentiating the type of fluid collection.²⁷¹

H. Dating of Injuries and the Use of CT and MRI

A combination of clinical factors and pathologic findings help clinicians to estimate the timing of injury, although the estimates are often insufficiently precise to eliminate all possible suspects. In general, the more severe the injury, the easier it is to date, since children who sustain very severe brain injuries are almost invariably symptomatic and unconscious from the time of the trauma.²⁷² Symptoms that are commonly associated with post-traumatic intracranial injury include immediate unconsciousness (the duration of which will vary depending on the degree and extent of brain injury), lethargy, breathing problems or apnea (cessation of breathing), decreased appetite, irritability, seizure, and vomiting.²⁷³ In the severely injured infant, seizures frequently develop in the immediate post-injury period. Brain swelling (which, as used here, includes edema and hyperemia) may develop quite rapidly.²⁷⁴

On CT and MRI scans, subdural hematomas and intracerebral hemorrhages have different appearances depending upon the length of time since the injury occurred. Dating an injury by imaging alone is sometimes difficult, as discussed below. The best, albeit still imperfect, means of dating intracranial injury is through the examination of pathologic material through operative intervention or at necropsy. Obviously, such material is often unavailable.

A frequently raised question is whether the findings might be referable to birth trauma. In most infants, this is easily addressed by evaluating the type and extent of

270. Robertson et al., *supra* note 269, at 384. The fact that the head is growing rapidly, and that fluid collections are present, does not mean that the brain is shrinking. This is a common error among radiologists, who often have limited clinical information. Following severe injury to the developing central nervous system, such as abusive head injury, the brain often experiences limited development and/or volume loss. Clinically, this correlates with a significant drop in head growth, resulting in what is known as microcephaly.

271. Bruce, *supra* note 137, at 85.

272. Bruce, *supra* note 137; Zimmerman et al., *Computed Tomography of Shearing Injuries*, *supra* note 180; see Marcus B. Nashelsky & Jay D. Dix, *The Time Interval Between Lethal Infant Shaking and Onset of Symptoms*, 16 AM. J. FORENSIC MED. & PATHOLOGY 154, 157 (1995).

273. Bruce, *supra* note 137; Duhaime et al., *The Shaken Baby Syndrome*, *supra* note 142; Gilles, *Abusive Head Injury in Children*, *supra* note 107; Ludwig & Warman, *supra* note 136.

274. Arthur Kобрine et al., *Demonstration of Massive Traumatic Brain Swelling Within 20 Minutes After Injury*, 46 J. NEUROSURGERY 256 (1977); Shiro Waga et al., *Traumatic Cerebral Swelling Developing Within 30 Minutes After Injury*, 11 SURGICAL NEUROLOGY 191 (1979).

injuries and the temporal clinical course (the timeline over which symptoms have developed). Cranial and intracranial injuries in the newborn infant range from scalp soft tissue injuries to epidural and subdural hematoma to peripheral nerve injuries.²⁷⁵ Typically these infants are presented with symptoms while still hospitalized after birth, making the differentiation from the abused infant fairly straightforward. An infant presenting, for instance, with the sudden onset of unconsciousness and apnea at eight weeks of age, an acute subdural hematoma, and retinal hemorrhages and an uncomplicated birth history did not incur those injuries as the result of birth trauma.²⁷⁶ Review of the birth and pediatric records is often helpful as part of the investigatory process in firmly establishing that birth-related complications and other conditions were or were not a contributing factor to the acute clinical presentation.

The most common imaging technique used is computerized tomography, or CT scan. On a CT scan, low density matter appears dark, and higher density matter appears light. The rank order of substances by increasing density from dark to light is water, white matter, gray matter, flowing blood, clotted blood, calcified tissue, and bone.²⁷⁷

Subdural hematomas change in appearance on CT scans as they age and the red blood cells within the hematoma break down.²⁷⁸ Fresh blood collections are dense, clotted blood denser still, and both appear white on CT scans.²⁷⁹ Up to one week of age, subdural hematomas generally appear white. Subacute hematomas (more than one week and up to three weeks old) are darker, and often have the same appearance as the surrounding brain (isodense). Chronic hematomas (over three weeks of age) are hypodense, and appear dark on the scan.²⁸⁰ The use of an intravenous contrast agent may allow for identification of subdural neomembranes, which form as subdural hematomas resolve. Only acute subdural hematomas are consistently diagnosed correctly.²⁸¹ Subdural hematomas that are mixed with cerebrospinal fluid are rapidly accumulating, and, in an anemic child, may be misdiagnosed in terms of

275. Abroms & Rosen, *supra* note 156 (A nice overview of neurologic and cranial pathology of the newborn.).

276. Bruce, *supra* note 137, at 66-107.

277. Jack deGroot & Joseph G. Chusid, *Ventricles & Coverings of the Brain*, in *CORRELATIVE NEUROANATOMY* 114, 229 (21st ed. 1991).

278. See Paul F.J. New & Saul Aronow, *Attenuation Measurements of Whole Blood and Blood Fractions in Computed Tomography*, 121 *RADIOLOGY* 635, 639 (1976) ("[C]hanges observed on CT scans are consistent with the progressive breakdown of red cells and removal of those elements of the red cell, predominantly protein, contributing the most heavily to the high density of clot," and are also consistent with redistribution of edema fluid throughout hematoma, which reduces density.).

279. *Id.* at 638-39.

280. See Scotti et al., *supra* note 268, at 312 (50 patients with subdural hematomas given CT scans; "in 100% of patients with acute symptoms the subdural hematomas were of increased density; in 70% of the subacute clinical group, they were isodense, while in 76% of the chronic variety they were hypodense").

281. *Id.*

their age.²⁸²

Although mixed density subdural hematomas are often attributed to more than one abusive event, this is not necessarily the case. A rapidly accumulating acute subdural hematoma may appear as mixed density, as may one mixed with cerebrospinal fluid.²⁸³ Some subdural hematomas (regardless of inciting events) continue to grow over time. In these subdural hematomas, both acute and subacute hematomas may be found within the chronic hematoma. Fresh blood clots may be found within a subdural hematoma of significant age (greater than one month), yet there is usually no history of recurrent trauma.²⁸⁴ The mechanism for this has been hypothesized to be the consequence of chronic rebleeding within the subdural hematoma possibly due to disruption of a fragile reovascular capillary network which develops within the subdural as well as other contributing factors. The most important of these factors is abnormalities of clot formation within the subdural hematoma. Rebleeding into a chronic subdural hematoma does not necessarily imply that recent significant trauma (intentional or unintentional) has occurred.²⁸⁵ It may only take minimal trauma, and possibly only a significant valsalva (which may occur from coughing or straining with defecation), to rebleed into an older, unresolved subdural hematoma. The clinical course is helpful in resolving this issue.

Magnetic resonance imaging (MRI) is a more recently developed diagnostic tool.²⁸⁶ In many ways, MRI is superior to CT. MRI provides much greater structural

282. See Gilles, *Abusive Head Injury in Children*, *supra* note 107, at 364 ("These guidelines are not always accurate if the infant is very anemic, there is a mix of cerebrospinal fluid with blood or there are different ages of subdural hematoma.").

283. J. Greenberg et al., *The "Hyperacute" Extra-Axial Intracranial Hematoma: Computer Tomographic Findings and Clinical Significance*, 17 *NEUROSURGERY* 48, 56 (1985); Anna B. Kelly et al., *Head Trauma: Comparison of MR and CT-Experience in 100 Patients*, 9 *AJNR* 699 (1988).

284. Daniel A. Crooks, *Pathogenesis and Biomechanics of Traumatic Intracranial Hemorrhages*, 418 *VIRCHOW ARCHIV A PATHOLOGICAL ANATOMY & HISTOPATHOLOGY* 479 (1991).

285. Crooks, *supra* note 284.

286. The following primer on MRI is based on WILLIAM G. BRADLEY, JR. ET AL., *MAGNETIC RESONANCE IMAGING OF THE BRAIN, HEAD, AND NECK* (1985) and Robert R. Edelman & Steven Warach, *Magnetic Resonance Imaging (First of Two Parts)*, 328 *NEW ENGLAND J. MED.* 708 (1993). Dr. Gilles did not participate in the drafting of this footnote. In magnetic resonance imaging (MRI), radio waves are applied to tissue within a magnetic field. The image reflects the density of protons (specifically, protons in hydrogen nuclei) as affected by the radio waves and the magnetic field. Proton densities vary depending upon the type of substance within which they are found. When protons are placed within a magnetic field they become magnetized—i.e., their vector sum (the "magnetization") is oriented along the main magnetic field. This is called "longitudinal magnetization." When short bursts of radio waves are applied, the magnetization flips 90 degrees. This is called "transverse magnetization." In magnetic resonance imaging, a series of radio wave bursts are applied, and the intensity and frequency of the substance's transverse magnetization are measured. Substances with more intense transverse magnetization appear brighter (are "hyperintense") on the MRI image. The frequency of a substance's transverse magnetization in the presence of a magnetic field gradient allows for its localization.

Several factors influence the amount of transverse magnetization that different substances exhibit at different times. These factors can be manipulated so that different substances can be distinguished on an MRI scan. After a burst ends, the protons move back towards their original orientation (back to longitudinal magnetization). Some substances take longer than others to recover longitudinal magnetization (a process called relaxation). The amount of time a substance requires to recover longitudinal magnetization is called its T1 relaxation time. If a new burst

detail than CT scans. It is generally more sensitive in identifying smaller convexity subdural hematomas, brain contusions, and other shearing injuries.²⁸⁷ MRI allows for greater visualization of the posterior fossa (the area under the tentorium) and the brain stem. On the other hand, CT scans are often superior for detecting subarachnoid hemorrhages.²⁸⁸

of radio waves is applied before a substance has recovered its longitudinal magnetization, this limits the magnitude of the resultant transverse magnetization of that substance.

The amount of time between successive bursts of radio waves is referred to as the repetition time (TR). With a short repetition time (short TR) substances that take a long time to recover their longitudinal magnetization will exhibit less transverse magnetization following a new burst of radio waves than substances that recover their longitudinal magnetization quickly. In other words, with a short repetition time (short TR), substances with long T1 relaxation times will produce less transverse magnetization than substances with short T1 relaxation times. With a long repetition time (long TR), most all substances will have recovered their longitudinal magnetization, and, all else being equal, will exhibit the same amount of transverse magnetization with the application of a new burst of radio waves. In sum, short TR means substances with short T1 relaxation times will produce more transverse magnetization. Long TR means T1 relaxation time differences are unimportant.

With each new burst, the amount of transverse magnetization exhibited by substances decays. Some substances decay more quickly than others. The speed with which a substance's transverse magnetization decays is called its T2 relaxation time. Substances whose transverse magnetization signal decays quickly have shorter T2 relaxation times. If one delays in measuring the transverse magnetization (rather than measuring it after the initial burst), the signal will be weaker.

The amount of time between the initial burst and the measurement of the transverse magnetization signal is referred to as the echo delay time (TE). With a short echo delay time (short TE), substances will not have decayed, and all else being equal, will exhibit the same amount of transverse magnetization. With a long echo delay time (long TE), substances with a short T2 relaxation time will exhibit less transverse magnetization than substances with a long T2 relaxation time. In sum, long TE means substances with long T2 relaxation times will produce more transverse magnetization. Short TE means T2 relaxation time differences are unimportant.

TR and TE can be selected so that substances can be differentiated on the basis of their T1 relaxation and T2 relaxation times. T1-weighted images capitalize on differences among substances' T1 relaxation times, and T2-weighted images capitalize on differences among substances' T2 relaxation times. If TR is short and TE is short, then one obtains a "T1-weighted image," i.e., one in which most of the contrast depends on differences in T1. Remember that short TR means that substances with short T1 times will produce more transverse magnetization, and that short TE means that T2 times are irrelevant. In T1-weighted images, substances with relatively short T1 times appear hyperintense (they are brighter than substances with longer T1 times). If TR is long and TE is long, then one obtains a T2-weighted image. Long TR means that T1 times are irrelevant and long TE means that substances with long T2 times will produce more transverse magnetization. In T2-weighted images, substances with relatively long T2 times appear hyperintense (bright).

It is also possible to create what is called a proton-density weighted image, if TR is long and TE is short. Making TR long and TE short ensures that both T1 times and T2 times are irrelevant, and therefore what affects the image is the substances' hydrogen or proton density.

287. See Launius et al., *supra* note 137, at 44 (MRI superior to CT in detecting nonhemorrhagic contusions of the white matter and other deep cerebral injuries); Yutaka Sato et al., *Head Injury in Child Abuse: Evaluation with MR Imaging*, 173 RADIOLOGY 65, 654 (1989) (19 abused children with head injuries, 79% less than one year of age; MRI superior to CT in detecting subdural hematomas, bland contusions, and shearing injuries); Robert D. Zimmerman et al., *Acute Intracranial Hemorrhage: Intensity Changes on Sequential MR Scans at 0.5T*, 150 AJR 651, 661 (1988) [hereinafter Zimmerman, *Acute Intracranial Hemorrhage*] ("[B]ecause of the absence of bone artifacts and the excellent contrast between brain and adjacent spinal fluid, small, extra[-]axial hematomas are detected much more easily on MR than on CT . . .").

288. Edelman & Warach, *supra* note 286, at 712; see Sato et al., *supra* note 287, at 654 (19 abused children with head injuries, 79% less than one year of age; CT superior to MRI in detecting subarachnoid hemorrhage); Zimmerman, *Acute Intracranial Hemorrhage*, *supra* note 287, at 661 (37 patients with intracranial bleeding; "[s]ubarachnoid hemorrhage is difficult to visualize on MR, not only because it fails to alter intensity, but also

Substances appear differently depending upon whether an MRI image is T1-weighted or T2-weighted, two of the more useful imaging sequences available.²⁸⁹ On T1-weighted MRI images, white matter is hyperintense (bright), gray matter is moderately hypointense and cerebrospinal fluid is hypointense. Edema, infarctions, and tumors generally appear hypointense,²⁹⁰ and diffuse axonal injury appears diffusely hypointense.²⁹¹

On T2-weighted MRI images, white matter is moderately dark, gray matter moderately bright, and cerebrospinal fluid very hyperintense.²⁹² Edema, infarctions, and tumors all appear hyperintense.²⁹³ Unlike T1-weighted images, T2-weighted images are not sensitive to diffuse axonal injury in the infant, due to the relatively high water content of the infant's brain, which gives the brain a diffusely bright appearance.²⁹⁴

Subdural hematomas and intracerebral hemorrhages have different appearances depending upon their age.

TABLE 1²⁹⁵

	T1-W1*	T2-W1*
Hyperacute (4-6 hours)	isointense	hyperintense
Acute (7-72 hours)	isointense	mixed central hypointense, rim hyperintense
Early Subacute (4-7days)	mixed central hypointense, rim hyperintense	mostly hypointense
Late Subacute (1-4 weeks)	hyperintense	hyperintense
Chronic (1 month and older)	hypointense	hypointense

* Major Imaging Pattern

because it fails to produce anatomic distortion").

289. For a technical explanation of these terms, see *supra* note 286.

290. Edelman & Warach, *supra* note 286, at 710.

291. Sato et al., *supra* note 287, at 656.

292. Edelman & Warach, *supra* note 286, at 710.

293. *Id.*; see Bruce & Zimmerman, *supra* note 138, at 487 (edema and infarction hyperintense because of high water content).

294. Sato et al., *supra* note 287, at 657.

295. ANNE G. OSBORN, INTRACRANIAL HEMORRHAGE IN DIAGNOSTIC NEURORADIOLOGY 154-98 (1994).

VI. CONCLUSION

Better understanding of the facts behind physicians' judgments has the paradoxical effect of both increasing and decreasing one's confidence in those judgments. On the one hand, one appreciates the fact that the physician's opinion is based on both an understanding of the mechanisms by which injuries occur and an awareness of the probabilities of various causes drawn from research. On the other hand, one's confidence may be decreased when one recognizes the margin of error within which physicians' operate, making it impossible to always be able to state with certainty that abuse occurred.

We believe, however, that knowledgeable evaluation is always superior to naive trust or impulsive skepticism. Attorneys will never know as much as the physicians upon whom they rely for expert advice. Nevertheless, the attorney who has some understanding of both the strengths and weaknesses of the expert's opinion is in a much better position to effectively translate that opinion into testimony that paints a compelling picture for the trier of fact.

