COMMONWEALTH OF MASSACHUSETTS

ESSEX, MIDDLESEX

SUPREME JUDICIAL COURT
NOS. SJC-11921, 11928

COMMONWEALTH

V.

DERICK EPPS

AND

OSWELT MILLIEN,
APPELLANTS

APPEAL FROM JUDGMENTS OF THE
ESSEX AND MIDDLESEX SUPERIOR COURTS

BRIEF AND APPENDIX OF CPCS, ACLUM, AND MACDL AS AMICI CURIAE

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Issues Presented

In these cases the Court solicited amicus briefs addressing the following issues:

> Epps: "Whether the defendant, tried and convicted in 2007 on a charge of assault and battery on a child causing substantial bodily injury, see G. L. c. 265, § 13J (b), second par., is entitled to a new trial because of newly discovered evidence (i.e., changes since the time of trial in the state of the scientific research on shaken baby syndrome and abusive head trauma) or on the ground of ineffective assistance of counsel (i.e., counsel's alleged failure to use to the defendant's advantage the state of the research as it existed at the time of trial)."
>
> Millien: "Whether the defendant, tried and convicted in 2010 on charges of assault and battery on a child causing bodily injury and substantial bodily injury, see G. L. c. 265, § 13J (b), first and second pars., was denied the effective assistance of counsel where his trial counsel did not consult an expert on shaken baby syndrome; whether the judge erred in denying the defendant's motion for a new trial, supported by expert testimony challenging the evidence at trial of shaken baby syndrome, where, despite finding subpar performance by counsel in failing to consult an expert, the judge ruled that the same did not deprive the defendant of a substantial ground of defense, given the powerful medical evidence presented by the Commonwealth."

Statements of Interest of the Amici Curiae

CPCS

The Committee for Public Counsel Services (CPCS), the Massachusetts public defender agency, is statutorily mandated to provide counsel to indigent defendants in criminal proceedings. The issues raised

in these cases concern whether, in a case in which an infant has allegedly been the victim of physical abuse and diagnosed with shaken baby syndrome (SBS), otherwise known as abusive head trauma (AHT), 1) trial counsel may render ineffective assistance by failing to consult with experts in the field, and 2) whether post-trial advances in the science may warrant a new trial based on newly discovered evidence. These issues are of immediate importance to CPCS because CPCS has and will represent clients accused of causing head trauma in children.[1]

It is in the interest of CPCS' clients, and the fair administration of justice, that CPCS' views be presented in order to contribute to this Court's full consideration of all aspects of the important issues raised in these cases. M.R.A.P. 17.

ACLUM

The American Civil Liberties Union of Massachusetts ("ACLUM"), an affiliate of the national American Civil Liberties Union, is a statewide nonprofit membership organization dedicated to the principles of liberty and equality embodied in the

[1] Trial counsel in the Epps case was a CPCS staff attorney. To avoid the potential appearance of a conflict of interest, this brief will not specifically address whether the Court should grant new trials to these defendants. Instead, it will discuss the evolution of the science regarding SBS and how that may give rise to well-founded claims of ineffective assistance of counsel and newly discovered evidence.

constitutions and laws of the Commonwealth and the United States. Through both direct representation and amicus briefs, ACLUM has participated in cases about wrongful convictions and the fair administration of justice. See, e.g., Bridgeman v. District Attorney for the Suffolk Dist., 471 Mass. 465 (2015); Commonwealth v. Scott, 467 Mass. 336 (2014); Commonwealth v. Charles, 466 Mass. 63 (2013).

MACDL

The Massachusetts Association of Criminal Defense Lawyers (MACDL), as amicus curiae, submits this brief in support of the defendants. MACDL is an incorporated association representing more than 1,000 experienced trial and appellate lawyers who are members of the Massachusetts Bar and who devote a substantial part of their practices to criminal defense. MACDL devotes much of its energy to identifying, and attempting to avoid or correct, problems in the criminal justice system. It files amicus curiae briefs in cases raising questions of importance to the administration of justice.

Summary of the Argument

These defendants were convicted of assault and battery on a child causing substantial bodily injury on the basis of diagnoses of SBS made by members of

the Children's Hospital Child Protection Program.[2] In each case the children had a subdural hemorrhage between the skull and the brain, retinal hemorrhages in both eyes, and brain swelling. In each case, the experts testified that those injuries were caused by shaking and that they were characteristic of SBS.[3] Pp. 25-29.

Shaking was first proposed as a mechanism for causing brain injury in children in 1971. Over the next decade that suggestion was greatly expanded. What came to be known as shaken baby syndrome could be diagnosed, it was believed, when an infant had subdural and retinal hemorrhages and direct injury to the brain, and there were no alternative explanations for the injuries comparable to a severe car accident or a fall from a multi-story building. It was further thought that the child would immediately lose consciousness on sustaining the injuries, and therefore the perpetrator had to be someone who was with the child at that time. Pp. 5-10.

Since the mid-1980s, studies have been published that cast doubt on the SBS theory. In particular, studies have questioned whether shaking alone can

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[2] Millien was also convicted of assault and battery on a child causing bodily injury on the basis of other injuries to the child. That conviction will not be addressed here.

[3] In Millien,the baby also had a skull fracture, an injury caused by impact, not shaking.

generate enough force to cause the injuries attributed to SBS, whether those injuries can be caused by accidental short falls or certain medical conditions, and whether a child sustaining such injuries can have a lucid interval before losing consciousness. The controversy led the American Academy of Pediatrics to recommend a change in terminology for what were believed to be non-accidental head injuries in children - from shaken baby syndrome to abusive head trauma. Pp. 10-24.

The evolution in the understanding of the causes of brain injuries in children may lead to claims of ineffective assistance on the ground that defense counsel failed to adequately investigate the state of the science at the time a client is charged with inflicting such an injury. Similarly, post-trial advances in the field may give rise to claims that newly discovered evidence warrants a new trial. This brief discusses the evolution in scientific understanding and how that evolution may give rise to well-founded claims of ineffective assistance and newly discovered evidence. Pp. 34-49.

Argument

I. THE SCIENTIFIC UNDERSTANDING OF THE CAUSES OF INFANT HEAD INJURY HAS EVOLVED OVER THE LAST 45 YEARS.

A. The Origins of the SBS Theory

A paper written in 1971 was the first to suggest

that shaking might cause a subdural hemorrhage in an infant. Guthkelch, *Infantile Subdural Haematoma and its Relationship to Whiplash Injuries*, 2 BRIT. MED. J. 430 (1971) (A. 110-11)[4].[5] Dr. Guthkelch reported that in a three year period a hospital had 13 cases "of proved or strongly suspected parental assault on children all under the age of 3 years" in which the child had a subdural hemorrhage, seven with no skull fracture and five of those with no external sign of injury. A. 110. He proposed shaking as a mechanism that might cause subdural hemorrhage without any external sign of injury. He theorized that shaking would create a whiplash action that could tear bridging veins that cross the subdural space between the skull and the brain. Id. He stated, "one has the impression that 'a good shaking,' is felt, at least by British parents, to be socially more acceptable and physically less dangerous than a blow on the head or elsewhere." A. 111. He concluded:

> One must keep in mind the possibility of assault in considering any case of infantile subdural haematoma, even when there are only trivial bruises or indeed no marks of injury at all; and inquire, however guardedly or tactfully, whether perhaps the baby's head could have been shaken.

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[4] Appendix citation format: Appendix to this brief, A.; Epps Appendix, EA.; Millien Appendix, MA.

[5] A diagram of the membranes and intervening cavities, including the subdural cavity, between the skull and the brain is at A. 208.

Id. (emphasis added).

In 1972 John Caffey reported on 27 "convincing recorded examples of pathogenic and even fatal shaking," and he concluded that "whiplash shaking" could cause subdural and retinal hemorrhages. Caffey, *On the Theory and Practice of Shaking Infants*, 124 AM. J. OF DISEASES OF CHILDREN 161, 163-64, 169 (1972) (A. 28, 30-31,36). In a follow-up article he coined the term "whiplash shaken infant syndrome" to explain findings of subdural and retinal hemorrhages "in the absence of signs of external trauma to the head or fractures of the calvaria [skull]" and where "there is no history of trauma of any kind." He noted, however, that "current evidence" was "manifestly incomplete and largely circumstantial." 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, 402-3 (1974) (A. 37,43-44).

One of the earliest uses of the term "shaken baby syndrome" appears to be in Ludwig & Warman, *Shaken Baby Syndrome: A Review of 20 Cases*, 13 ANNALS OF EMERGENCY MED. 104 (1984) (A. 141-43). One of the authors' goals was "to increase the emergency physician's suspicion for and ability to detect this form of child abuse." A. 141.

> All official child abuse reporting forms sent to the County Child Protective Services Agency from the Children's Hospital of Philadelphia during the years 1977 to 1982 were reviewed. Cases of possible shake injury were selected based on parent's admission of shaking or suspicion by medical staff of this form of abuse when the history and evaluation could not account for the patient's injuries.

Id. (emphasis added) The authors found 20 cases that fit their criteria, and in only three were there admissions of shaking, each "in an effort to perform resuscitation." A. 142. The authors stated that imaging could "confirm SBS by showing acute interhemispheric subdural hematoma or cerebral contusion in the absence of external trauma." A. 143.

Eventually what became widely known as SBS was thought to be characterized by three injuries - subdural and retinal hemorrhages and injury to the brain itself. The theory holds that when an infant is held by the chest, arms, or legs and shaken, the head swings back and forth, accelerating and decelerating, causing the brain to move inside the skull. That action creates shearing forces that 1) tear bridging veins that cross the subdural space resulting in subdural hemorrhage, 2) directly injure the brain by tearing axons, impairing the transmission of electrical signals from nerve cells, and 3) cause retinal hemorrhages by damaging veins in the retina, either due to direct tearing forces or due to

increased pressure caused by the swelling brain. It was thought that serious injuries would cause an immediate loss of consciousness, and that, absent evidence of impact comparable to that caused by a serious car accident or multi-story fall, the injuries had to be caused by shaking. Committee on Child Abuse and Neglect, Am. Acad. of Pediatrics, *Shaken Baby Syndrome: Rotational Cranial Injuries - Technical Report*, 108 PEDIATRICS 206, 206-208 (2001) (EA 433-35); Case et al., P*osition Paper on Fatal Abusive Head Injuries in Infants and Young Children*, 22 AM. J. FORENSIC MED. AND PATHOLOGY 112, 115-18, 120-21 (2001) (EA 940-44, 946-47).

Prosecutors rapidly made use of the SBS theory. In State v. Lane, 39 N.C.App. 33, 34-35, 37 (1978), an appeal decided only a few years after the Guthkelch and Caffey articles appeared, an involuntary manslaughter conviction was affirmed where the defendant stated that he had shaken a baby and the medical examiner testified that the shaking made the baby's head move "in a whiplash fashion" causing subdural hemorrhage. State v. Schneider, 1984 WL 3719 (Ohio App. 1984), an unpublished decision, appears to be the first appellate decision referring to shaken baby syndrome. There, too, the court affirmed an involuntary manslaughter conviction. State v. Mallar, 127 N.H. 816, 817 (1986) is the first published

decision using the term "shaken baby syndrome."[6] In that case the court affirmed a negligent homicide conviction. Id. at 822.

B. Challenges to the SBS Theory

1. Many Studies Supporting the SBS Hypothesis Have Employed a Flawed Methodology.

Guthkelch and Caffey did not prove that shaking causes subdural and retinal hemorrhages in children. Rather, they knew that some parents shake their children as a form of discipline, and they theorized that shaking might be a cause of those injuries in some cases.

Since then, many efforts have been made to verify the SBS hypothesis. One obvious hurdle, though, is that it would be unethical to run experiments shaking healthy infants with varying degrees of force and measuring the effects. In an effort to overcome that hurdle, though, many researchers have employed flawed methods.

Some researchers have cherry-picked data to support the SBS hypothesis. For example, one article, noting the importance of differentiating accidental from inflicted injuries in children, sought to

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[6] This Court first used the term in Commonwealth v. Avellar, 416 Mass. 409, 414 (1993), a first degree murder case in which the medical examiner "concluded that the injuries were not consistent with 'shaken baby syndrome.'"

determine "whether or not infants and children can receive fatal injuries in short falls" by examining hospital records of children reported to have fallen from various heights. Faced with an unexpectedly large number of deaths after reported short falls, the authors simply assumed those reports were false. Chadwick et al., *Deaths from Falls in Children: How Far is Fatal?*, 31 J. TRAUMA 1353, 1353, 1355 (1991) (A. 58,58,60).

Another flaw in many studies is the use of circular reasoning. An example of that arises in an article that sought to determine whether children who die of head injuries might have a lucid interval before losing consciousness. Included in the cases reviewed were those with "diagnoses of child abuse, suspected child abuse," and others where abuse was not suspected. Those cases that had the injuries that fit within the SBS hypothesis were defined as shaking cases. With that definition in place, the authors proceeded to conclude that shaking would be less likely to result in a lengthy lucid interval than other causes of head injury. Gilliland, *Interval Duration Between Injury and Severe Symptoms in Nonaccidental Head Trauma in Infants and Young Children*, 43 J. FORENSIC SCI. 723, 723-24 (1998) (A. 103,103-4). The paper by Ludwig, supra (A. 141), is similarly flawed: the authors selected cases based on

the assumption that certain brain injuries are evidence of shaking, and they concluded that SBS is confirmed when those injuries are found.

A 2003 paper applied the techniques of evidence-based medicine to articles about SBS that had been published through 1998. Evidence-based medicine evaluates the quality of evidence supporting a theory. Theories with quality-of-evidence rating I(QER I) have the strongest support:

> Consistent evidence obtained from more than 2 independent, randomized, and controlled studies or from 2 independent, population-based epidemiologic studies. Studies included here are characterized by sufficient statistical power, rigorous methodologies, and inclusion of representative patient samples. Meta-analysis of smaller, well-characterized studies may support key findings.

Donohoe, *Evidence-Based Medicine and Shaken Baby Syndrome Part I: Literature Review, 1966-1998*, 24 AM. J. FORENSIC MED. PATHOLOGY 239, 240 (2003) (A. 64,65). Theories with QER IV have the weakest support: "Consensus opinions of authorities according to clinical experience or descriptive reports." Id. The author found:

> There was no evidence on the subject of SBS that exceeded QER III-2[7] by the end of 1998, which means that there was inadequate scientific evidence to come to a firm conclusion on most aspects of causation, diagnosis, treatment, or

[7] Category III is divided into four subcategories, III-1 to III-4. A. 65.

> any other matters pertaining to SBS. The majority of evidence achieved only a level of QER IV, opinions that shed no new light upon SBS and did not add to knowledge about SBS. Many of the authors repeated the logical flaw that if RH [retinal hemorrhage] and SDH [subdural hemorrhage] are nearly always seen in SBS, the presence of RH and SDH "prove" that a baby was shaken intentionally. Many other studies assumed that the presence of RH and SDH was sufficient to make the diagnosis of SBS in terms of case selection.

A. 66. The author concluded:

> Before 1999, there existed serious data gaps, flaws of logic, inconsistency of case definition, and a serious lack of tests capable of discriminating NAI [non-accidental injury] cases from natural injuries. . . The commonly held opinion that the finding of SDH and RH in an infant was strong evidence of SBS was unsustainable, at least from the medical literature.

Id.

2. Biomechanical Modeling Studies Have Found That Shaking Is No More Likely to Cause the Injuries Attributed to SBS Than Short Falls, and Impact Would Be a Much More Likely Cause.

Biomechanical studies were the first to raise doubts about the reliability of the SBS theory. One early study had adults violently shake models of a one-month-old infant and then hit them against an object. The results showed that the acceleration caused by violent shaking was fifty times less than that caused by impact, and that it was well below the level believed necessary to cause brain injuries.

Duhaime et al., *The Shaken Baby Syndrome: A Clinical, Pathological, and Biomechanical Study*, 66 J. NEUROSURGERY 409, 413-14 (1987) (A. 68,72-73).

In the ensuing years researchers have attempted to develop models that would more accurately reflect the behavior of an infant head and neck. A 2003 study used models of a 1.5-month-old infant and compared the forces generated by vigorous shaking, inflicted impact after shaking onto a foam mattress, carpet, and stone bench, and falls of 1-5 feet onto the mattress, carpet, and a concrete floor. It found that shaking generated a similar amount of force as inflicted impact and falls onto a foam mattress, but the forces generated by falls from any height and inflicted impact onto carpet or a hard surface were much greater. The authors then compared their data to injury thresholds reported in other studies, and found support for the conclusion that falls from as little as one foot onto a hard surface or from three feet onto carpet could cause subdural hemorrhage and brain injury. Prange et al., *Anthropomorphic simulations of falls, shakes, and inflicted impacts in infants*, 99 J. NEUROSURGERY 143, 146-48 (2003) (MA 221-23). A subsequent study using a model of a heavier child, an 18-month-old toddler, found that the force that could be generated by shaking was ten times less than with a 1.5-month-old infant. The authors concluded, “it may

be difficult to deliver large inertial forces to the toddler head by shaking alone." Ibrahim et al., *The Response of Toddler and Infant Heads During Vigorous Shaking*, 22 J. NEUROTRAUMA 1207 (2005) (A. 135).

The Commonwealth's child abuse expert in Millien cited another biomechanical study. In 2007, researchers reported on a computer simulation comparing shaking to a three meter per second impact. A three meter per second impact is what would be caused by a fall of only 18 inches. A. 169. The computer model attempted to more accurately simulate the separate plates of the infant skull. The simulation indicated that the pressure and shearing stresses on the brain "are significantly lower for shaking than for an impact," but that the strains on the bridging veins were similar. Thus the authors concluded that an 18 inch fall would be as likely to cause subdural hemorrhage as shaking.[8] Roth et al., *Finite Element Analysis of Impact and Shaking Inflicted to a Child*, 121 INT'L J. LEGAL MED. 223, 225 (2007) (A. 163,165).

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[8] The theory that torn bridging veins cause subdural hemorrhages in babies that have been shaken has been cast into doubt by studies finding that the hemorrhages in cases where abuse is suspected are usually thin films, not the massive hemorrhages Caffey suggested would occur. Geddes et al., *Neuropathology of Inflicted Head Injury in Children, Part I. Patterns of Brain Damage*, 124 BRAIN 1290, 1297 (2001) (A. 86, 93).

3. Other Conditions Can Cause the Injuries Attributed to SBS.

The SBS theory is that the combination of subdural and retinal hemorrhages and brain injury must be caused by shaking absent evidence of something comparable to a serious car accident or multi-story fall. Beginning in the late-1980s, however, reports began to appear of other causes for the injuries.

Two early studies confirmed what the biomechanical studies suggested. They reported instances of fatalities in children under two years old who suffered brain injuries after short falls. Reiber, *Fatal Falls in Childhood: How Far Must Children Fall to Sustain Fatal Head Injury? Report of Cases and Review of the Literature*, 14 AM. J. FORENSIC MED. PATHOLOGY 201, 203, 206-7 (1993) (A.156,159-60); Hall et al., *The Mortality of Childhood Falls*, 29 J. TRAUMA 1273, 1274 (1989) (A. 112,113). Another author reviewed data on falls from playground equipment. He found 18 fatalities in children 1-13 years old who fell from 2-10 feet. Most had subdural hemorrhage, four of the six whose eyes were examined had retinal hemorrhages, and 12 of the children had lucid intervals. Plunkett, *Fatal Pediatric Head Injuries Caused by Short-Distance Falls*, 22 AM. J. FORENSIC MED. PATHOLOGY 1, 10 (2001) (A. 144,153). Similar cases have been reported since. See, e.g., Lantz & Couture,

Fatal Acute Intracranial Injury, Subdural Hematoma, and Retinal Hemorrhages Caused by Stairway Fall, 56 J. FORENSIC SCI. 1 (2011) (EA 383).

Other studies have raised doubts about the connection between retinal hemorrhage and shaking. A 1999 article reported the cases of three children, ages 7-13 months, who had retinal hemorrhages after accidents at home, two falling down stairs and the other a short fall while playing. The authors stated:

> Differentiating hemorrhages caused by accidental injury from those caused by abuse may be difficult, because either cause can result in unilateral or bilateral hemorrhages and can involve all layers of the retina.

Christian et al., *Retinal Hemorrhages Caused by Accidental Household Trauma*, 135 J. PEDIATRICS 125, 127 (1999) (A. 61,63); see also Lantz et al., *Perimacular Retinal Folds From Childhood Head Trauma*, 328 BMJ 754 (2004) (14-month-old child had subdural and retinal hemorrhages and cerebral edema after television fell on him) (A. 136). A more recent study examined the eyes of 118 children who had died of known or suspected abuse. In seven of those there had been an admission of shaking. In the only one of those seven in which there was no evidence of blunt force to the head or other part of the body, no retinal hemorrhages were found. Conversely, retinal hemorrhages were found in 47 cases in which there was no evidence of shaking. Emerson et al., *Ocular Autopsy and*

Histopathologic Features of Child Abuse, 114 OPHTHALMOLOGY 1384, 1388, 1391-93 (2007) (A. 75,79,82-84). Retinal hemorrhages have also been found in infants with meningitis. Lopez et al., *Severe Retinal Hemorrhages in Infants with Aggressive Fatal Streptococcus Pneumonia Meningitis*, 14 J. AM. A. PEDIATRIC OPHTHALMOLOGY & STRABISMUS 97 (2010) (A. 139).

There are also many medical conditions that can mimic SBS. A 1990 article reported that retinal hemorrhages in six-week and two-year-old children were caused by the use of CPR in efforts to resuscitate the children. Goetting & Sowa, *Retinal Hemorrhage After Cardiopulmonary Resuscitation in Children*, 85 PEDIATRICS 585, 585-86 (1990) (A. 106,106-7). Another article summarized the wide variety of conditions that have been found to cause subdural hemorrhages, citing articles dating back to 1971. Hymel et al., *Intracranial Hemorrhage and Rebleeding in Suspected Victims of Abusive Head Trauma: Addressing the Forensic Controversies*, 7 CHILD MALTREATMENT 329, 332-337 (2002) (A. 115,118-23). A more recent article noted that brain injury had been found in infants in nontraumatic cases, and it summarized the wide variety of conditions known to cause subdural and retinal hemorrhages, including neonatal and developmental abnormalities, genetic, metabolic, and blood disorders, and infectious diseases, to name a few.

Barnes & Krasnokutsky, *Imaging of the Central Nervous System in Suspected or Alleged Nonaccidental Injury, Including the Mimics*, 18 TOPICS IN MAGNETIC RESONANCE IMAGING 53, 55-56, 67-70 (2007) (A. 6,8-9,20-23).

The SBS theory postulates that shaking causes brain injury primarily by generating traumatic shearing forces that tear axons throughout the brain. That theory was cast into doubt by a study that reviewed records of infants believed to have died as a result of inflicted head injury. It found that they did have damaged axons, but it was not the traumatic damage proposed by the SBS theory; rather, it was damage caused by lack of oxygen and the resulting pressure caused by the swelling brain. Geddes et al., *Neuropathology of Inflicted Head Injury in Children, II. Microscopic Brain Injury in Infants*, 124 BRAIN 1299, 1304 (2001) (A. 95,100). There are many potential causes of loss of oxygen to the brain, though, and rather than being immediately symptomatic, symptoms may develop only over time as the loss of oxygen continues and the brain damage increases. Squier, *Shaken Baby Syndrome: The Quest for Evidence*, 50 DEVELOPMENTAL MED. & CHILD NEUROLOGY 10, 11-12 (2008) (EA 328-29).

4. A Child With the Injuries Attributed to SBS May Have a Lucid Interval Before Symptoms Become Apparent.

According to the SBS theory, symptoms will become

immediately apparent when the injuries attributable to it are inflicted, and therefore the perpetrator must be a person who was with the child at that time. Studies have shown, however, that a child with these injuries may have a lucid interval before symptoms appear. Plunkett, supra (A. 145); Gilliland, supra (A. 104).[9] A more recent study found 314 cases of fatal head injuries in children under four over a 17 year period, and it compared the likelihood of a lucid interval for cases that had been classified as due to inflicted injury, falls, and car accidents. It found that lucid intervals occurred in all three categories, and the likelihood of being lucid or moderately conscious was greatest in cases classified as due to inflicted injury, 10.7%. Arbogast et al., *Initial Neurologic Presentation in Young Children Sustaining Inflicted and Unintentional Fatal Head Injuries*, 116 PEDIATRICS 180, 181-83 (2005) (A. 1,2-4).

C. Reacting to the Challenges

Despite the questions that had been raised about the SBS theory, in 2001 the American Academy of Pediatrics (AAP) adopted a position paper that

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[9] Even with their biased definition of "shaking," p. 11, the Gilliland study found that lucid intervals occur. They found five cases that fell within their definition of shaking alone. In four, the lucid interval was up to 24 hours and in the other it was between one and two days. In at least one case they defined as a combination of shaking and impact, the lucid interval was over three days. A. 104.

accepted the theory without qualification. Committee on Child Abuse and Neglect, supra (EA 433). It noted that shaking causes subdural and retinal hemorrhages and cerebral edema, in severe cases "the child usually becomes immediately unconscious," and such injuries "are rarely accidental unless there is another clear explanation, such as trauma from a motor vehicle crash." EA 433-35. It rejected the possibility that these injuries could occur "with short falls, seizures, or as a consequence of vaccination." EA 433. It concluded that there should be a "presumption of child abuse when a child younger than 1 year has suffered an intracranial injury." Id.

In 2009 the AAP issued a new position paper which acknowledged that 1) biomechanical modeling had raised doubts as to whether shaking alone could cause the injuries that had been attributed to SBS, and 2) impact and certain medical conditions could cause those injuries. Christian, et al., *Abusive Head Trauma in Infants and Children*, 123 PEDIATRICS 1409, 1409 (2009) (EA 439). The AAP no longer claimed that short falls could not cause the injuries. The new paper stated:

> Controversy is fueled because the mechanisms and resultant injuries of accidental and abusive head injury overlap, the abuse is rarely witnessed, an accurate history of trauma is rarely offered by the perpetrator, there is no single or simple test to determine the accuracy of the diagnosis, and the legal consequences of the diagnosis can

be so significant.

EA 440. It noted, "The term [SBS] is sometimes used inaccurately to describe infants with impact injury alone or with multiple mechanisms of head and brain injury and focuses on a specific mechanism of injury rather than the abusive event that was perpetrated against a helpless victim." Id. Consequently, the AAP deemed it necessary to eliminate the focus on shaking as the mechanism of injury. "To broaden the terminology," the paper recommended abandonment of the phrase "shaken baby syndrome" in favor of "the term 'abusive head trauma' as the diagnosis used in the medical chart to describe the constellation of cerebral, spinal, and cranial injuries that result from inflicted head injury to infants and young children." Id.

In 2012, Dr. Guthkelch, the author of the original 1971 article that first suggested shaking as a possible cause of subdural hemorrhage in children, published a new paper in which he explained how that article had been misinterpreted. Guthkelch, *Problems of Infant Retino-Dural Hemorrhage With Minimal External Injury*, 12 HOUSTON J. HEALTH L. & POLICY 201 (2012) (EA 486). His 1971 paper had described two cases in which shaking, with no malicious intent, might have caused subdural hemorrhages. In his 2012 paper, he stated:

> While these events may have triggered a subdural hemorrhage or rehemorrhage, it is unwarranted to go from this possibility to the assumption that unexplained subdural hemorrhages, with or without retinal hemorrhage or encephalopathy, are caused by violent shaking or other forms of abuse.

EA 491. He pointed out, "subdural and retinal hemorrhages (with or without cerebral edema) may also be observed in accidental or natural settings." EA 487.

> While society is rightly shocked by any assault on its weakest members and demands retribution, there seem to have been instances in which both medical science and the law have gone too far in hypothesizing and criminalizing alleged acts of violence in which the only evidence has been the presence of the classic triad or even just one or two of its elements. Often, there seems to have been inadequate inquiry into the possibility that the picture resulted from natural causes.

EA 488-89. He stated that due to the complexity of the infant brain:

> we should not expect to find an exact or constant relationship between the existence or extent of retino-dural hemorrhage and the amount of force involved, let alone the state of mind of the perpetrator. Nor should we assume that these findings are caused by trauma, rather than natural causes.

EA 489. He concluded:

> "Getting it right" requires that we distinguish between hypotheses and knowledge. SBS and AHT are hypotheses that have been advanced to explain findings that are not yet fully understood. There is nothing wrong in advancing such hypotheses; this is how medicine and science progress. It is wrong, however, to fail to advise parents and courts when these are simply hypotheses, not

> proven medical or scientific facts, or to attack those who point out problems with these hypotheses or who advance alternatives.

EA 492.

D. Recent Survey Articles in Law Journals Have Brought These Issues to Wider Attention in the Legal Community.

In the last few years, survey articles discussing the SBS controversy have appeared in law journals. Findley et al., *Shaken Baby Syndrome, Abusive Head Trauma and Actual Innocence: Getting It Right*, 12 HOUSTON J. HEALTH LAW & POLICY 209, 230 (2012) (EA 718); Imwinkelried, *Shaken Baby Syndrome: A Genuine Battle of the Scientific (and Non-Scientific) Experts*, 46 CRIM. L. BULL. 156 (2010) (EA 627); Tuerkheimer, *The Next Innocence Project: Shaken Baby Syndrome and the Criminal Courts*, 87 WASH. U. L. REV. 1 (2009) (EA 268). These articles have brought wider attention in the legal community to the controversy surrounding SBS. As will be seen below, this increased attention has led to rising numbers of challenges to the SBS theory in courts.

II. THE OPINIONS OF THE EXPERTS IN THESE CASES HAVE EVOLVED AND BEEN CHALLENGED OVER THE YEARS.

A. The Expert Evidence in These Cases

The opinions expressed by the Children’s Hospital experts in these cases evolved in conjunction with the

changing positions of the AAP.[10]

1. Epps

The Epps case was tried in 2007, two years before the AAP acknowledged that there could be causes other than shaking for the injuries that had been attributed to SBS and recommended use of the term AHT in lieu of SBS.

When two-year-old Veronica Gonzalez arrived at Children's Hospital on October 9, 2004, she was unresponsive. She had subdural and subarachnoid hemorrhages, extensive brain swelling, and retinal hemorrhages in both eyes. Surgery was performed to relieve the brain swelling. It was reported that she had fallen down stairs and from a stool that day. ET 4/42,55-56,61-63,66,99,104.

Veronica was seen after the surgery by Dr. Celeste Wilson, a pediatrician who had been working with the hospital's child protection program since 1998 and was its associate medical director at the time of trial. Wilson reviewed emergency records and spoke to the surgeon. She was aware of the reported falls, though she was unable to speak to the parents. ET 4/37-38,43,53-54,97-9.

Wilson testified that the injuries were recent and they were consistent with SBS. She described SBS

[10] The transcripts are cited as ET (Epps trial), MT (Millien trial), and MM (hearing on Millien motion for a new trial). Pages are cited as Volume #/Page #.

as a diagnosis based on findings of subdural and retinal hemorrhages, and that there may or may not be bruises or fractures. She testified that shaking creates extreme acceleration and deceleration which tears bridging veins causing subdural hemorrhage and it tears nerves which release cytokines causing brain swelling. She stated that immediately after injury she would expect a decline in consciousness, possibly vomiting or seizure, and that unconsciousness may develop. The majority of cases where SBS is diagnosed, she agreed, are in children between three and eight months. ET 4/68-71,77,81,84,104,108.

Wilson testified that a high speed car accident in which the victim was ejected or a 70 foot fall could cause head injuries like those present in this case, but in those situations she would expect to see a skull fracture and there was none here. She stated that neither a three foot fall, a fall down stairs, nor normal activities of a toddler would explain the injuries. Tests showed no blood disease that could explain the subdural hemorrhage, and she deemed it unlikely that cumulative injury over time could have caused the injuries. ET 4/78,83,85,87,100.

2. Millien

a. The Trial

This case was tried in 2010, one year after the AAP issued its new position paper.

Six-month old Jahanna Millien was unconscious when she arrived at Children's Hospital on October 20, 2009. She had a subdural hemorrhage, extensive brain swelling, retinal hemorrhages in both eyes, and a comminuted skull fracture (one with multiple branches rather than a single fracture line).[11] She underwent surgery that night to remove blood and relieve pressure on the brain. The parents reported that she had been in normal health until she fell off a couch. At that point she immediately became unconscious and vomited. MT 5/25-27,29-30,33,57,63.

The next day Jahanna was seen by Dr. Alice Newton, the medical director of the hospital's child protection program. Newton spoke to the parents and doctors, and she reviewed the medical records. She was aware of the reported fall the previous day. She was not aware of any other falls in Jahanna's history. She stated that the brain swelling and hemorrhages were acute injuries that had happened within hours. A skull fracture itself cannot be dated, but given the other head injuries and the reported history, Newton believed it was also a recent injury. MT 5/19,21, 25-26,53,65-66.

Newton stated that her opinion to a reasonable

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[11] Imaging also revealed older healing fractures of ribs and the tibia, and vertebral fractures that could not be dated. MT 5/40-41,43,47. Those injuries are not relevant here.

medical certainty was that the subdural and retinal hemorrhages and the brain injury where caused by violent shaking, and that the skull fracture required an impact such as a blow by hand, or by throwing or slamming the child against something. She stated that the hemorrhages and brain swelling "fit the definition of Shaken Baby Syndrome" and "do not have any other medical explanation."[12] MT 5/64,66-67,95.

The syndrome, she said, refers to direct brain trauma and subdural and retinal hemorrhages, though all three are not always present. A person who cannot care for a crying infant may hold the child around the chest and shake violently until the baby loses consciousness or the caretaker may throw the infant down. When an infant is shaken, the brain moves back and forth within the skull, which may bruise brain tissue and damage nerve cells in the brain, vessels from outer layers around the brain are torn causing subdural bleeding, and the vitreous in the eye pulls on the retina causing hemorrhages there. She stated that in a severe case like this, the injury can be timed by noting when the child became very sick after acting normally. MT 5/56-59,61-62,65.

Newton testified that a short fall might cause a

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[12] By that time, she testified, medical literature more often used the terms "Inflicted Traumatic Brain Injury or Abusive Head Trauma" rather than shaken baby syndrome. MT 5/56.

skull fracture and a small subdural hemorrhage in that area, but that it could not explain the subdural and retinal hemorrhages and the brain injury here. Furthermore, she stated that the comminuted skull fracture would be very unlikely to result from the reported short fall. She stated that she assesses any information that may be important to a child's health, including whether there are medical or other risk factors. In this case she noted that Jahanna had previously been seen for two episodes of vomiting with blood and there had been concern about reflux, she was a little behind developmentally but essentially normal, and there was no evidence of a bleeding disorder or massive accidental head injury. MT 5/27-28,70,67-68,73,79.

b. The Motion for a New Trial Hearing

Dr. Newton testified again at the hearing on Millien's motion for a new trial in 2014.

She stated that she suspected abuse right away. Although she had testified at trial that the hemorrhages and brain swelling were caused by shaking, she now testified that "being slammed" contributed to those injuries. She stated that those injuries and the skull fracture were inconsistent with a household fall but were consistent with shaking combined with slamming on a hard surface. She stated that the likely point of impact was on the left side of the

head where the skull was fragmented rather than on the back of the head which was reportedly the point of impact when Jahanna fell. MM 3/31-32,34,51,66-67.

Newton testified that there were three clinical findings commonly ascribed to SBS - "subdural hemorrhage and brain injury and retinal hemorrhaging" - but she stated, "I don't think I would say now that things are that simplistic." MM 2/107; 3/88. She noted that the AAP had adopted the term AHT because studies had shown that impact could also be a source of the injuries. AHT would be diagnosed when there was a brain injury, often coupled with subdural and retinal hemorrhages, and no history of accidental trauma or medical issues that could explain the injuries.[13] She noted that infants are particularly susceptible due to their small size relative to adults, and that it is rare to find a non-infant with injuries similar to SBS. MM 2/106-7,110-11; 3/87-89.

Newton also discussed some of the biomechanical studies. She stated that it is generally accepted that impact generates much greater force than shaking. She did maintain, though, that shaking could cause the injuries attributed to SBS. Interestingly, the study she highlighted to support that position was the one by Roth - the computer simulation that found that

[13] She noted that skull or other fractures, and injuries to the spinal cord and neck might also be found. MM 2/110-11.

shaking would be no more likely than an 18 inch fall to cause subdural hemorrhage. MM 2/116-19; 3/4-12.

Newton discussed another article that reviewed a state database of deaths from 1999-2003 and concluded that the risk of death from a fall of less than five feet was less than one in a million. Chadwick et al., *Annual Risk of Death Resulting From Short Falls Among Young Children: Less Than 1 in 1 Million*, 121 PEDIATRICS 1213 (2008) (MA 227). While the risk may be small, the article confirmed that short falls can cause death. Furthermore, that article may understate the risk of death from a short fall. The time period studied is a period when the SBS theory was widely accepted, before the AAP issued its new position paper in 2009. Cases where caretakers' descriptions of short falls that were rejected at that time might be viewed differently now. Newton acknowledged that the database reviewed by Chadwick would have relied on the doctor's determination as to whether a death was accidental, not the caretaker's description of the incident. MM 3/22-26,71.

B. In the Last Decade There Have Been Cases in Which the Children's Hospital Experts' Opinions Have Been Deemed Insufficient to Prove That Injuries Were Caused by Shaking.

Since 2006, at least one jury and the medical examiner's office have rejected Dr. Newton's diagnoses that injuries were caused by shaking.

In 2003 a three-month old child lost consciousness while she was at Ann Power's home daycare center. Commonwealth v. Power, 76 Mass. App. Ct. 398, 399, 401-2 (2010), rev. denied 456 Mass. 1105 (2010). She died the next day. Id. at 402. Newton diagnosed SBS. A. 170-73. The defense presented a biomechanical expert who testified that another child could have caused the baby's injuries by rocking the seat she was in. 76 Mass. App. Ct. at 403. The Commonwealth argued that the evidence of shaking would support first or second degree murder verdicts, or involuntary manslaughter by battery. Id. at 399. Alternatively, it argued that Power could be convicted of reckless involuntary manslaughter on the theory that she was trying to care for too many children to prevent an accident or injury caused by another child. Id. at 408-9. The jury rejected the shaking theory and convicted Power of reckless involuntary manslaughter. Id. at 399.

In 2010, Geoffrey Wilson was charged the murder after his six-month-old son died. Newton again concluded that shaking was the cause of death and the medical examiner ruled the death a homicide. After defense experts found evidence of a genetic condition that could have caused the child's injuries, though, the medical examiner changed his opinion, ruling that the cause of death could not be determined. In

September 2014, the Commonwealth filed a nolle prosequi. A. 175-80,189-95.

In January 2013, a one-year-old child lost consciousness while in the care of her babysitter, Aisling McCarthy. She died two day later. Newton concluded that she had been shaken based on findings of subdural and retinal hemorrhages and brain injury, and that a bruise on her head was evidence of blunt force. She determined that the case was consistent with AHT on the day she first saw the child - the day after the baby lost consciousness - and she confirmed that diagnosis later that week even though she had never reviewed the child's birth records. After defense experts provided evidence that the child may have had a bleeding disorder, the medical examiner changed the cause of death from homicide to undetermined. The Commonwealth filed a nolle prosequi on August 31, 2015. A. 182-88,199-202.

On October 13, 2015, the Boston Globe published a story in which it quoted notes the medical examiner in the Wilson case had written about his interactions with the district attorney's office while he was reviewing his opinion. In those notes, he wrote, "I told them I felt bullied and at times as though I was being forced to sign the case out in a way I did not think was honest." He described the prosecutors' behavior as "unethical and unprofessional." A. 204-5.

Two days later District Attorney Marian Ryan was interviewed by WBUR.[14] During that interview she acknowledged that Wilson had been arrested shortly after the child was taken off life support, before the medical examiner had issued his initial ruling that the death was a homicide, apparently on the basis of Newton's conclusion that shaking was the cause of death. When pressed toward the end of the interview as to whether she would continue to use Newton as an expert, she stated that going forward:

> We are going to operate differently. . . Going forward I think there needs to be a collaborative effort on behalf of everyone involved in these processes to think through what we're doing. I think peer review is essential. . . I think that perhaps greater use of outside experts early on is important.

III. CASES ALLEGING SBS OR AHT MAY GIVE RISE TO WELL-FOUNDED CLAIMS OF INEFFECTIVE ASSISTANCE OF COUNSEL OR NEWLY DISCOVERED EVIDENCE.

A. Trial Counsel May Render Ineffective Assistance by Failing to Consult With Experts in the Field.

The Sixth Amendment right to the effective assistance of counsel is denied if, in the totality of the circumstances, counsel's performance was

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[14] The interview is at http://wgbhnews.org/post/da-marian-ryan-talks-shaken-baby-syndrome-cases?utm_referrer=http%3A//m.wgbhnews.org/%3Futm_referrer%3Dhttp%253A%252F%252Fcommonwealthmagazine.org%252Fpolitics%252Fas-media-magnet-trump-is-yooge%252F%23mobile/47377. The segments cited here are at 2:40-3:00 and 8:00-9:20.

unreasonable "under prevailing professional norms," and "there is a reasonable probability that, absent the errors," the result would have been different. Strickland v. Washington, 466 U.S. 668, 688, 695 (1984).

Similarly, under art. 12 a new trial is required if counsel renders ineffective assistance that "has likely deprived the defendant of an otherwise available, substantial ground of defence." Commonwealth v. Saferian, 366 Mass. 89, 96 (1974). When considering the prejudice prong, a court must "evaluate the defendant's evidence in the light most favorable to him to determine whether it might have influenced a jury's conclusion." Commonwealth v. Roberio, 428 Mass. 278, 281 (1998). The credibility of the defense evidence is a matter for the jury to decide, and therefore the court must assume the new defense evidence is true when evaluating whether that evidence would be a factor in deliberations in light of the other evidence presented at trial. Id. at 281-82; Commonwealth v. Lang, 473 Mass. 1, 21 n.1 (2015) (Lenk, J.); Commonwealth v. Hampton, 88 Mass. App. Ct. 162, 168, 171 (2015).

"Counsel has a duty to make reasonable investigations or to make a reasonable decision that makes particular investigations unnecessary." Strickland, 466 U.S. at 690-91; Commonwealth v.

Holliday, 450 Mass. 794, 811-12 (2008), cert. denied sub nom. Mooltrey v. Massachusetts, 555 U.S. 947 (2008).

> In assessing the reasonableness of an attorney's investigation, however, a court must consider not only the quantum of evidence already known to counsel, but also whether the known evidence would lead a reasonable attorney to investigate further.

Wiggins v. Smith, 539 U.S. 510, 527 (2003).

"Prevailing norms of practice as reflected in American Bar Association standards and the like . . . are guides to determining what is reasonable." Strickland, 466 U.S. at 688. The ABA standards state that counsel's duty to investigate includes "evaluation of the prosecution's evidence (including possible re-testing or re-evaluation of physical, forensic, and expert evidence)," and that "defense counsel should determine whether the client's interests would be served by engaging . . . forensic . . . or other professional witnesses." ABA Criminal Justice Standards for the Defense Function, Standard 4-4.1 (a,c,d) (4th ed. 2015).

The CPCS Performance Standards Governing Representation of Indigents in Criminal Cases are another relevant guide to what is reasonable. Commonwealth v. Marinho, 464 Mass. 115, 126 n.16 (2013). Those standards state, "Counsel should promptly investigate the circumstances of the case and

explore all avenues leading to facts relevant both to the merits and to the penalty in the event of conviction," Standard IV(A), and, "Among the motions that counsel should consider filing are . . . funds for experts . . . under G.L. c.261, §§27A–D. Counsel should consider retaining experts as consultants to aid in trial preparation, not only as witnesses," Standard IV(G)(8).

In Hinton v. Alabama, 134 S.Ct. 1081 (2014), the Supreme Court applied the standards regarding the duty to investigate in the forensic evidence context. In that case the state's ballistics experts testified that bullets found at two murder scenes had been fired by Hinton's gun. Defense counsel sought funds to retain an independent expert, and the judge allowed what he wrongly believed to be the statutory maximum. Counsel was unable to hire a well-qualified expert with the limited funds available. The expert he was able to hire was subject to withering cross-examination regarding his qualifications. In holding that counsel had rendered ineffective assistance by failing to adequately familiarize himself with the law, the Court stated:

> "Criminal cases will arise where the only reasonable and available defense strategy requires consultation with experts or introduction of expert evidence." Harrington v. Richter, 131 S.Ct. 770, 788 (2011). . . Prosecution experts, of course, can sometimes

> make mistakes. Indeed, we have recognized the threat to fair criminal trials posed by the potential for incompetent or fraudulent prosecution forensics experts, noting that "serious deficiencies have been found in the forensic evidence used in criminal trials. One study of cases in which exonerating evidence resulted in the overturning of criminal convictions concluded that invalid forensic testimony contributed to the convictions in 60% of the cases." Melendez–Diaz v. Massachusetts, 557 U.S. 305, 319 (2009). This threat is minimized when the defense retains a competent expert to counter the testimony of the prosecution's expert witnesses.

Id. at 1088, 1090.

These principles are clearly relevant to cases in which a caretaker is alleged to have injured a child by shaking. In fact, defense attorneys in Massachusetts were among the first to challenge SBS diagnoses. In Commonwealth v. Woodward, 427 Mass. 659 (1998), the Commonwealth contended that a baby had died as a result of SBS and blunt force. To rebut that claim, the defense retained experts who contested the shaken baby hypothesis and presented alternative explanations for the child's injuries. Id. at 662 n. 4, 671, 673 n.20, 674 nn. 22 & 24. After the jury returned a second degree murder verdict, the judge reduced the conviction to involuntary manslaughter, and this Court affirmed that decision. Id. at 670, 672.

Similarly, in Commonwealth v. Azar, 435 Mass. 675 (2002), the defendant was convicted in 1989 of first

degree murder of his four month old daughter. He testified that when he found her unresponsive in bed, he shook her in an effort to revive her, and when he ran to the phone she fell out of his arms. The Commonwealth's experts testified that she had a fracture to her skull caused by severe blunt force, and that other injuries were consistent with shaking. Defense experts, on the other hand, testified that the injuries were inconsistent with shaking, they indicated the application of only mild force, and they could have been caused in the manner described by the defendant. The Court reversed the conviction due to an erroneous instruction on third prong malice, stating, "The conflicting evidence in this case prevents us from concluding that the jury were required to find a plain and strong likelihood of death." Id. at 688.

Other case law highlights the importance of investigating the cause of death. In Commonwealth v. Haggerty, 400 Mass. 437 (1987), the defendant beat and robbed an 82-year-old woman with a history of heart trouble, and she died two months later. The Commonwealth's experts testified that her death was caused by a heart attack that she suffered on the day of the incident. Trial counsel cross-examined them about her medical history and the possibility that she may have suffered an unrelated heart attack on the day

of her death, but he had not retained his own experts to review the evidence. The defendant was convicted of first degree murder. He subsequently filed a motion for a new trial supported by expert evidence that a heart attack would have been the natural result of the woman's heart disease and that it could not be concluded that the beating played any role in the heart attack that caused her death. The Court reversed the conviction, stating:

> Defense counsel did not investigate the only realistic defense the defendant had to the charge of murder in the first degree. . . Failure to investigate the only defense a defendant has, if facts known to or with minimal diligence accessible to counsel support that defense, falls beneath the level of competency expected. . . The question whether death proximately was caused by the defendant required counsel to investigate that defense by seeking the opinion of an expert.

Id. at 441-42.

In recent years a number of convictions based on the SBS theory have been reversed due to trial counsel's failure to conduct an adequate investigation of the reliability of the diagnosis. In Ex Parte Briggs, 187 S.W.3d 458 (Tex. Crim. App. 2005), the defendant, who acknowledged shaking her two-month-old son, pleaded guilty in 2000 to injury to a child causing death. Five years later her motion to withdraw her plea was allowed due to counsel's failure to investigate the baby's medical history which led to the conclusion that he had died due to an undiagnosed

birth defect. Id. at 470.

In State v. Hales, 152 P.3d 321 (Utah 2007), the defendant was convicted of murder in 2004. The state's experts testified that a CT scan showed brain injury consistent with violent shaking that would have caused immediate unconsciousness. Trial counsel, however, never had an expert review the scan. At trial, the defendant testified that during a near-accident two days before, the baby had hit the car dashboard and fallen, and a defense expert discounted the SBS theory and testified that the near-accident was the likely cause of the injuries. Post-trial, an expert reviewed the CT scan and stated that the injury occurred at least six hours before the scan was taken and the injury did not preclude a lucid interval. The court reversed the conviction, stating, "Because the State's interpretation of the CT scans was critical to the State's case against Hales, Hales's trial attorneys' failure to hire a qualified expert to review the CT scans constituted a failure to conduct an adequate investigation." Id. at 338.

In DelPrete v. Thompson, 10 F.Supp.3d 907 (N.D. Ill. 2014), a three month old baby died months after losing consciousness at a daycare center when DelPrete was the only adult present. At the 2005 trial, the state presented expert testimony that the baby had SBS and that the injuries occurred immediately before she

lost consciousness. The defense expert, a pathologist who had only glanced at some articles about SBS, testified that the injuries occurred at least 18 hours before the baby collapsed. The jury convicted DelPrete of first degree murder. In this habeas corpus proceeding, DelPrete argued that trial counsel rendered ineffective assistance by failing to challenge the admission of the SBS testimony and failing to present appropriate expert testimony in rebuttal. At an evidentiary hearing at which the parties presented eleven expert witnesses, the court heard evidence that the injuries had occurred weeks earlier and that babies can have lucid intervals after head trauma. The court concluded "that Del Prete has established that it is more likely than not that no reasonable juror would have found her guilty of murder beyond a reasonable doubt" if counsel had presented that evidence at trial. Id. at 957. In reaching its conclusion, the court noted:

> As respondent's expert . . . testified, science cannot even yet establish an injury threshold. This, in addition to the other more recent developments in this area previously discussed, arguably suggests that a claim of shaken baby syndrome is more an article of faith than a proposition of science.

Id. at 957 n.10.

Most recently, in People v. Ackley, 497 Mich. 381 (2015), a three year old child died while in the

defendant's care. He stated that she had been napping and he found her lying unresponsive on the floor, the apparent victim of an accidental fall. Defense counsel consulted with one expert who stated that he would not be an appropriate person to retain and referred him to another expert, but counsel never followed up with the other expert and never reviewed any scientific studies. He did not present any expert testimony at trial. The prosecution, on the other hand, presented five experts to support its theory that the child died of blunt force, shaking, or a combination of the two. In support of a motion for a new trial, a defense expert testified that the child's injuries were likely caused by a mild impact rather than shaking. The motion was granted, and the Michigan Supreme Court affirmed that decision, stating, "We fail to see how counsel's sparse efforts satisfied his 'duty to make reasonable investigations or to make a reasonable decision that makes particular investigations unnecessary,' Hinton [v. Alabama], 134 S Ct [1081,] 1088 [2014], especially in light of the prominent controversy within the medical community regarding the reliability of SBS/AHT diagnoses." Id. at 391-92.

Other shaken baby cases have recently been reversed on grounds other than ineffective assistance of counsel. Council v. State, 98 So.3d 115, 117 (Fla.

App. 2012) (2011 conviction for aggravated child abuse reversed due to trial court's exclusion of biomechanics expert who would have testified that injuries could have been caused by short fall but not by shaking as state claimed); Brown v State, 152 So. 3d 1146, 1169 (Miss. 2014) (2006 murder conviction reversed because trial court denied funds to enable defense to hire pathologist to rebut SBS theory). As set forth in the following section, other convictions have been reversed due to newly discovered evidence regarding SBS.

As the cited cases demonstrate, when the Commonwealth relies in whole or in part on the SBS/AHT theory, the failure of an attorney to retain an expert to evaluate the evidence may violate the defendant's Sixth and Fourteenth Amendment right to the effective assistance of counsel. For almost 30 years now, studies have been appearing that raise doubts about SBS diagnoses, including whether shaking is sufficient to cause the injuries and whether the injuries may be caused by short falls and many medical conditions. In cases where these issues may be relevant, defense counsel has a duty to investigate.

The more stringent requirements of art. 12 may be violated as well. In Commonwealth v. Murphy, 448 Mass. 452, 465 (2007), the Court stated:

> In deciding whether art. 12 offers more protection of the right to counsel than the Sixth

> Amendment, "our guiding consideration is whether the Federal rule adequately protects the rights of the citizens of Massachusetts." Commonwealth v. Mavredakis, 430 Mass. 848, 858 (2000). This court looks "to the text, history, and our prior interpretations of art. 12, as well as the jurisprudence existing in the Commonwealth." Id.

The text of art. 12, which states that a defendant has a right "to be fully heard in his defence by . . . council," suggests that it should be given broader scope than the Sixth Amendment, which states that a defendant has a right "to have the assistance of counsel for his defence" (emphasis added). The Court has "long interpreted that text generously to recognize the fundamental right of a person accused of a serious crime to have the aid and advice of counsel." Commonwealth v. Rainwater, 425 Mass. 540, 553 (1997), cert. denied, 522 U.S. 1095 (1998). It should interpret that right to include a duty to fully investigate SBS/AHT diagnoses in cases where the challenges to that theory may be relevant.

B. Scientific Evidence May Be Deemed Newly Discovered Evidence Warranting a New Trial Where There Have Been Post-Trial Changes in Scientific Understanding or to the Admissibility of the Evidence in Court.

Evidence is newly discovered if it was "unknown to the defendant or his counsel and not reasonably discoverable by them at the time of trial." Commonwealth v. Grace, 397 Mass. 303, 306 (1986). To determine whether newly discovered evidence warrants a

new trial, "The motion judge decides not whether the verdict would have been different, but rather whether the new evidence would probably have been a real factor in the jury's deliberations." Id.

Newly available evidence, which is judged by the same standard, "is evidence that was unavailable at the time of trial" and it includes scientific discoveries that "had not yet been developed or gained acceptance by the courts." Commonwealth v. Sullivan, 469 Mass. 340, 350-51 & n.6 (2014) (newly available DNA test warranted new trial); Commonwealth v. Cowels, 470 Mass. 607, 615-17 (2015) (DNA test deemed experimental at time of trial but which became admissible in court after trial was newly available and warranted new trial); Commonwealth v. Meggs, 30 Mass. App. Ct. 111, 114 (1991) (new forensic tests for biological fluids); Bunch v. State, 964 N.E.2d 274, 297 (Ind. App., 2012), rev. denied 999 N.E.2d 416 (Ind. 2013) (advances in fire analysis warranted new trial in felony murder/arson case).

Courts in other jurisdictions have held that the evolution in the scientific understanding of shaking as a potential cause of brain injury in children warranted new trials based on newly discovered evidence. In State v. Edmunds, 746 N.W.2d 590 (Wis. App. 2008), rev. denied 308 Wis. 2d 612 (2008), the defendant was convicted in 1996 of involuntary

manslaughter of a seven-month-old baby based on the SBS theory. Ten years later she sought a new trial, presenting evidence from six expert witnesses about the evolving debate as to whether shaking can cause brain injuries. Applying a standard similar to the Grace standard, the court reversed the denial of her motion for a new trial. Id. at 595, 598. In reaching that decision, the court stated:

> The newly discovered evidence in this case shows that there has been a shift in mainstream medical opinion since the time of Edmunds's trial as to the causes of the types of trauma [the baby] exhibited. We recognize . . . that there are now competing medical opinions as to how [the baby's] injuries arose and that the new evidence does not completely dispel the old evidence. Indeed, the debate between the defense and State experts reveals a fierce disagreement between forensic pathologists, who now question whether the symptoms [the baby] displayed indicate intentional head trauma, and pediatricians, who largely adhere to the science as presented at Edmunds's trial. However, it is the emergence of a legitimate and significant dispute within the medical community as to the cause of those injuries that constitutes newly discovered evidence.

Id. at 598-99.

Another case, Ex parte Henderson, 384 S.W.3d 833 (Tex. Crim. App. 2012), bears some similarity to the Wilson and McCarthy cases. There the defendant was convicted in 1995 of murder due to the death of a 3.5-month-old baby. The defendant stated that the baby's injuries resulted from an accidental short fall. The

medical examiner, however, testified that was impossible - the baby must have been swung and then slammed into a hard surface. Twelve years later, though, the medical examiner, after reviewing recent biomechanical studies, changed the cause of death from homicide to undetermined. The appellate court affirmed the grant of a new trial. Id. at 834.

Given the rapidly changing understanding of what can cause brain injuries in young children, valid claims of newly discovered evidence may arise in a number of ways.

First, as in Meggs and Edmunds, research published after trial may cast doubt on expert testimony that had been presented at trial.

Second, as in Cowels, research published before trial but which was not yet generally recognized or accepted, but which becomes more widely accepted after trial may raise doubts about the trial evidence. In this area, the 2009 AAP paper indicates wider acceptance in the pediatric community of some of the criticisms of the SBS hypothesis. In addition, the survey articles about SBS that have recently appeared in law journals have brought wider attention to these issues in the legal community, resulting in greater acceptance by courts to challenges to SBS diagnoses.

Third, as in Henderson, published studies may cause the experts who testified at trial to modify or

change their own opinions in significant ways. Indeed, while not as radical a change as in Henderson, it is clear that the change in the AAP's position in 2009 from one of wholehearted acceptance of the SBS theory to one of recognition that shaking alone may not explain the injuries seen has led the Children's Hospital doctors to modify their testimony. At the Epps trial in 2007, Dr. Wilson testified that the child's injuries were consistent with SBS and that such injuries could not be caused by a short fall; rather, a fall of about 70 feet would be required. At the Millien trial in 2010, Dr. Newton testified that the term AHT was now favored rather than SBS, and she agreed that a short fall could cause a subdural hemorrhage. She testified, though, that the subdural and retinal hemorrhages and brain injury in this case were the result of shaking and could not be explained by a short fall. But she modified her testimony at the 2014 hearing on the motion for a new trial. Then she testified that impact contributed to those injuries and she acknowledged that short falls could be fatal.

Conclusion

The understanding of the causes of brain injuries in young children has undergone a considerable evolution in the 44 years since Dr. Guthkelch first proposed that shaking could be a cause. Now it is

recognized that impact generates far more force than shaking, and that short falls and many medical conditions can give rise to the injuries that had been attributed to shaking. This evolution in understanding will give rise to well-founded claims of ineffective assistance of counsel and newly discovered evidence.

Respectfully submitted,

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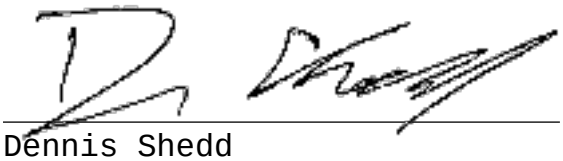
CERTIFICATE OF COMPLIANCE

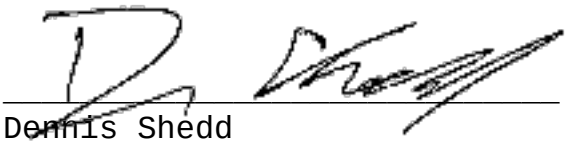
I certify that this brief complies with the rules of court that pertain to the filing of briefs, including but not limited to M.R.A.P. 16(a)(6),(e),(f),(h), 18, and 20.



Dennis Shedd

CERTIFICATE OF SERVICE

I certify under the penalties of perjury that on this date I served this document by mail on counsel for the Commonwealth, Assistant District Attorney David O'Sullivan, 10 Federal Street, Salem, MA 01970, and Assistant District Attorney Kate Cimini, 15 Commonwealth Avenue, Woburn, MA 01801, and on counsel for the defendants, David Hirsch, P.O. Box 900, Portsmouth, NH 03802.



Dennis Shedd

Dated: November 23, 2015

APPENDIX

Journal Articles

Initial Neurologic Presentation in Young Children Sustaining Inflicted and Unintentional Fatal Head Injuries

Kristy B. Arbogast, PhD*‡; Susan S. Margulies, PhD§; and Cindy W. Christian, MD‖

ABSTRACT. *Background.* **It remains unclear if fatal brain injuries in young children are characterized by immediate rapid deterioration or can present after an initial period of lucidity. This issue has legal implications in child abuse, for which understanding the clinical course affects perpetrator identification.**

Objective. **To determine patterns of neurologic presentation on hospital admission in infants and toddlers who die of inflicted and unintentional injury.**

Design/Methods. **Data on children <48 months of age who sustained a fatal head injury from 1986–2002 were extracted from the Pennsylvania Trauma Outcomes Study. Only those with external-causes-of-injury codes for inflicted injury, falls, and motor vehicle crashes (MVCs) with a recorded Glasgow Coma Scale (GCS) on admission were included. The GCS was compared across mechanisms and age groups (0–11, 12–23, 24–35, and 36–47 months).**

Results. **Of the 314 fatally injured children, 37% sustained inflicted injury, 13% sustained a fall, and 49% sustained an MVC. At admission, 6.8% of all children had a GCS score of >7, and 1.9% presented with a GCS score of >12 (lucid). The incidence of admission a GCS score of >7 varied by mechanism. Overall, children with inflicted injury were 3 times more likely to present with a GCS score of >7 than those injured in MVCs (odds ratio [OR]: 3.6; 95% confidence interval [CI]: 1.2–10.3), but incidence of a GCS score of >7 did not differ between inflicted injuries and falls. Similarly, when considering only those children ≥24 months old, a GCS score of >7 did not differ by mechanism. In contrast, in those <24 months old, children who died as a result of inflicted injury were >10 times more likely to have a GCS score of >7 than those who died as a result of a MVC (OR: 9.36; 95% CI: 1.3–80.9).**

Conclusions. **The data suggest an age- and mechanism-dependent presentation of neurologic status in children with fatal head injury. Although infrequent, young victims of fatal head trauma may present as lucid (GCS score: >12) before death. Furthermore, children <48 months old sustaining inflicted injury are 3 times more likely to be assessed with a moderate GCS score (>7) than those in MVCs. This effect is amplified in the youngest children (<24 months old): those with inflicted injury were 10 times more likely to present with moderate GCS scores than those in MVCs. In addition, this youngest age group seems to be overrepresented in those who present as lucid (GCS score: >12 [5 of 6]). It is unclear whether these differences are the result of inadequate tests to evaluate consciousness in younger children or differences in biomechanical mechanisms of inflicted trauma.** *Pediatrics* **2005;116:180–184;** ***abuse, accidents, falls, head injuries, lucid interval.***

ABBREVIATIONS. PTOS, Pennsylvania Trauma Outcomes Study; ICD-9, *International Classification of Diseases, Ninth Revision*; MVC, motor vehicle crash; GCS, Glasgow Coma Scale; OR, odds ratio; CI, confidence interval.

Homicide is the leading cause of injury death in infancy, and half of infant homicides occur during the first 4 months of life.[1] The majority of infant homicides involve inflicted traumatic brain injury. It remains unclear if fatal head injuries in young children are characterized by immediate rapid deterioration or can present after an initial period of lucidity.[2–5] This issue has legal implications in child abuse, for which understanding the clinical course affects perpetrator identification. Because inflicted injuries are typically unwitnessed and perpetrators are unwilling to provide a truthful history, the timing between injury and first clinical presentation may be unknown, further confounding the description of the clinical course.

In an attempt to determine if fatal head injuries in abused children are obviously symptomatic immediately, previous researchers have focused on the presence of a lucid interval by studying children who sustained unintentional fatal head injuries, for which the injury event is typically well documented. Plunkett[3] reported 18 short falls that resulted in fatal head injuries and documented 12 lucid intervals. In this study the average age (5.2 years) was significantly higher than that associated with child abuse. It is notable that no children were <1 year of age, and for those children <4 years of age, the lucid intervals documented were limited to <15 minutes. Willman et al[2] reviewed 95 cases of accidental fatalities involving head injury through a retrospective chart review and identified 2 children with a lucid interval: 1 child died with an epidural hemorrhage, and 1 died as a result of abdominal exsanguination. In their study, as well, the average age (8.5 years) was significantly older than the typical child involved in

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abusive head injury. Only 2 infants were included in their study sample. Because of variability in myelination and the size of subdural spaces between infants and older children,[6] the generalizability of their results to the inflicted-head-injury population remains unclear.

In this study, we build on this previous work and study a younger population. Specifically, we identify patterns of neurologic presentation for children <4 years of age who died as a result of inflicted and unintentional head injury.

METHODS

For the period 1986–2003, the Pennsylvania Trauma Systems Foundation Trauma Outcomes Study (PTOS) was reviewed to extract data on children <48 months of age who sustained a fatal head injury. PTOS is a statewide registry of clinical data from all 26 accredited trauma centers (adult and pediatric) throughout the state of Pennsylvania. A standardized set of data elements for cases that meet specific criteria is collected by trained trauma registrars in individual institutions and submitted to a central database. Inclusion criteria for the PTOS study are patients (all ages) admitted for treatment of a diagnosis of trauma including all intensive care admissions, all patients who were dead on arrival, all trauma deaths, and all admissions for at least 48 hours, including transfers. The diagnosis of trauma is defined as *International Classification of Diseases, Ninth Revision* (ICD-9) codes of 800 through 995. Cases are enrolled regardless of specific injury patterns. Specific exclusions include those patients with an isolated hip fracture, isolated asphyxiation, drowning, and poisoning. To minimize data errors, registrars attend mandatory training sessions semiannually, 1 of which focuses on interabstractor reliability. Audits are also performed regularly to monitor coding accuracy of the data submitted.

For this study, we selected those children <48 months of age with fatal head injuries from this database. Determination of fatal head injuries was based on ICD-9 coding of injuries. Only children who died with external-causes-of-injury codes (E codes) for inflicted injury (E967), falls (E880–E888), and motor vehicle crashes (MVCs) (E810–E819) with a recorded Glasgow Coma Scale (GCS) score on hospital admission were included. All 3 injury events were studied to determine the neurologic presentation of young children who sustained fatal head injuries from 3 varied mechanisms. MVCs, in particular, were chosen as a comparison to inflicted-injury events because the timing of the MVC relative to hospital presentation is well defined; thus, the occurrence of lucidity, if present, would be more likely to be documented. GCS scores were compared across mechanisms and age groups (0–11, 12–23, 24–35, and 36–47 months) by using the χ^2-test method. For the purposes of this study, relevant categorization of GCS scores was determined a priori (poor neurologic status: GCS score 3–7; moderate neurologic status: GCS score 8–12; good neurologic status [lucid]: GCS score 13–15).

RESULTS

Of the 314 fatally injured children identified, 37% (121) sustained inflicted injury, 13% (40) sustained falls, and 49% (153) were in MVCs. This distribution varied significantly by age ($P < .001$) (Fig 1).

Although most children had poor neurologic status on admission, 6.8% had an admission GCS score of >7, and 1.9% presented with a GCS score of 13–15 (lucid). Figure 2 demonstrates the distribution of GCS scores for each year of age.

The incidence of admission GCS scores varied by mechanism (Fig 3). Overall, children with inflicted injury were 3 times (odds ratio [OR]: 3.6; 95% confidence interval [CI]: 1.2–10.3) more likely to present with a GCS score of >7 than those in MVCs, but incidence of a GCS score of >7 did not differ between inflicted injury and falls (OR: 1.5; 95% CI: 0.4–5.5) (Table 1). When considering only children ≥24 months old, a GCS score of >7 did not differ by mechanism. In contrast, in those <24 months old, children who died as a result of inflicted injury were >10 times more likely to have a GCS score of >7 than those who died as a result of a MVC (OR: 10.6; 95% CI: 1.3–80.9). Thus, the influence of mechanism on the incidence of a GCS score of >7 varied by age.

Overall, 6 children were lucid (GCS score: >12) at admission, and children <24 months were overrepresented in this group (5 of 6). Moreover, 3 of 121 (2.5%) children with ultimately fatal inflicted injury were thought to be lucid at presentation to a Pennsylvania trauma hospital. All 3 were <24 months old (Table 2). Of the 6 children with a GCS score of >12 on admission, 4 sustained a subdural hematoma as part of their head injury.

DISCUSSION

Whether children can be lucid after sustaining a fatal head injury has important implications in the investigation of child abuse. Historically, infants and toddlers who have sustained fatal inflicted trauma are assumed to have been immediately and obviously symptomatic.[4,5] With a large sample of age-relevant cases, our data suggest that although infrequent, young victims of fatal head trauma may present as lucid (GCS score: 13–15) before death. In this series, ~2% of children <4 years of age were recorded as having a GCS score of 13–15 on arrival to a trauma hospital. By using PTOS data, our study only included children who were admitted to a Pennsylvania trauma hospital and did not capture data on children who died at the scene of the injury or at a local hospital before transfer to a trauma

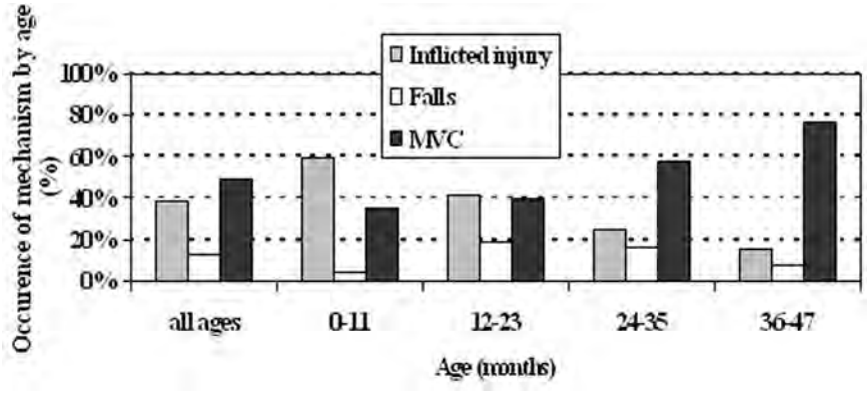

Fig 1. Distribution of injury mechanism (inflicted injury, falls, and MVCs) according to child age.

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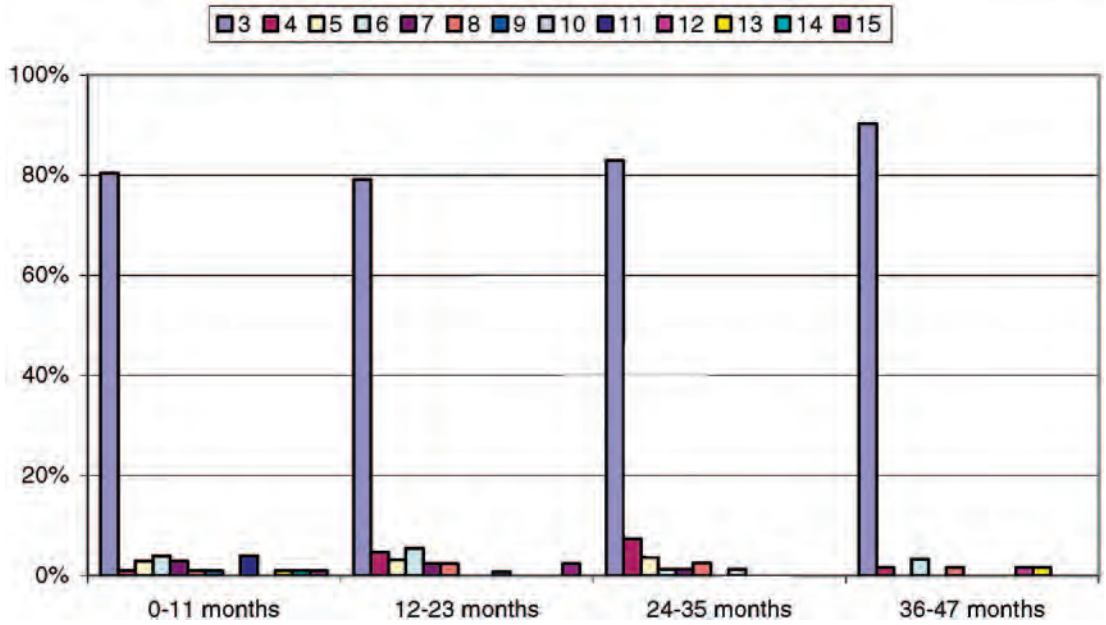

Fig 2. Distribution of GCS score according to child age.

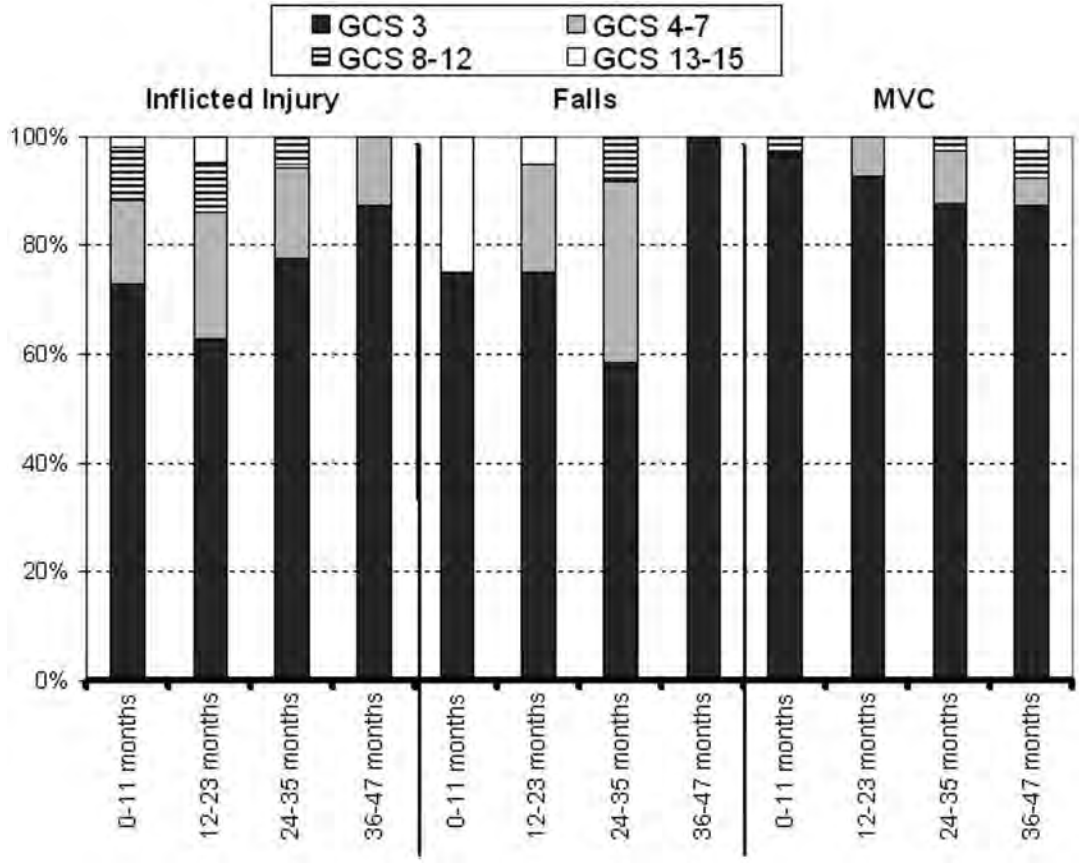

Fig 3. Distribution of GCS score according to child age and injury mechanism.

hospital. Therefore, our findings most likely overestimate the true percentage of children who seem to be lucid after fatal injury.

In our study, children <48 months old sustaining inflicted injury were 3 times more likely to be assessed with a moderate GCS score (>7) than those in MVCs. These results from a large sample support similar conclusions from single case reports.[4,5] In MVCs resulting in fatal injuries, the event is often a single, well-defined, high-energy impact. Inflicted injuries are typically more complex loading events that may include multiple purely inertial (shaking) as well as impact conditions, may encompass a wider magnitude range than MVCs, and may be repeated

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TABLE 1. Percentage of Children With GCS Scores of >7

Age Group	Inflicted Injury, % (*n*)	MVCs, % (*n*)	Falls, % (*n*)	Inflicted Injury vs MVCs/Inflicted Injury vs Falls, OR (95% CI)
Overall	10.7 (13/121)	3.3 (5/153)	7.5 (3/40)	3.6 (1.2–10.3)/1.5 (0.4–5.5)
0–23 mo	12.6 (12/95)	1.4 (1/72)	8.3 (2/24)	10.6 (1.3–80.9)/1.6 (0.3–7.6)
24–47 mo	3.8 (1/26)	4.9 (4/81)	6.2 (1/16)	0.8 (0.08–7.2)/0.6 (0.03–10.3)

TABLE 2. Children With GCS Scores of >12 (*n* = 6)

Mechanism (Age Group)	Head Injury	Hospital Length of Stay, d
MVCs		
36–47 mo	Closed skull fracture, SAH, SDH, EDH	3
Falls		
0–11 mo	Closed skull fracture, SAH, SDH, EDH	1
12–23 mo	Closed skull fracture, intracranial hemorrhage	9
Inflicted injury		
0–11 mo	Intracranial hemorrhage	1
12–23 mo	SDH	2
12–23 mo	SDH, MMA/intracranial hemorrhage	47

SAH indicates subarachnoid hematoma; SDH, subdural hematoma; EDH, epidural hematoma; MMA, middle meningeal artery.

at intervals of hours, days, or weeks. It remains unclear if repeated mild head injuries exacerbate or modulate the response compared with a single, more severe injury event.[7–9]

Our data indicate that the initial neurologic presentation of children with fatal head injury also depended on age. In the youngest children (<24 months old), those with inflicted injury were 10 times more likely to present with a moderate GCS score than those in MVCs. In addition, this youngest age group seems to be overrepresented in those who present as lucid (GCS score: 13–15 [5 of 6]). For those >24 months old, there was no difference in GCS scores among the 3 injury mechanisms. This result can be interpreted in several ways. It may suggest that inflicted injuries in infants occur more frequently, or that there are distinct responses of an infant's and toddler's brain to traumatic loading. Studies that examine the biomechanical, neurologic, and physiologic responses of the young child's brain across the age range are necessary to fully explain these results.

Alternatively, the overrepresentation of the youngest infants in the moderate-GCS-score group may simply reflect the inadequacy of the GCS in assessing neurologic damage or mental clarity in the very youngest patients. The GCS relies on motor and verbal skills that cannot be assessed accurately in infants and toddlers. For example, the motor component of the GCS assesses localization of pain, which does not typically develop in a child until ~18 months of age. The verbal response options are inappropriate for preverbal children, greatly limiting the reliability of the overall scale. Additionally, spontaneous eye opening and/or nonspecific movements in response to pain can be seen in infants who have suffered severe traumatic brain injury affecting the cortex, which can result in the assigning of an erroneously high GCS score.[10] To overcome these limitations, a number of pediatric-appropriate scales have been developed. Some of them do not overcome the challenges of finding practical but accurate ways to assess infant neurologic function and remain inappropriate for intubated patients.[11–13] Although appropriate scales have been developed for use in infants and toddlers, their prognostic ability has not been tested.[10] Despite progress to create and validate appropriate coma scales for young children, none have been broadly adopted by clinicians, and the GCS remains the scale used in most trauma centers, including those in Pennsylvania. However, because in this series the majority of patients who died as a result of inflicted injury were infants, the imprecision of GCS at younger ages may confound our findings.

Although our data suggest that a small percentage of infants and toddlers with fatal head injury can present as lucid to a trauma center, it does not imply that these children were completely asymptomatic. The GCS does not measure common symptoms of head injury such as vomiting, irritability, or subtle changes in alertness. It is possible that some children with GCS scores of 13–15 had clinical signs of head trauma not assessed by that scale.[14]

Specific clinical presentation among a sample of children with fatal head injuries is variable. Researchers have suggested that an infant's response to traumatic loading differs from that of an older child[6] and that the acceleration magnitude varies by the mechanism.[15,16] Little research has been conducted that correlates specific clinical presentations of young children with neuropathological findings and how that varies by age and injury mechanism. Future work should focus on making these linkages to determine if those fatal cases that present with a lucid interval demonstrate unique pathology from the overwhelming majority of children who are immediately obviously symptomatic.

These analyses were conducted on a specific population of young children with fatal head injuries from Pennsylvania's statewide trauma registry. This registry has been used previously to study injury events (the rates and injury characteristics of firearm

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injuries in adolescents and young adults[17] and risk of pulmonary embolism after head injury in adults[18]). Of Pennsylvania's 67 counties, 51 have a trauma center in the county or an adjoining county. These 51 counties include 84% of Pennsylvania's population. We hypothesize that the majority of those children with fatal head injuries not contained in the Pennsylvania Trauma Systems Foundation database died at the scene and did not enter the health care system. This consideration would only serve to emphasize the rarity of a lucid interval, because it would increase the underlying population of children with fatal head injuries from which our 6 children with GCS scores of 13–15 are drawn.

A population-based database such as that used in this study provides an appropriate sample to be able to conduct relevant statistical analysis. Data in the database were deidentified and abstracted from medical records, preventing the review of the actual medical details, confirmation of the injury mechanism, and determination of the nuances of the clinical time course of an individual patient. Future studies should utilize multicenter networks such as the Pediatric Emergency Care and Applied Research Network (PECARN) to identify substantial numbers of young children with fatal head injuries in which detailed clinical data and injury-mechanism information can be obtained.

CONCLUSIONS

Our data suggest that on rare occasion, an infant or toddler can sustain a fatal head injury yet present as lucid to hospital clinicians before death. Whether this is because of differences in pathologic injury, neurologic responses unique to the infant brain, or limitations of the bedside methods used to assess neurologic function in young children cannot be determined by this study. Because fatal head injury is a relatively rare event in infancy, improved understanding about the clinical course of infants and toddlers after fatal head injury will be served best by cooperative, multicenter work.

ACKNOWLEDGMENTS

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Imaging of the Central Nervous System in Suspected or Alleged Nonaccidental Injury, Including the Mimics

Patrick D. Barnes, MD and Michael Krasnokutsky, MD

Abstract: Because of the widely acknowledged controversy in nonaccidental injury, the radiologist involved in such cases must be thoroughly familiar with the imaging, clinical, surgical, pathological, biomechanical, and forensic literature from all perspectives and with the principles of evidence-based medicine. Children with suspected nonaccidental injury versus accidental injury must not only receive protective evaluation but also require a timely and complete clinical and imaging workup to evaluate pattern of injury and timing issues and to consider the mimics of abuse. All imaging findings must be correlated with clinical findings (including current and past medical record) and with laboratory and pathological findings (eg, surgical, autopsy). The medical and imaging evidence, particularly when there is only central nervous system injury, cannot reliably diagnose *intentional* injury. Only the child protection investigation may provide the basis for *inflicted* injury in the context of *supportive* medical, imaging, biomechanical, or pathological findings.

Key Words: child abuse, computed tomography, magnetic resonance imaging, nonaccidental injury, nonaccidental trauma

(*Top Magn Reson Imaging* 2007;18:53–74)

Traumatic central nervous system (CNS) injury is reportedly the leading cause of childhood morbidity and mortality in the United States, resulting in about 100,000 emergencies annually and half the deaths from infancy through puberty.[1–5] The major causes are accidental injuries (AIs) and include falls, vehicular accidents, and recreational mishaps. However, nonaccidental, inflicted, or intentional trauma is said to be a frequent cause, with peak incidence at the age of about 6 months and accounting for about 80% of the deaths from traumatic brain injury in children younger than 2 years. Nonaccidental injury (NAI)—or nonaccidental trauma (NAT)—is the more recent terminology applied to the traditional labels *child abuse*, *battered child syndrome*, and *shaken baby syndrome* (SBS).[4,5] A modern restatement of the definition of SBS is that it represents a form of physical NAI to infants characterized by "the triad" of (1) subdural hemorrhage (SDH), (2) retinal hemorrhage (RH), and (3) encephalopathy (ie, diffuse axonal injury [DAI]) occurring in the context of inappropriate or inconsistent history and commonly accompanied by other apparently inflicted injuries.[6] The short-term life-threatening presentations and long-term outcomes have become a major concern in health care, dating back to the original reports of Kempe,[7] Caffey,[8] and Silverman.[9] Later reports on the incidence rate of CNS trauma in alleged NAI estimate a range of 7% to 19%.[4,5]

However, a number of reports from multiple disciplines have challenged the evidence base (ie, quality of evidence [QOE] analysis) for NAI/SBS as the cause in all cases of the triad.[4,5,10] Such reports indicate that the triad may also be observed in AI (including those associated with short falls, lucid interval, and rehemorrhage) and in nontraumatic or medical conditions. These are the "mimics" of NAI that often present as acute life-threatening events (ALTE). This includes hypoxia-ischemia (eg, apnea, choking, respiratory or cardiac arrest), ischemic injury (arterial vs venous occlusive disease), seizures, infectious or postinfectious conditions, coagulopathy, fluid-electrolyte derangement, and metabolic or connective tissue disorders. Many cases seem multifactorial and involve a combination or sequence of contributing events or conditions.[4,5,10] For example, an infant is dropped and experiences a head impact with delayed seizure, choking spell, or apnea, and then undergoes a series of prolonged or difficult resuscitations, including problematic airway intubation with subsequent hypoxic-ischemic brain injury. Another example is a young child with ongoing infectious illness, fluid-electrolyte imbalance, and coagulopathy, and then experiences seizures, respiratory arrest, and resuscitation with hypoxic-ischemic injury.

Often, the imaging findings are neither characteristic of nor specific for NAI. Because of the widely acknowledged controversy in NAI, the radiologist involved in such cases must be thoroughly familiar with the imaging, clinical, surgical, pathological, biomechanical, and forensic literature from all perspectives and with the principles of evidence-based medicine (EBM).[4,5,10] Children with suspected NAI versus AI must not only receive protective evaluation but also require a timely and complete clinical and imaging workup to evaluate the pattern of injury and timing issues and to consider the mimics of abuse.[4,5,10] All imaging findings must be correlated with clinical findings (including current and past medical record) and with laboratory and pathological findings (eg, surgical, autopsy). The medical and imaging evidence, particularly when there is only CNS injury, cannot reliably diagnose *intentional* injury. Only the child protection investigation may provide the basis for *inflicted* injury in the context of *supportive* medical, imaging, biomechanical, or pathological findings.[4,5,10]

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MECHANISMS AND MANIFESTATIONS OF TRAUMATIC CNS INJURY

The spectrum of CNS injury associated with trauma (AI or NAI) has been classified into primary versus secondary, focal versus diffuse, and acute versus chronic categories.[4,5,10,11] The primary injury is immediate, irreversible, and is the direct result of the initial traumatic force (eg, contusion, shear injury). Secondary injury denotes the reactive phenomena that arise from or are associated with the primary injury (eg, swelling, hypoxia-ischemia, herniation). Direct contact or impact phenomena produce localized cranial distortion or deformation and thus produce *focal* injury (eg, fracture [Fx], contusion, epidural hematoma [EDH]). Accidental injury is said to be typically associated with this mechanism and result (Fig. 1). Although reported also in cases of NAI, it has been stated that impact injury, with the exception of EDH, is usually not life threatening.

It is *indirect* trauma (ie, independent of skull deformation) that has been considered responsible for the most severe CNS injury in SBS/NAI.[4,5,10–13] Inertial loading accompanying sudden angular acceleration/deceleration of the head on the neck (as with shaking) produces shear strain deformation and disruption at tissue interfaces, therefore *diffusing* the injury (Fig. 2). The young infant is said to be particularly vulnerable because of weak neck muscles, a relatively large head, and an immature brain. It is the shaking mechanism that is traditionally postulated to result in the triad, including primary traumatic injury (ie, SDH, RH, and DAI), with or without the secondary injury pattern (ie, edema, swelling, hypoxia-ischemia, herniation). Reportedly, such patterns are associated with the most severe and fatal CNS injuries and are readily demonstrated by means of neuroimaging, surgical neuropathology, and postmortem neuropathology.[4,5,10–13]

On a medical forensic basis, it is further stipulated that (1) retinal hemorrhages of a particular pattern are diagnostic of SBS/NAI, (2) such CNS injury on an accidental basis can only be associated with a massive force equivalent to a motor vehicle accident or a fall from a 2-story building, (3) such injury is immediately symptomatic and cannot be followed by a lucid interval, and (4) changing symptoms in a child with previous head injury is caused by newly inflicted injury and not just a *rebleed*. Using this reasoning, the last caretaker is automatically guilty of abusive injury, especially if not witnessed by an independent observer.[4,5,10–13]

The range of acute primary and secondary CNS injury reported to occur with NAI significantly overlaps that of AI.[4,5,10,11] This includes multiple or complex cranial fractures, acute interhemispheric SDH (Fig. 2), acute-hyperacute convexity SDH, multiple contusions, shear injury (DAI, white matter tears), brain swelling, edema, and hypoxia-ischemia (Fig. 2). The range of chronic CNS injury includes chronic SDH, communicating hydrocephalus, atrophy, or encephalomalacia. The combination of acute and chronic findings suggests more than 1 traumatic event. Imaging evidence of CNS injury may occur with or without other clinical findings of trauma (eg, bruising) or other traditionally *higher-specificity* imaging findings associated with violent shaking (eg, metaphyseal, rib, or other typical skeletal injuries).[4,5,10] Therefore, clinical and imaging findings of injury disproportional to the history, and injuries of differing age, have become 2 of the key diagnostic criteria indicating the *probability* of NAI/SBS, particularly when encountered in the premobile, young infant.[4,5,10] Such clinical and imaging findings have traditionally formed the basis from which health professionals, including radiologists, have provided a medical diagnosis and offered expert testimony that such *forensic* findings are *proof* of NAI/SBS.[10]

CONTROVERSY

Fundamental difficulties persist in formulating a *medical* diagnosis or *forensic* determination of NAI/SBS on the basis of a causative event (ie, shaking) that is inferred from clinical, radiological, and/or pathological findings in the often *subjective* context of (1) an unwitnessed event, (2) a *noncredible* history, or (3) an admission or confession.[4,5,10] This problem is further confounded by the lack of consistent and reliable criteria for the diagnosis of NAI/SBS, and that the vast body of literature on child abuse is comprised of

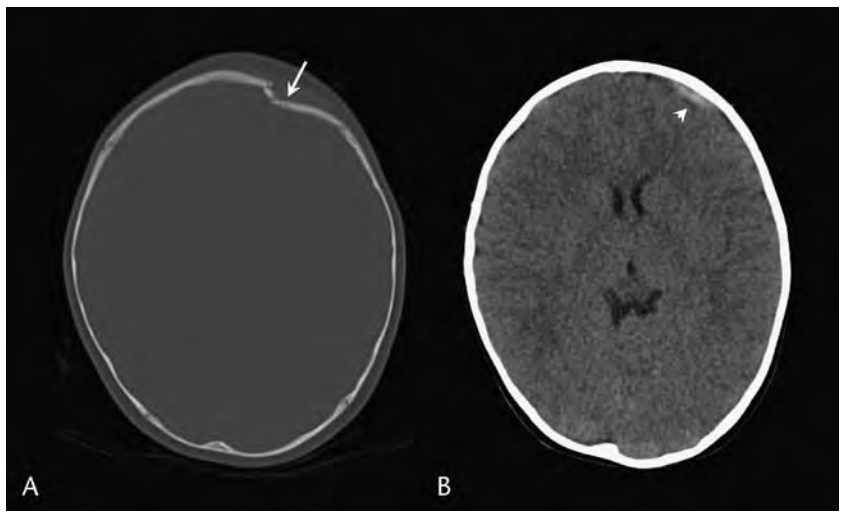

FIGURE 1. Images obtained from a 22-month-old female motor vehicle accident victim with depressed left-side frontal skull fracture (A, arrow), overlying scalp swelling, and a small, high-density epidural hematoma (B, arrowhead).

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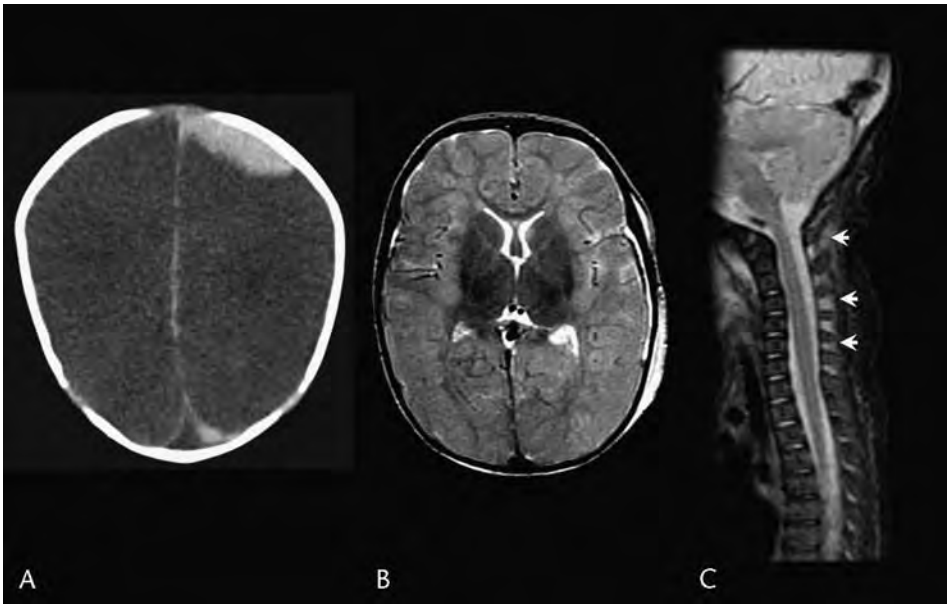

FIGURE 2. Images obtained from a 25-day-old female neonate with history of drop and RH (alleged NAI). A, Axial CT image shows high-density left-side frontal SDH (surgically drained before MRI), bilateral cerebral low densities with decreased gray-white matter differentiation (hypoxia-ischemia?), and interhemispheric high-density hemorrhage. B, Axial T2 MRI scan shows bilateral cerebral cortical and subcortical T2 high intensities plus interhemispheric T2 low intensities. C, Sagittal STIR cervical spine MRI scan shows posterior ligamentous high intensities (arrows) but no definite cord injury (NAI? SCIWORA?).

anecdotal case series, case reports, reviews, opinions, and position papers.[10,14] Furthermore, many reports include cases having impact injury that not only raises doubt regarding the *shaking-only* mechanism but also questions that this injury is always NAI based on a *shaken-impact* mechanism. From the perspective of EBM, QOE ratings for SBS/NAI diagnostic criteria reveal that few published reports merit a rating above class IV (ie, any design where test is not applied in blinded evaluation, or evidence provided by expert opinion alone or in a descriptive case series without controls).[10,14] The inclusion criteria provided in many reports often seem arbitrary, such as *suspected abuse*, *presumed abuse*, *likely abuse*, and *indeterminate*.[15,16] Furthermore, the diagnostic criteria often seem to follow *circular logic* (ie, SBS = SDH + RH [inclusion criteria], therefore SDH + RH = SBS [conclusion]). Such low QOE ratings hardly earn a EBM diagnostic recommendation level of *optional*, much less as a *guideline* or a *standard*.[10,14] This has traditionally been true of the neuroimaging literature, the clinical literature that uses neuroimaging, and the forensic pathology literature.[10,17–44]

The most widely reported attempt of a scientific study to test NAI/SBS used a biomechanical approach, measured stresses from shaking versus impact in a doll model, and correlated those stresses with injury thresholds in subhuman primate experiments established in another study.[45–47] Only stresses associated with impact, whether using an unpadded or padded surface, exceeded the injury thresholds that correlated with the pathological spectrum of concussion, SDH, and DAI. The authors concluded that CNS injury in SBS/NAI in its most severe form is usually not caused by shaking alone. These results obviously contradicted many of the original reports that had relied on the "whiplash" mechanism as causative of the triad.[47–49] These authors also concluded that fatal cases of SBS/NAI, unless occurring in children with predisposing factors (eg, subdural hygroma [SDHG], atrophy, etc), are not likely to result from shaking during play, feeding, and swinging, or from more vigorous shaking by a caretaker for discipline. A number of subsequent studies using various biomechanical, animal, and computer models have failed to convincingly invalidate this study, although many contend that there is no adequate model yet designed to properly test shaking versus impact.[50–61] Some of these reports also indicate that shaking alone cannot result in brain injury (ie, the triad) unless there is concomitant neck, cervical spinal column, or cervical spinal cord injury (Fig. 2).[53,54]

A number of past and more recent reports raise serious doubt that abuse is the cause in all cases of infant CNS injury using traditional SBS/NAI diagnostic criteria.[10,14,16,46,49,62–68] This includes reports of skull fracture or acute SDH from accidental simple falls in young infants, such as those associated with wide extracerebral spaces (eg, benign external hydrocephalus, benign extracerebral collections of infancy, SDHGs),[69–83] and fatal pediatric head injuries caused by witnessed, accidental short-distance falls, including those with a lucid interval and RH.[84–102] Recent neuropathologic studies in alleged SBS cases indicate that (1) the cerebral swelling in young infants is more often caused by diffuse axonal injury of hypoxic-ischemic origin rather than traumatic origin (traumatic origin is more appropriately termed *multifocal traumatic axonal or shear injury*); (2) although Fx, SDH (eg, interhemispheric), and RH are commonly present, the usual cause of death was increased intracranial pressure from brain swelling associated with hypoxia-ischemia; and (3) cervical EDH and focal axonal brain stem, cervical cord, and spinal nerve root injuries were characteristically observed in these infants (presumably caused by shaking, although most had impact findings).[103–109] Such upper cervical cord/brainstem injury may result in apnea/respiratory arrest and be responsible for the hypoxic-ischemic brain injury. Additional neuropathologic series have shown that dural hemorrhages are also observed in nontraumatic fetal, neonatal, and infant cases, and that the common denominator is likely a combination of cerebral

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venous hypertension and congestion, arterial hypertension, brain swelling, and immaturity with vascular fragility further compromised by hypoxia-ischemia or infection.[107–109] Reports of neurosurgical, neuroradiological, and neuropathologic findings in head trauma, as correlated with biomechanical analyses, indicate that SDH and RH occur with rotational deceleration injuries, whether *accidental* (eg, axis or center of rotation internal to the skull, including those resulting from short-distance falls) or *nonaccidental* (ie, axis of rotation external to the skull [eg, at the craniocervical junction or cervical spinal level]).[50–53] There is no scientific basis to date to indicate how much or how little force is necessary to produce traumatic injury to the developing CNS.

Furthermore, the specificity of RH for child abuse and its dating has also been questioned.[4,5,10,16,49,67,68,73,84,110–113] Such hemorrhages have been reported with a variety of conditions, including AT, resuscitation, increased intracranial pressure, increased venous pressure, subarachnoid hemorrhage (SAH), sepsis, coagulopathy, certain metabolic disorders, systemic hypertension, and other conditions. Furthermore, many cases of RH (and SDH) are confounded by the existence of multiple factors or conditions that often have a synergistic influence on the type and the extent of RH. For example, consider the child who has trauma, hypoxia-ischemia, coagulopathy, and has undergone resuscitation.

IMAGING PROTOCOLS

Proper imaging evaluation includes not only computed tomography (CT) and a radiographic or radionuclide skeletal survey but also magnetic resonance imaging (MRI) and, in some cases, serial imaging.[4,10,114–118] Occasionally, ultrasonography (US) may be useful. The imaging protocols should be designed to evaluate not only NAI versus AI but also the nontraumatic mimics. Computed tomography is the primary modality in acute neurological presentations because of its access, speed (particularly using multidetector technology), and ability to demonstrate abnormalities requiring immediate neurosurgical or medical intervention (eg, an expanding hematoma, brain swelling, impending herniation) (Figs. 1, 2).[4,10,114] Nonenhanced head CT with soft tissue and bone algorithms is performed. Facial and spinal (eg, cervical) CT may also be needed, including reformatting. Three-dimensional computed tomographic reconstructions can be important to evaluate fractures versus developmental variants (eg, accessory sutures, fissures, synchondroses). Computed tomographic angiography (CTA) or computed tomographic venography (CTV) may be helpful to evaluate the cause of hemorrhage (eg, vascular malformation, aneurysm) or infarction (eg, dissection, venous thrombosis). Intravenous contrast-enhanced CT or US with Doppler may be used to separate subarachnoid and subdural compartments by identifying bridging veins within the subarachnoid space; however, MRI is usually needed for more definite evaluation. In addition, in the unstable infant, initial and repeat cranial US (eg, transcranial Doppler) at the bedside may assist in evaluating structural abnormalities and monitoring alterations in cerebral blood flow and intracranial pressure.

Magnetic resonance imaging should be conducted as soon as possible because of its sensitivity and specificity regarding pattern of injury and timing parameters.[4,10,114–118] Brain MRI should include 3 planes and at least T1, T2, fluid-attenuated inversion recovery (FLAIR), gradient-recalled echo (GRE) T2*, and diffusion imaging (diffusion-weighted imaging [DWI]/apparent diffusion coefficient [ADC]) (Fig. 3). Gadolinium-enhanced T1 images should probably be used along with MRA and magnetic resonance venography (MRV).

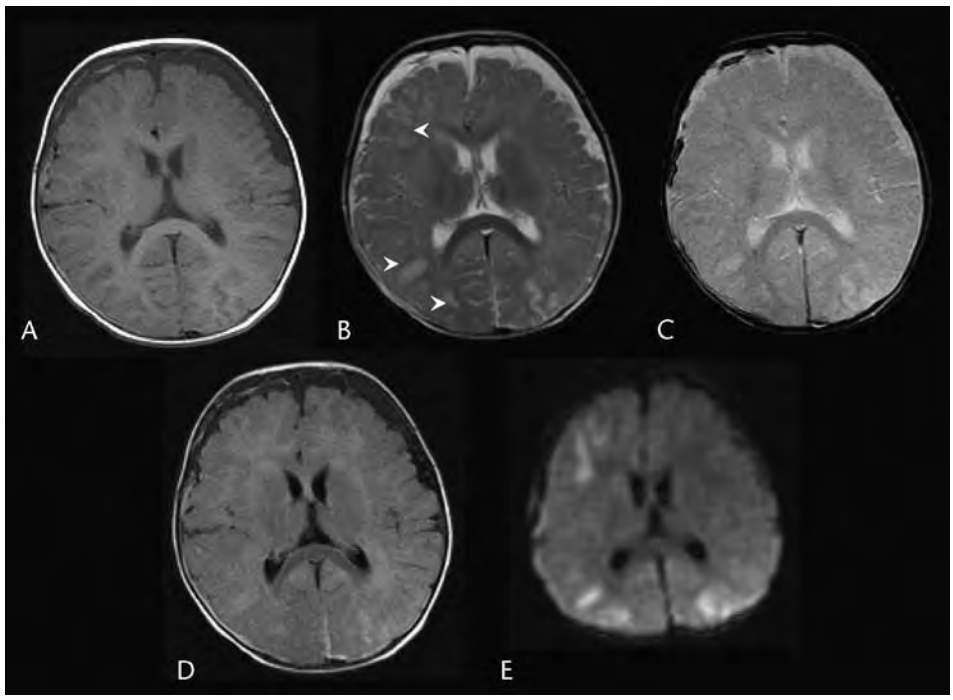

FIGURE 3. Images obtained from an 8-month-old male infant after viral illness, right-side humeral fracture, and RH (alleged NAI). Axial T1 (A), T2 (B), GRE (C), FLAIR (D), and DWI (E) images show bilateral frontal extracerebral CSF-intensity collections with right-side frontal extracerebral hemorrhage that is T1/FLAIR hyperintense and T2/GRE hypointense. Also seen are multifocal cerebral T2/FLAIR hyperintensities (arrowheads) that are DWI hyperintense (shear vs infarction?).

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TABLE 1. Magnetic Resonance Imaging of Intracranial Hemorrhage and Thrombosis*

Stage	Biochemical Form	Site	T1 MRI	T2 MRI
Hyperacute (+ edema) (<24 hours)	Fe II oxyHb	Intact RBCs	Iso-low I	High I
Acute (+ edema) (1–3 days)	Fe II deoxyHb	Intact RBCs	Iso-low I	Low I
Early subacute (+ edema) (3–7 days)	Fe III metHb	Intact RBCs	High I	Low I
Late subacute (− edema) (1–2 weeks)	Fe III metHb	Lysed RBCs (extracellular)	High I	High I
Early chronic (− edema) (>2 weeks)	Fe III transferrin	Extracellular	High I	High I
Chronic (cavity)	Fe III ferritin and hemosiderin	Phagocytosis	Iso-low I	Low I

*Modified from Wolpert and Barnes,[119] Kleinman and Barnes,[4] Bradley,[120] and Zuerrer et al.[121]
RBCs indicates red blood cells; I, intensity; plus sign (+), present; minus sign (−), absent; Hb, hemoglobin; Fe II, ferrous; Fe III, Ferric; Iso, isointense.

The cervical spine should also be imaged, along with other levels when indicated, and especially by using short TI inversion recovery (STIR) (Fig. 2). T1- and T2-weighted imaging techniques are necessary for characterizing the nature and timing (whether hyperacute, acute, subacute, or chronic) of hemorrhages and other collections by using established criteria (Table 1). Gradient-recalled echo or other susceptibility-weighted (T2*) techniques is most sensitive for detecting hemorrhage or thromboses that are often not identified on other sequences. However, GRE cannot be used for timing alone because it shows most hemorrhages (new and old) as hypointense (eg, deoxyhemoglobin, intracellular methemoglobin, hemosiderin).[4,10,114] The FLAIR sequence suppresses cerebrospinal fluid (CSF) intensity and allows for a better assessment of brain abnormalities, especially when adjacent to a CSF space or collection. FLAIR is also sensitive (but nonspecific) for subarachnoid space abnormalities, which appear as high intensity (eg, hemorrhage, exudate, inflammatory or neoplastic leptomeningeal infiltration, occlusive vascular slow flow, and hyperoxygenation during sedation or anesthesia). DWI plus ADC can be quickly obtained to show hypoxia-ischemia or vascular occlusive ischemia. Magnetic resonance spectroscopy (MRS) may show a lactate peak. It must be remembered, however, that restricted or reduced diffusion may be observed in other processes, including encephalitis, seizures, or metabolic disorders, and with suppurative collections and some tumors.[4,10,114] Gadolinium-enhanced sequences and MRS can be used to evaluate these other processes. In addition, MRA and MRV are important to evaluate arterial occlusive disease (eg, dissection) or venous thrombosis. The source images should be viewed along with the reprojected images. In some cases of partial occlusion/thrombosis, the abnormality may be more conspicuous on CTA/CTV, especially in infants. For evaluating arterial dissection by means of MRI, an axial fat-suppressed T1 sequence from the aortic arch to the circle of Willis may detect T1-hyperintense hemorrhage or thrombosis (ie, methemoglobin) within the false lumen, especially if the process is in the subacute phase.

INJURY EVALUATION

The range of CNS injury in childhood trauma, whether AI or NAI, often demonstrated by imaging may be categorized according to being primary or secondary (as previously described) and according to specific anatomical involvement, including scalp, cranial, intracranial, vascular, spinal, and head and neck.[2,4,5,10] A thorough analysis of the injury requires a systematic breakdown into injury components for both pattern of injury and timing parameters.

SCALP INJURY

Scalp injuries include hemorrhage, edema, or laceration and may be localized to any layer (SCALP [*s*kin, sub*c*utaneous,

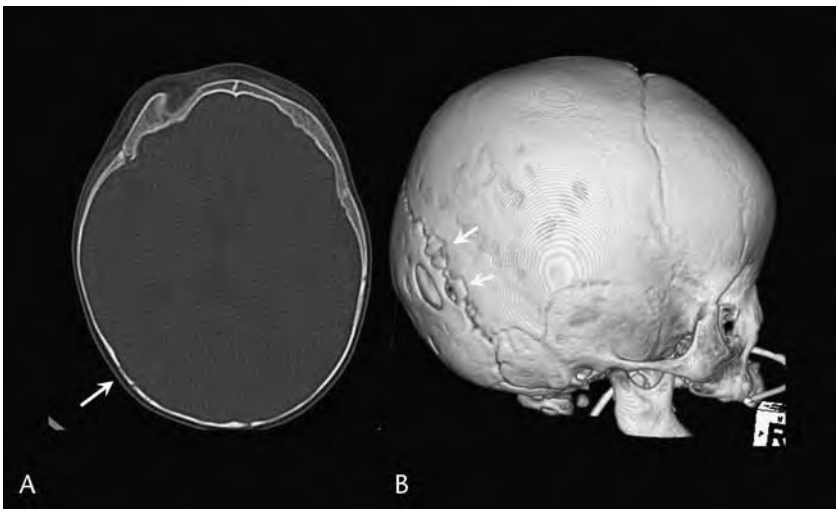

FIGURE 4. Images obtained from a 10-month-old male infant with intrasutural (wormian) bones versus fractures. A, CT image shows right-side parietal cranial defects (arrow). B, Three-dimensional computed tomographic surface reconstruction confirms intrasutural bones (arrows).

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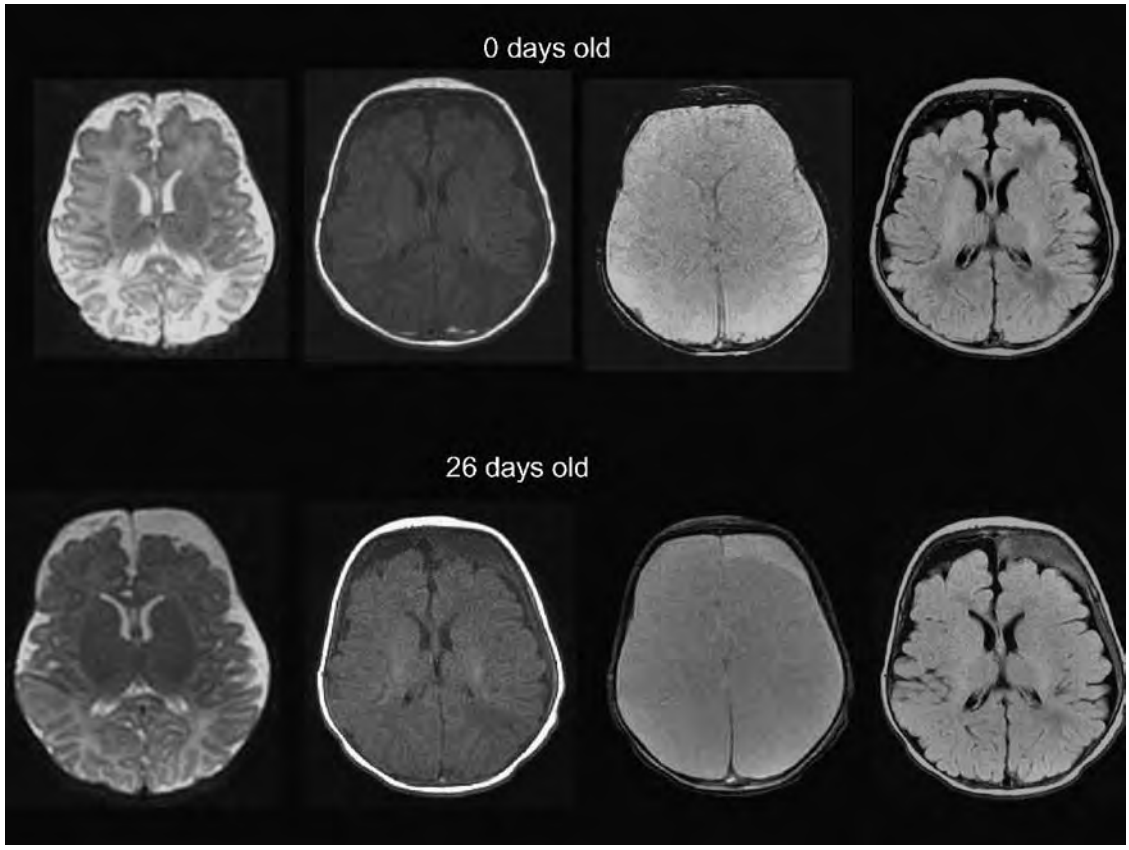

FIGURE 5. Images obtained from an infant with benign extracerebral collections of infancy and spontaneous subdural hemorrhage. Axial T2, T1, GRE, and FLAIR images (left to right) show CSF-intensity frontal subarachnoid collections at birth (top row). At 26 days postnatal age (bottom row), superimposed subdural collections that don't conform to CSF signal are present (courtesy of Veronica J. Rooks, MD, Tripler Army Medical Center, Honolulu HI).

galea *a*poneurotica, *l*oose or subga*l*eal space, *p*eriosteum]).[2,4,5,10] Although CT or MRI may not precisely resolve scalp layers, the site of a collection may be inferred by means of morphological findings (Fig. 1). Subperiosteal collections (eg, cephalohematoma) are usually confined by the sutures. Subcutaneous or subgaleal collections are not as contained, may be more extensive, and can contribute to circulatory compromise. Scalp injuries are difficult to precisely time on imaging studies, unless serial examinations are available; in addition, timing depends on the nature and the number of traumatic events or other factors (eg, circulatory compromise). Unless there is direct vascular injury that results in an acute hematoma, collections or edema may not be identified on early imaging. Scalp injuries may become evident several hours later or on the next day. Nonvisualization of scalp or skull abnormalities on imaging should not be interpreted as absence of impact injury.

SKULL INJURY

The spectrum of cranial injury includes Fx*s* and suture splitting.[2,4,5,10] Fractures may be simple (eg, single, linear, nondisplaced) or complex (eg, bilateral, multiple, diastatic, depressed, or growing [ie, leptomeningeal cyst]). Localized suture splitting may indicate traumatic diastasis where widening occurs as a part of Fx extension. Diffuse or multiple suture widening may indicate increased intracranial pressure from any cause to include edema, expanding collection, or hydrocephalus. Evaluating the skull in neonates, infants, and young children is challenging because Fx may not be distinguished from sutures, synchondroses, or their normal variations. This is particularly difficult in the parietooccipital region and skull base where accessory sutures, fissures, and synchondroses are common. The significance of this distinction is important because the reporting of a skull Fx is *evidence* of trauma (Fig. 1). In such cases, 3-dimensional computed tomography with surface reconstructions may provide clarification (Fig. 4). In general, the morphology of an Fx does not differentiate NAI from AI. Complex or bilateral skull Fx in this age group can arise from a single event under circumstances other than a 2-story fall or a motor vehicle accident. Such examples include a fall or a drop with impact to the skull vertex, impact against more than 1 surface (eg, table, wall, or floor), fall or drop downstairs, and an adult or older child falling with or onto a smaller child. Skull Fx*s* are also difficult to time by using plain films and CT because of the lack of periosteal reaction during healing. A simple skull Fx in an infant may require 6 months for complete healing. In an older child and adult, this may take up to a

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year.[2,4,5,10] Intracranial air densities (ie, pneumocephalus) may be related to fracture involving the paranasal sinuses or otomastoid structures, caused by penetrating trauma (eg, open skull fracture), arise from CSF access (eg, lumbar puncture) or vascular access (eg, indwelling catheter), or may be associated with gas-forming infections.

EXTRACEREBRAL COLLECTIONS

The range of intracranial injury includes abnormal fluid collections and brain injury.[2,4,5,10] Abnormal collections may be subarachnoid, intraventricular, subdural, or epidural. These may contain hemorrhage of any age (eg, hyperacute, acute, subacute, chronic, combined), cerebrospinal fluid (CSF [eg, hygroma, hydrocephalus]), protein, exudate, or any combination of elements. On imaging, it may be impossible to specifically define the components or age of a collection (eg, SDHG vs chronic SDH). Subarachnoid and subdural collections may be localized or extensive and occur near the convexities, interhemispheric (along the falx), and along the tentorium. Epidural hemorrhage, whether arterial or venous in origin, tends to be more localized (limited by the periosteal layer of the dura mater along the inner calvarial table) and can cross midline (Fig. 1). Epidural (intradural) hemorrhage may split the leaves of dura and collect within the tentorium or falx. Epidural collections usually appear lentiform. Subdural collections tend to be crescentic and follow the contour of the adjacent cerebrum or cerebellum (Fig. 3). Subarachnoid collections may be less well defined (unless loculated) and extend into cisterns, fissures, or sulci. Occasionally, a collection cannot be determined to be specifically subarachnoid, subdural, or epidural because collections in multiple spaces may be present, owing to membrane layer disruption (Fig. 2). Intraventricular hemorrhage is a rare but reported finding in trauma. It may also be an indicator of associated hypoxia-ischemia, coagulopathy, or venous thrombosis.

Prominent subarachnoid CSF spaces may normally be present in infants (aka benign extracerebral collections [BECC], benign extracerebral subarachnoid spaces, benign external hydrocephalus).[10,79–83,114] These should be of the same density/intensity as CSF on CT and MRI (Fig. 5). This condition predisposes infants to SDH, which may be spontaneous or associated with trauma of any type (Fig. 5). A hemorrhagic collection may continually change or evolve with regard to size, extent, location, and density/intensity characteristics. Cases of rapid resolution and redistribution of acute SDH for a few hours to 1 to 2 days have been reported.[117,122] A tear in the arachnoid may allow SDH washout into the subarachnoid space or CSF dilution of the subdural space. An SDH may also redistribute within the subdural space as a gravity-dependent process (eg, a convexity SDH migrating to the peritentorial and posterior interhemispheric regions)[114,117] (Fig. 6). Subdural hemorrhage migration may lead to misinterpretation of a new hemorrhage. The distribution or migration of the sediment portion of a hemorrhage with blood levels (ie, hematocrit effect) may

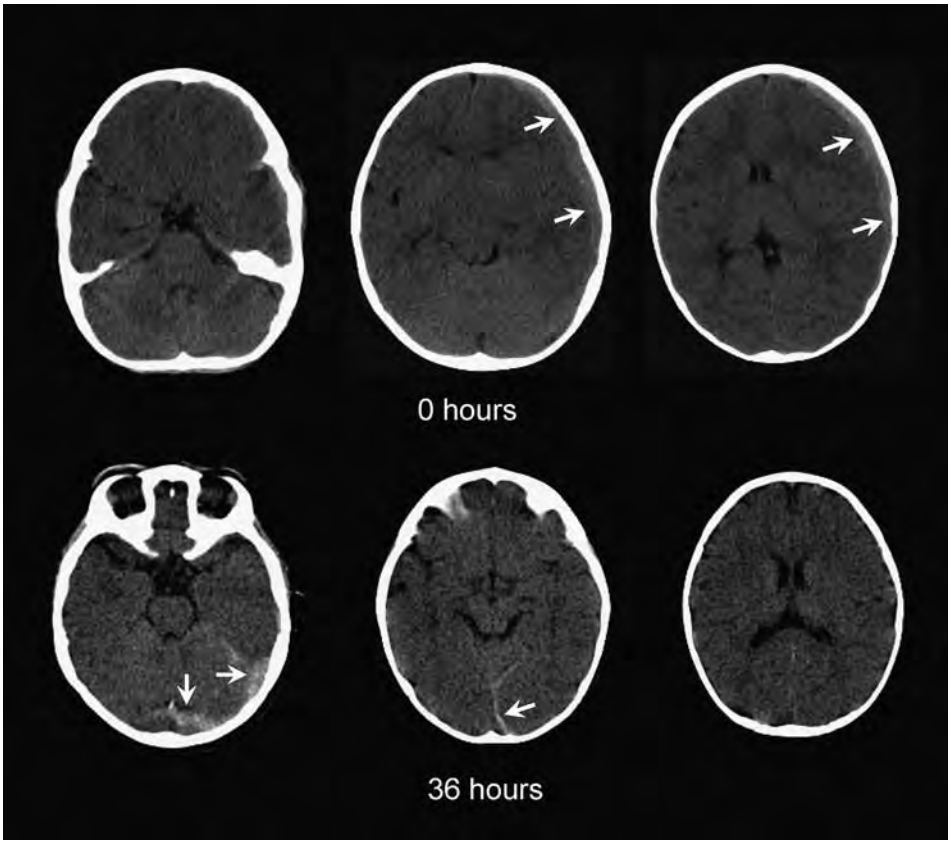

FIGURE 6. Images obtained from a 9-month-old female infant who had accidental trauma from left-side frontal impact. Computed tomographic images at presentation (top row) show left-side frontotemporal-convexity high-density subdural hemorrhage (arrows). Computed tomographic images obtained after 36 hours in the hospital (bottom row) show redistribution of the high-density hemorrhage to the peritentorial region and posterior interhemispheric fissure (arrows).

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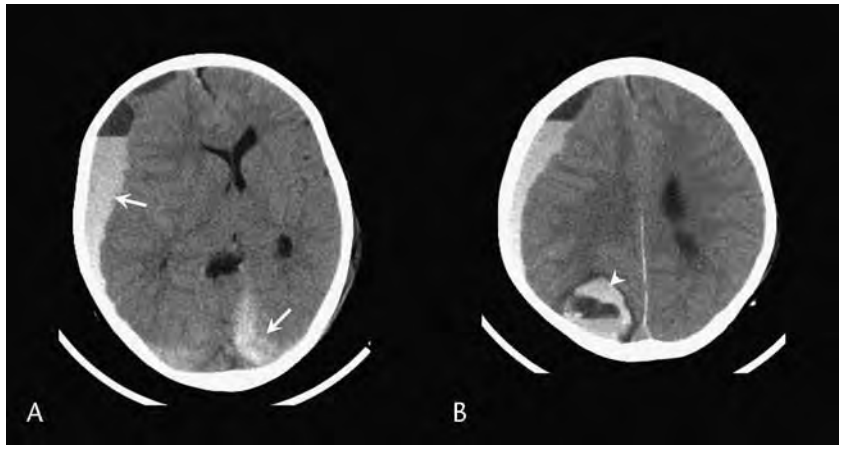

FIGURE 7. Images obtained from a 2-year-old boy with congenital heart disease and ECMO. Axial computed tomographic images show bilateral subdural hematomas (A, arrows) and right-side parietal intracerebral hematoma (B, arrowhead) with low-density over high-density fluid levels.

cause further confusion because the density/intensity differences between the sediment and supernatant may be misinterpreted as hemorrhages (and trauma) of differing age and location (Figs. 7, 8).[117] In addition, more recent reports further substantiate that (1) the interhemispheric SDH may be observed in AI and, therefore, is not specific for NAI; (2) mixed-density SDH also occurs in AI; (3) SDH may occur in BECC either spontaneously or as a result of minor trauma (ie, AI); and (4) rehemorrhage within SDH may occur spontaneously or with minor AI.[10,82,114–118]

BRAIN INJURY

Traumatic brain injury includes contusion, shear injury, hemorrhage, and edema.[2,4,5,10] Contusions represent focal or multifocal impact injury, are usually hemorrhagic, and typically occur in cortical gray matter along brain surfaces that impact skull bone or dura mater (eg, falx, tentorium). The inner table of the immature, infant skull is not as *rough* as in older children and adults. Therefore, sliding contusions of the frontal or temporal lobes along the floor of the anterior or middle cranial fossa, respectively, occur less often. Infant contusions more commonly occur at the primary site of impact (ie, coup injury) or at a secondary, "recoil" site opposite the primary impact (ie, contracoup injury). Shear injury (ie, traumatic axonal injury, white matter tear) is also focal or multifocal and typically occurs at deep gray matter–white matter junctions, along the corpus callosum, and within the brain stem (Fig. 3). They are more often nonhemorrhagic but may become hemorrhagic. In severe cases, shear injuries may appear as gross tears. This type of injury has been previously referred to as *diffuse axonal injury* or DAI. It is more properly termed *multifocal or traumatic axonal injury* because diffuse axonal injury is more characteristic of hypoxic-ischemic injury (Fig. 2).[104–109]

Edema or swelling may be traumatic, hyperemic, hypoxic-ischemic, or related to other factors (eg, seizures, metabolic).[2,4,5,10] Traumatic edema is related to direct traumatic effects such as contusion, shear, or the result of a vascular injury (eg, dissection, herniation) (Figs. 2, 3). Malignant brain edema, a term used for severe cerebral swelling leading to rapid deterioration, may also occur in children with head trauma. The edema may be related to cerebrovascular congestion (ie, hyperemia) as a vasoreactive rather than an autoregulatory phenomenon. There may be rapid or delayed onset.[84–96] Predisposing factors are not well established but likely include a genetic basis. Global hypoxia (eg, apnea, respiratory failure) or ischemia (eg, cardiovascular failure or dissection) is likely a major cause of or contributor to brain edema in the child with head trauma (Fig. 2). Other contributors to edema or swelling include such complicating factors as seizures (eg, status epilepticus), fluid-electrolyte imbalance, other systemic or metabolic derangements (eg, hypoglycemia, hyperglycemia, hyperthermia), or hydrocephalus. The type (eg, cytotoxic, vasogenic, hydrostatic) and pattern of edema tend to conform to the nature and distribution of the causative insult. Traumatic edema is often focal or multifocal (eg, in areas of contusion, shear, or hemorrhage) (Fig. 3). Hyperemic edema is often diffuse and may appear early as accentuated gray-white matter differentiation on CT, then progressing to loss of differentiation (Fig. 2). Hypoxic-ischemic injury, depending on its severity and duration, may have a diffuse appearance acutely with decreased gray-white matter differentiation throughout the cerebrum on CT (eg, white cerebellum sign) and then evolve to a more specific pattern on CT or MRI (eg, border zone or watershed, basal ganglia/thalamic, cerebral white matter necrosis, reversal sign) (Fig. 2).[10,114,123–126] The subacute to chronic sequelae of traumatic brain injury include hydrocephalus, atrophy, encephalomalacia, gliosis, mineralization, and chronic extracerebral collections.

VASCULAR INJURY

Arterial trauma may result in dissection or pseudoaneurysm.[2,4,5,10,123, 127] The vascular injury may be the result of penetrating or nonpenetrating trauma, may be spontaneous, or caused by existing disease (eg, arteriopathy). Internal carotid artery dissection typically involves the cervical or supraclinoid segments. Vertebrobasilar dissection most commonly involves the distal cervical portion of the vertebral artery at the C1-C2 level. Intracranial or multiple dissections may rarely occur. Dissection may result in stenotic, thrombotic, or embolic infarction. Pseudoaneurysms may be associated with hemorrhage. The vascular injury may be initially detected by means of CT and CTA (Fig. 9) or of MRI (eg, DWI, axial fat-suppressed T1 sections of the neck and skull base) with MRA. Catheter

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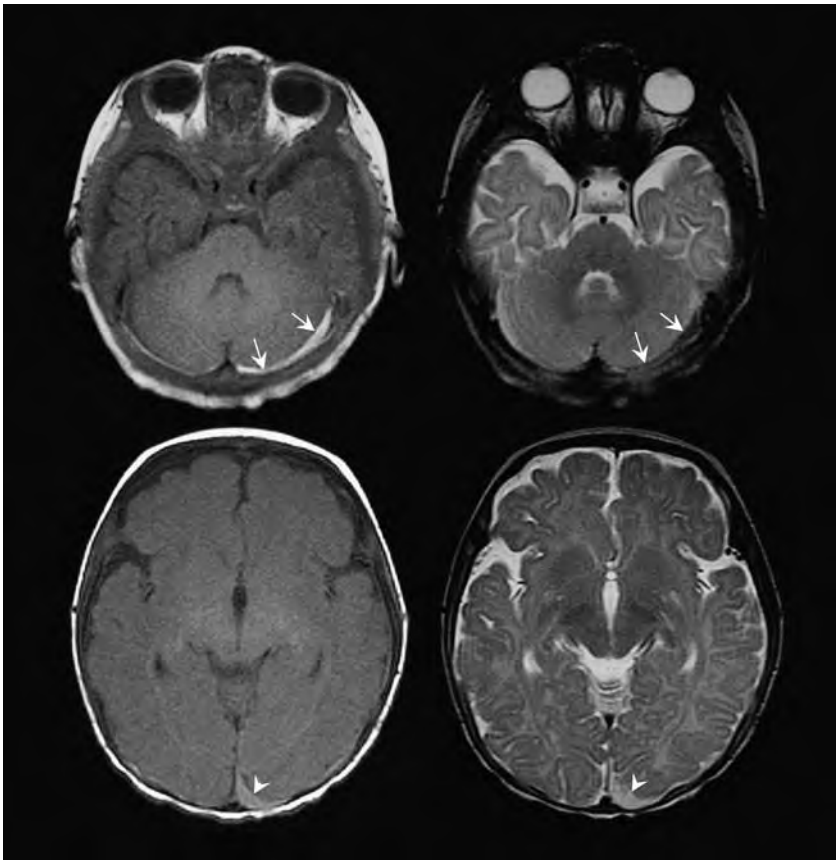

FIGURE 8. Images obtained from a 2-month-old female infant with left-side peritentorial and posterior interhemispheric subdural hemorrhage. Axial MRI images show T1-hyperintense and T2-hypointense sediment along the tentorium (top row, arrows) with T1- and T2-isohyperintense supernatant above (bottom row, arrowheads).

angiography may be necessary for definitive evaluation. Arterial occlusive infarction also occurs with the various types of herniation, in which relatively specific distributions are observed. Dural sinus and venous thrombosis may also occur with trauma (eg, adjacent to fracture, associated or predisposing coagulopathy) or as a mimic of NAI (eg, infection, coagulopathy).[128] Computed tomography may show hyperdensity within the venous system, a focal venous enlargement with associated subarachnoid or subdural hemorrhage, or infarction that is often hemorrhagic. A more definitive diagnosis may be made by means of CTV or of MRI and MRV.

SPINAL INJURY

The spectrum of spinal injury in NAI significantly overlaps that of AI.[2,4,5,10,123] This spectrum differs with age (degree of spinal development) and includes either single or

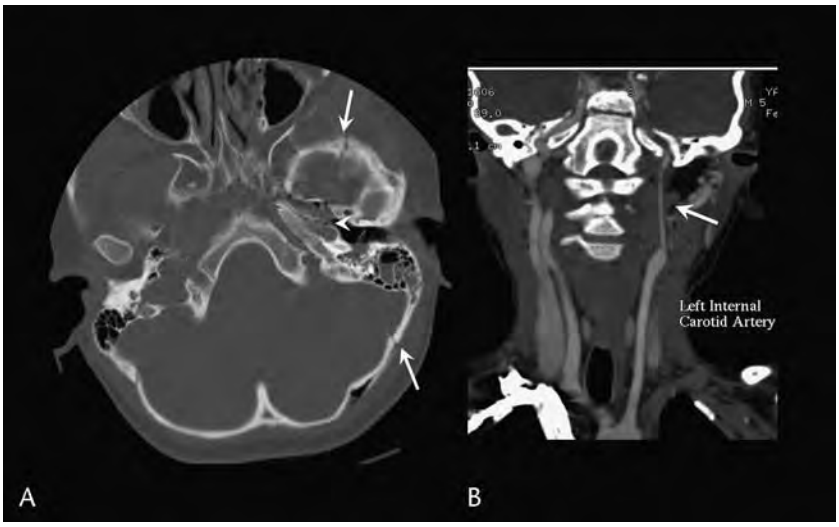

FIGURE 9. Images obtained from a 5-year-old boy. A, Computed tomographic image shows left-side skull base fractures involving left-side occiput, petrous bone, and sphenoid wing (arrows). Air densities are seen within the carotid canal (arrowhead). B, Computed tomography angiogram shows left-side cervical internal carotid arterial dissection with marked luminal narrowing (arrow).

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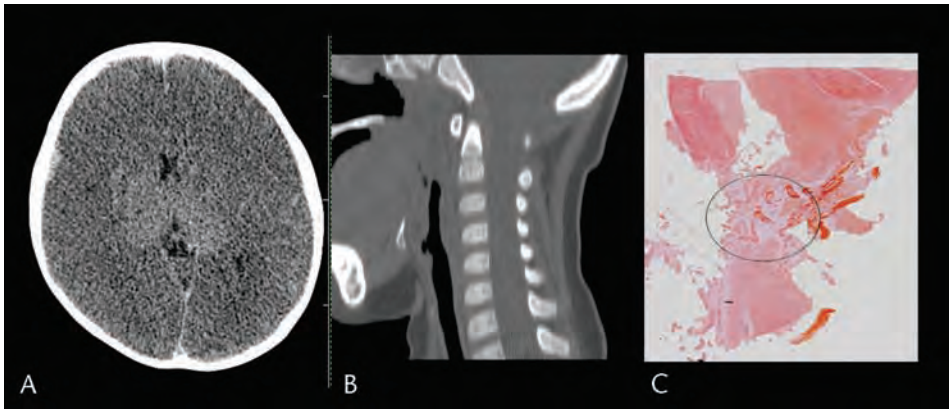

FIGURE 10. Images obtained from a 22-month-old boy with SCIWORA (caused by backward fall and parietal head impact) and hypoxic-ischemic injury and RHs. A, Axial brain CT image shows (1) bilateral cerebral low densities with decreased gray-white matter differentiation (edema) and (2) small high-density asymmetrical cerebral, extracerebral, and posterior interhemispheric hemorrhages. B, Sagittal reformatted cervical spinal computed tomographic image shows no spinal column abnormality (MRI not performed). C, Postmortem midsagittal section shows cervicomedullary disruption (circle). Diffuse hypoxic-ischemic axonal brain injury was also confirmed.

multiple lesions involving the cervical, thoracic, lumbar, or sacral level. The mechanisms of injury include hyperflexion, hyperextension, axial loading or rotation, and distraction. The range of spinal column and paraspinal injury includes vertebral or neural arch fractures, bony fragment or disk displacement, dislocations, instability, and paraspinal ligamentous, muscular, or vascular injury. Such injuries may not be apparent on plain films (eg, spinal cord injury without radiographic abnormality [SCIWORA]) and require additional CT plus MRI for complete evaluation.[129–131] Magnetic resonance imaging is particularly important for evaluating ligamentous injury and intraspinal injury. The range of intraspinal injury includes displaced bone or disk fragments and hematomas (eg, epidural) with spinal cord or nerve root compression. There may be edema, contusion, hemorrhage, transection of the spinal cord, or avulsion of 1 or more nerve roots. Computed tomographic angiography or MRA may be needed to evaluate vascular injury (eg, dissection). Cervical spinal cord injury may be associated with head injury or may be the unsuspected cause of respiratory failure and hypoxic-ischemic brain injury (eg, SCIWORA) (Fig. 10).[129–131] This should be evaluated by means of MRI in all such cases, whether AI or NAI. In addition, one must be aware of predisposing conditions that may result in major neurological deficits associated with *minor* head and neck trauma mechanisms (eg, craniocervical anomaly with instability Fig. 11; Chiari I malformation Fig. 12).

IMAGING ANALYSIS—COMPUTED TOMOGRAPHY

Regarding the initial computed tomographic examination, the findings are often nonspecific with regard to pattern of injury and timing and require a differential diagnosis (DDX). To properly analyze such a case from an imaging perspective, each injury component must be addressed separately, and then collectively, and then correlated with clinical and other data.[4,10,114] The major findings are often (1) extracerebral and cerebral high densities, (2) extracerebral isohypodensities, (3) cerebral low densities, with or without (4) scalp or skull abnormalities. In general, the DDX may include trauma (AI vs NAI), hypoxia-ischemia, ischemic injury (arterial vs venous occlusive disease), seizure edema, infectious or postinfectious conditions, coagulopathy, fluid-electrolyte derangement, metabolic or connective tissue disorder, and multifactorial.

Extracerebral high densities are often seen posteriorly along the tentorium, falx, interhemispheric fissure, and dural

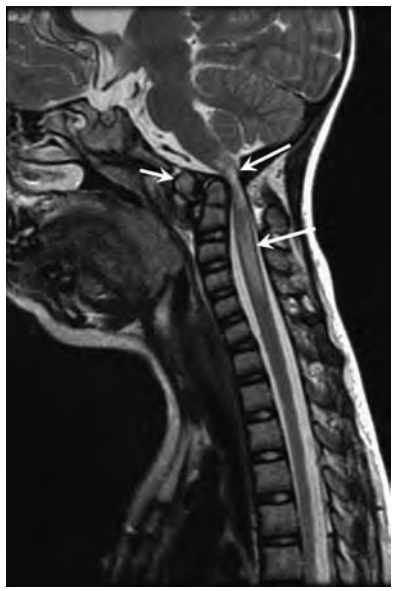

FIGURE 11. Image obtained from an 8-year-old girl with Down syndrome and minor trauma with quadriparesis. Sagittal T2 MRI scan shows hypoplastic dens, os odontoideum (anterior arrow), and anterior atlantoaxial instability (confirmed by means of CT) with cervicomedullary compression and high-intensity edema (posterior arrows).

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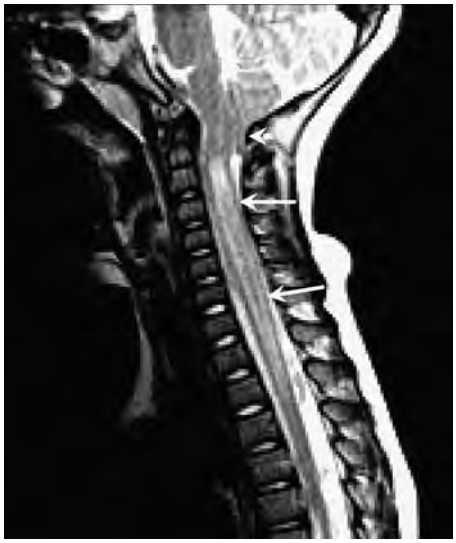

FIGURE 12. Image obtained from a 3-year-old boy with Chiari I malformation, minor trauma, and subsequent quadriparesis. Sagittal T2 MRI scan shows cerebellar tonsils extending into the upper cervical canal (upper arrowhead) and diffuse high-intensity edema of the cervical spinal cord (lower arrows). No abnormality was present on plain films or CT (SCIWORA).

venous sinuses that may vary in laterality and symmetry (Figs. 2, 6, 7, 10, 13–16). These and other extracerebral high densities may be laminar, linear, nodular, or punctate. Using published criteria and timing parameters (discussed in the succeeding sections), these represent either acute to subacute hemorrhages (subarachnoid, subdural) or thromboses (eg, venous).[4,10,114–118] For apparent intracerebral high densities, it may be difficult to differentiate cerebral from SAHs (including those within the perivascular spaces) from vascular thromboses (eg, cortical, subependymal, or medullary venous thromboses). Computed tomography may not be able to distinguish focal or multifocal cerebral high densities as hemorrhagic contusion, hemorrhagic shear, or hemorrhagic infarction (Figs. 13, 16, 18). Extracerebral isohypodensities may represent subarachnoid spaces (eg, BECC), SDHG, hyperacute SDH, or chronic SDH (Figs. 14, 17). According to the literature, the timing for any of the mentioned findings is as follows: (1) hemorrhage or thromboses that are high density (ie, clotted) on CT (ie, acute to subacute) have a wide timing range of 3 hours to 7 to 10 days (Figs. 1, 2, 6, 7, 10, 13–18), (2) hemorrhage that is isohypodense on CT (ie, nonclotted) may be hyperacute (timing, <3 hours) or chronic (timing, >10 days) (Figs. 14, 17), (3) the low density may also represent preexisting wide, CSF-containing subarachnoid spaces (eg, BECC) or SDHG (ie, CSF containing) that may be acute or chronic (Figs. 14, 17), (4) blood levels are unusual in the subacute unless there is coagulopathy (Fig. 7), (5) CT cannot distinguish acute hemorrhage from rehemorrhage on existing chronic collections (BECC or chronic SDHG) (Fig. 17), and (6) the interhemispheric SDH is no longer considered characteristic of NAI (Figs. 2, 6, 7, 13–16).[4,10,114–118]

Cerebral low densities may vary in bilaterality and symmetry and be associated with decreased gray-white matter differentiation or mass effect (Figs. 2, 10, 17). In general, this indicates edema/swelling, the timing of which depends on causation. If related to trauma, such edema/swelling may represent primary injury or secondary injury and be acute-hyperacute (eg, timing of few hours) or delayed (eg, timing of several hours to a few days), including association with lucid interval and short falls.[4,10,114,123–126] Bilateral diffuse edema is most commonly observed in hypoxia-ischemia but may also be observed in other diffuse processes (eg, fluid-electrolyte imbalance, status epilepticus, encephalitis, etc). Focal or multifocal edema may be observed in contusion (eg, gray matter), shear (eg, white matter), infarction (gray or white matter), encephalitis, or demyelination (eg, acute disseminated encephalomyelitis).

Cranial defects may represent Fx, and their timing range is very broad (eg, hours to months old) (Fig. 1).[4,10,114] Furthermore, Fx morphology (eg, multiple, growing) does not reliably distinguish accidental from nonaccidental causation. Scalp collections (hemorrhage, edema, blood level) are also nonspecific with regard to causation and timing (Fig. 1).[4,10,114] If caused by trauma, the timing range is also rather broad (eg, hours to days old). Sutural widening may indicate diastatic Fx

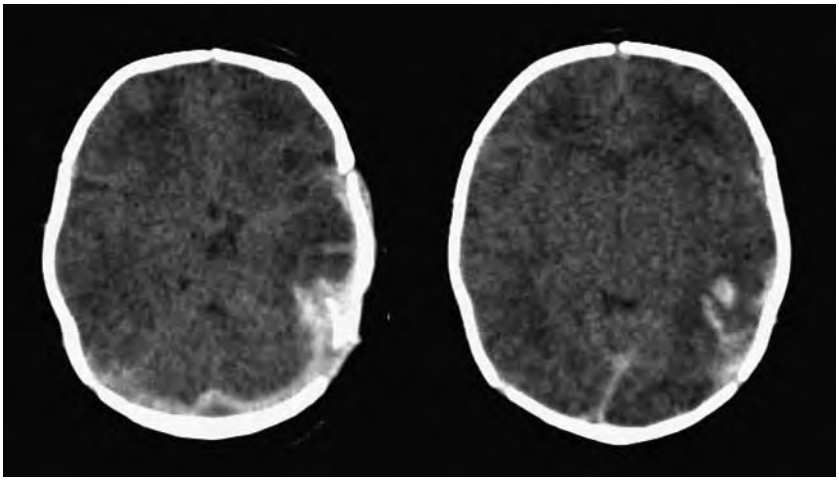

FIGURE 13. Images obtained from a 1-day-old female infant delivered by means of spontaneous vaginal delivery and with subsequent apneic episodes. Computed tomography demonstrates left-side temporal cerebral and extracerebral high-density hemorrhage (or thromboses); high-density hemorrhage is also demonstrated along the interhemispheric fissure, tentorium, and dural venous sinuses. The results of coagulopathy test and sepsis workup were negative (final diagnosis, birth trauma?).

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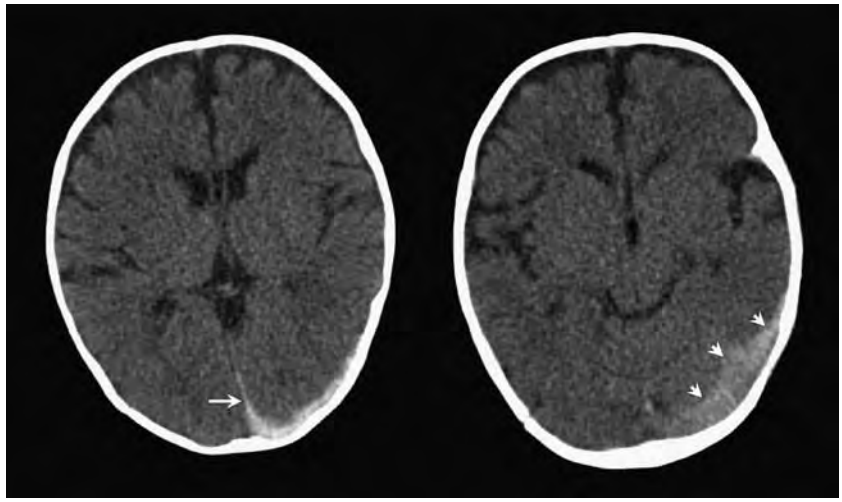

FIGURE 14. Images obtained from a 4-month-old male infant with 2-week viral illness who progressed to septic shock (*Staphylococcus aureus*), endocarditis, severe mitral regurgitation, and coagulopathy. Noncontrast axial CT images show high-density extracerebral hemorrhages (and/or thromboses) along the left-side tentorium, dural venous sinuses, falx, and interhemispheric fissure (arrows). In this case, the bifrontal low-density extracerebral spaces likely represent slightly prominent infantile subarachnoid spaces (BECC?) or underdevelopment, rather than chronic SDH or subdural hygroma.

or increased intracranial pressure. Accessory sutures or synchondroses and developmental fissures may mimic Fx. Intrasutural bones (eg, wormian) may be associated with a skeletal dysplasia or metabolic disorder (Fig. 4).

Subsequent or follow-up computed tomographic examinations may show surgical changes (eg, postevacuation, ventricular catheter, pressure-monitoring device), evolving, redistributing, or recurrent/new hemorrhages, and evolving cerebral densities (edema/swelling). Subsequent CT examinations during the weeks or months may show evolution to permanent cerebral tissue loss (ie, atrophy, encephalomalacia).

IMAGE ANALYSIS—MAGNETIC RESONANCE IMAGING

On an imaging basis, only MRI may provide more precise information regarding pattern of injury and timing, particularly with regard to (1) hemorrhage versus thromboses, and (2) brain injury. The MRI should be performed as soon as feasible, and the findings be compared with the findings from the earlier CT. As a result, MRI has become the standard for such evaluation in these matters.[4,10,114–117,121,123–126]

Hemorrhages and Thromboses

Using published MRI guidelines (Table 1), in general, the evolutionary timing for hemorrhages or thromboses (eg, venous) are as follows: (1) hyperacute phase (timing, <12 hours): T1 isohypointense, T2 hyperintense; (2) acute phase (timing, 1–3 days): T1 isohypointense, T2 hypointense; (3) early subacute phase (timing, 3–7 days): T1 hyperintense, T2 hypointense; (4) late subacute phase (timing, 7–14 days): T1 hyperintense, T2 hyperintense; (5) early chronic phase (timing, >14 days): T1 hyperintense, T2 hyperintense; (6) late chronic phase (timing, >1 to 3 months): T1 isohypointense, T2 hypointense.[4,10,114–117,121,123–124] Mixed intensity collections are problematic regarding timing. Matching the MRI findings with the computed tomographic findings may help, along with follow-up MRI. Blood levels may indicate subacute hemorrhage versus coagulopathy. The timing guidelines are better applied to the sediment than to the supernatant. In addition, a single MRI may not reliably differentiate T1-hypointense/T2-hyperintense collections as representing CSF collections (eg, BECC, acute SDHG) versus hyperacute SDH versus chronic collections (SDH, SDHG). Gradient-recalled echo hypointensities are iron sensitive but do not assist with timing unless matched with

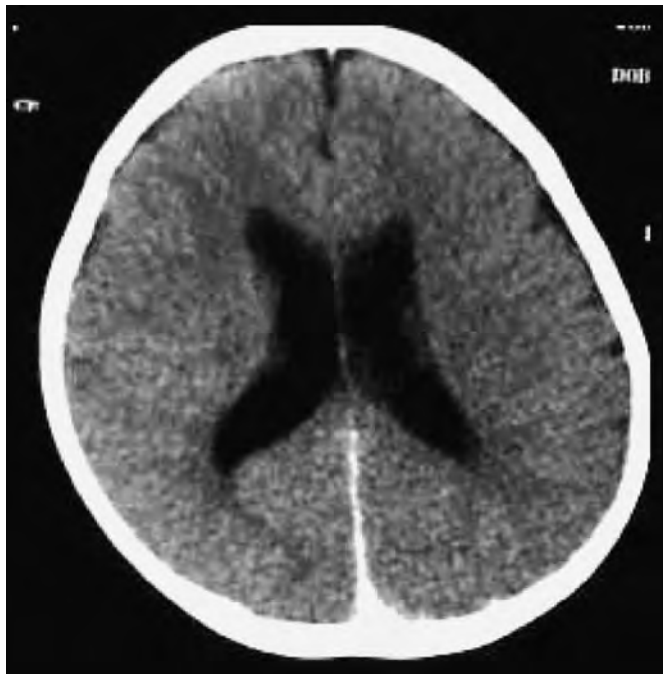

FIGURE 15. Image obtained from a 23-month-old girl who had recent viral gastrointestinal illness, ALTE, RHs, then brain death. Computed tomographic image shows posterior interhemispheric high densities at the level of portions of the inferior sagittal, straight, and superior sagittal sinuses, plus poor cerebral gray-white matter differentiation and moderate ventriculomegaly. Autopsy showed extensive dural and cerebral venous sinus thrombosis with extensive hypoxic-ischemic diffuse axonal brain injury.

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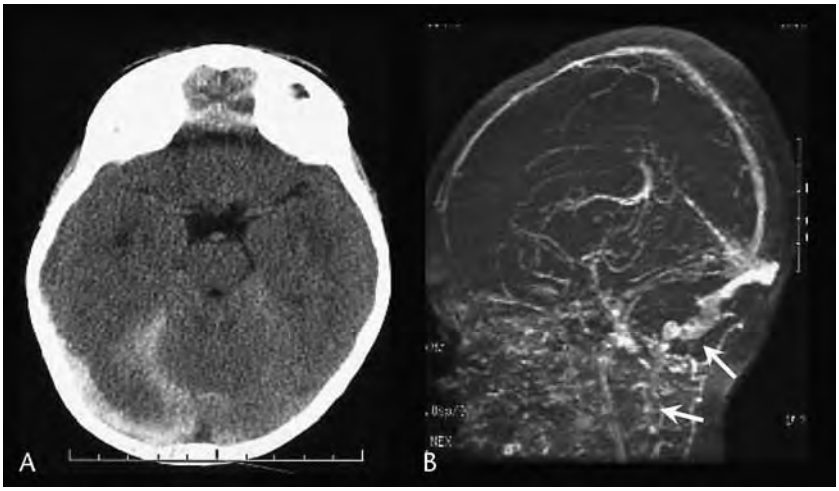

FIGURE 16. Images obtained from a 19-month-old boy who had 1 week of febrile illness (treated with antibiotics), followed by ALTE with RHs. A, Computed tomographic image shows high-density hemorrhages (or thromboses) along the right tentorium and dural venous sinuses. B, Magnetic resonance imaging with MRV shows irregular flow gaps with incomplete opacification of the right-side internal jugular vein and sigmoid sinus. Other flow gaps were demonstrated within the superior sagittal and straight sinuses, along with multiple venous collaterals (diagnosis, DVST).

T1, T2, and computed tomographic densities. Gradient-recalled echo and other magnetic susceptibility sequences are also sensitive to venous thromboses (eg, cortical, medullary, subependymal) that are not detected by means of MRV.

Brain Injury

With regard to brain injury, MRI may distinguish hypoxic-ischemic injury (diffuse relatively symmetrical DWI/ADC restricted diffusion with or without matching T1/T2 abnormalities) from shear and contusional injury (focal/multifocal restricted diffusion, GRE hypointensities, with T2/FLAIR edema). Shear and contusional injury, however, may not be reliably differentiated from focal/multifocal ischemic or hemorrhagic infarction (eg, dissection, vasculitis, venous, embolic) without supportive MRA, CTA, MRV, or angiography.[4,10,114,123–125] In addition, similar cortical or subcortical intensity abnormalities (including restricted diffusion) may also be observed in encephalitis, seizures, and metabolic disorders. Using published MRI criteria and parameters,[114,123–126] in general, the evolutionary timing for ischemic injury is as follows: (1) hyperacute phase (timing, <1 day): DWI hyperintense, ADC hypointense; MRS result, lactate peak; (2) early acute phase (timing, 1–2 days): additional T2 hyperintensity; (3) late acute phase (timing, 2–4 days): additional T1 hyperintensity; (4) early subacute phase (timing, 6–7 days): additional T2 hypointensity; (5) late subacute phase (timing, 7–14 days): additional DWI isohypointense, ADC isohyperintense; (6) chronic phase (timing, >14 to 21 days): additional atrophy. If related to trauma, focal/multifocal ischemic findings may be caused by arterial injury (eg, dissection), venous injury (eg, tear, thrombosis), arterial spasm (as with any cause of hemorrhage), herniation, or edema with secondary perfusion deficit or seizures (eg, status epilepticus). Hypoxia-ischemic brain injury caused by apnea/respiratory arrest may occur with head trauma or with neck/cervical spine/cord injuries (eg, SCIWORA), whether AI or NAI.[114,123,129–131] It may also occur with any nontraumatic cause (eg, choking, paroxysmal coughing, aspiration).[132] In addition to the diffuse brain injury, there may be associated subarachnoid and subdural hemorrhage without mass effect.[104–109]

CONDITIONS MIMICKING NONACCIDENTAL INJURY

Traumatic and nontraumatic conditions may mimic the clinical presentations (ie, the triad) and imaging findings of NAI. These include accidental trauma (as previously discussed), birth trauma, hypoxia-ischemia, cardiopulmonary

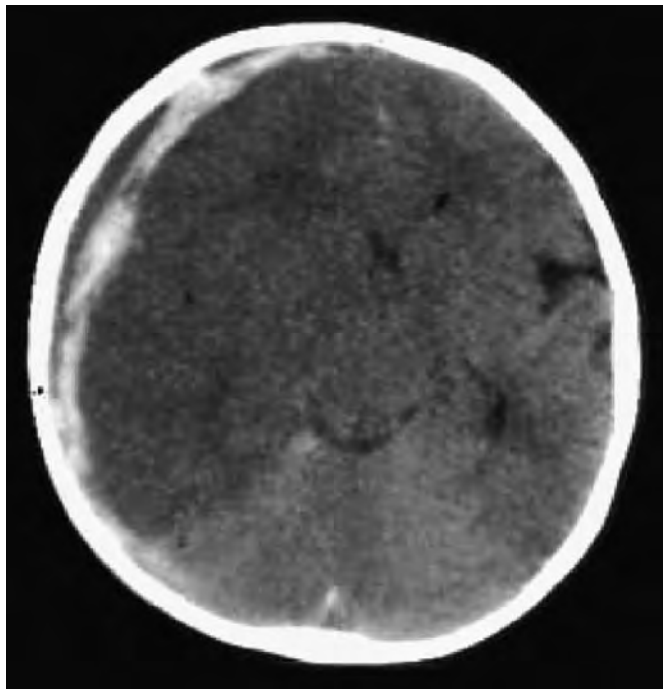

FIGURE 17. Image obtained from an 8-month-old male infant who had ALTE, right-side occipital skull fracture (not shown), a healing right-side distal radial fracture, and then had brain death. Computed tomographic image shows a right-side, mixed density extracerebral collection with right-side cerebral low density, mass effect, and leftward shift. High-density hemorrhages (or thromboses) are also present along the tentorium. There was disagreement among the forensic experts regarding hyperacute-acute SDH versus chronic SDH with rehemorrhage.

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resuscitation, infectious or postinfectious conditions (eg, sepsis, meningoencephalitis, postvaccinial), vascular diseases, coagulopathies, venous thrombosis, metabolic disorders, neoplastic processes, certain therapies, extracorporeal membrane oxygenation (ECMO), and other conditions.[4,5,10,114,115,133] Regarding the pathogenesis of the triad (with and without other organ system involvement [eg, skeletal]), and whether caused by NAI, AI, or nontraumatic etiologies, the pathophysiology seems to be some combination or sequence of factors, including increased intracranial pressure, increased venous pressure, systemic hypotension or hypertension, vascular fragility, hematologic derangement, and/or collagenopathy superimposed on the immature CNS and other systems.[107,115,123,132–146]

Although the initial medical evaluation, including history, laboratory tests, and imaging studies, may suggest an alternative condition, the diagnosis may not be made because of a *rush to judgment* regarding NAI. It is important to be aware of these mimics because a more extensive workup may be needed beyond the routine *screening* tests. In addition, the lack of confirmation of a specific condition does not automatically indicate the *default* diagnosis of NAI. In all cases, it is critical to review all records dating back to the pregnancy and birth, the postnatal pediatric records, the family history, the more recent history preceding the short-term presentation, the details of the short-term event itself, the resuscitation, and the subsequent management, all of which may contribute to the clinical and imaging findings.[4,5,10,115,133]

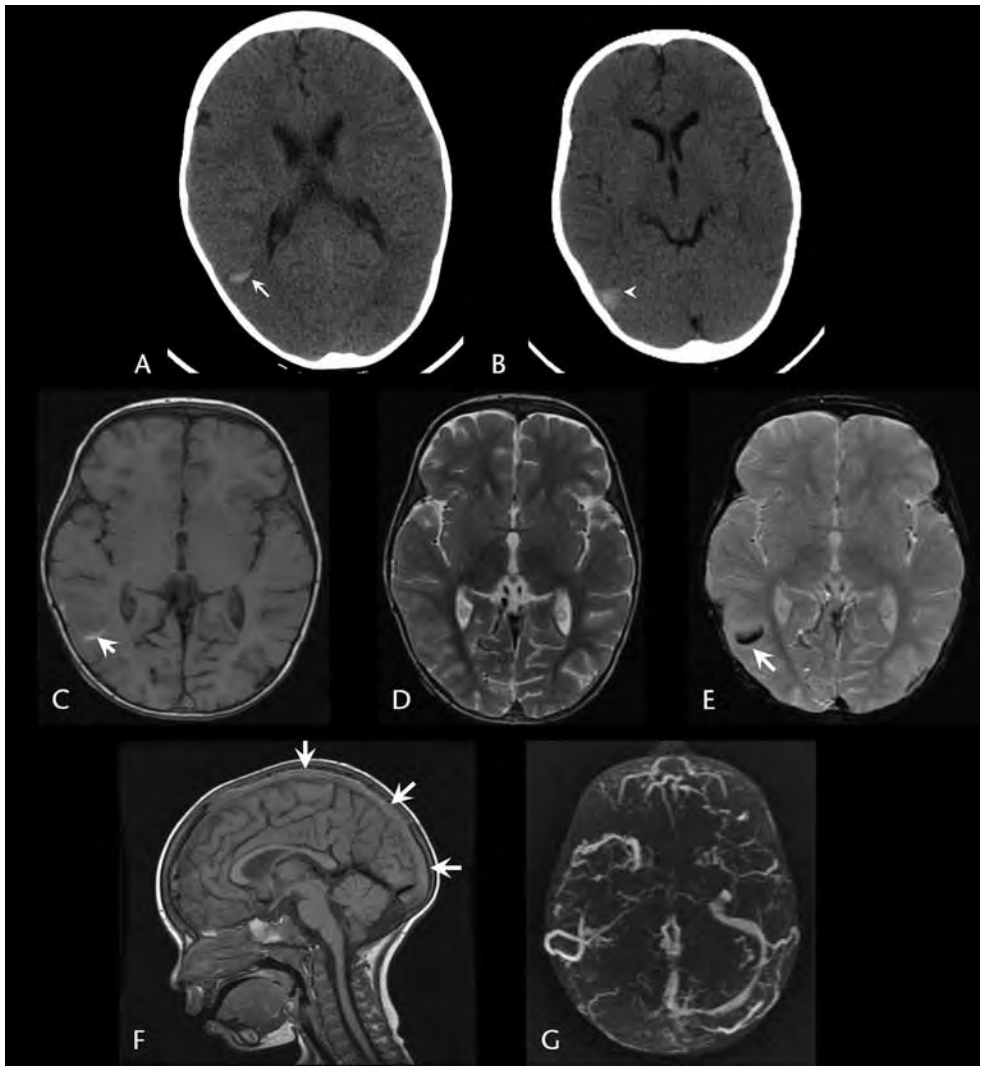

FIGURE 18. Images obtained from a 22-month-old boy who experienced lethargy, vomiting, and seizures after a viral illness, plus thrombocytopenia and iron deficiency anemia. A–B, Computed tomographic images show right-side posterior temporal and peritentorial high-density foci of hemorrhage or thrombosis (arrows). Axial T1 (C), T2 (D), and GRE (E) images show corresponding T1-hyperintense and GRE-hypointense foci with associated T2 hyperintensity (arrows). F, Sagittal T1 MRI scan shows hyperintensity along the superior sagittal sinus (arrows [thrombosis vs slow flow]). G, Axial MRV projection image shows nonvisualization of the superior sagittal, right-side transverse, and right-side sigmoid sinuses (diagnosis, postviral dural and cerebral venous thrombosis [extensive coagulopathy workup continues]).

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A recent review presented by Sirotnak[133] extensively catalogues the many conditions that may mimic abusive head trauma. These include perinatal conditions (birth trauma and congenital malformations), accidental trauma, genetic and metabolic disorders, hematologic diseases and coagulopathies, infectious diseases, autoimmune and vasculitic conditions, oncological disease, toxins, poisons, nutritional deficiencies, and medical and surgical complications. The reader is encouraged to read this review.[133] An abbreviated discussion is presented in this article along with some examples.

Birth Trauma and Neonatal Conditions

Manifestations of birth trauma, including Fx, SDH, and RH, may persist beyond the neonatal period and mimic CNS findings of abuse.[145–151] Other examples are the cases of infants following ECMO therapy, at-risk preterm neonates, and infants with congenital heart disease.[4,5,10,123,124,152] When evaluating the condition of a young infant with apparent NAI, it is important to consider that the clinical and imaging findings may actually stem from parturitional and neonatal issues. This includes hemorrhage or rehemorrhage into collections existing at birth (Figs. 5, 8, 13).

Developmental Anomalies

Vascular malformations of the CNS in neonates and infants are relatively rare.[115,133,153,154] The most common are the vein of Galen malformations. Aneurysms are also rare in childhood but may arise within the circle of Willis. Aneurysms outside the circle are usually mycotic or traumatic in origin. Increased risk of aneurysm is associated with certain conditions, such as coarctation of the aorta, polycystic kidney disease, neurofibromatosis, and a family history positive for aneurysm. A number of syndromes in childhood are associated with vascular anomalies and may present with intracranial hemorrhage. These syndromes include, as examples, PHACE (*p*osterior fossa brain malformations, *h*emangiomas, *a*rterial anomalies, *c*oarctation of the aorta, *c*ardiac defects, and *e*ye abnormalities), Sturge-Weber, Beckwith-Wiedemann, Klippel-Trenaunay-Weber, Maffucci, and Olser-Weber-Rendu. Arachnoid cysts are also known to be associated with SDH and RH, spontaneously and with trauma (Fig. 19).[133,155]

Genetic and Metabolic Disorders

A number of conditions in this category may present with intracranial hemorrhage (eg, SDH) or RH. These include osteogenesis imperfecta, glutaric aciduria type I, Menkes kinky hair disease, Ehlers-Danlos and Marfan syndromes, homocystinuria, and others (Fig. 19).[115,133,135,136,156]

Hematologic Disease and Coagulopathy

Many conditions in this category predispose to intracranial hemorrhage and RH.[4,5,10,114,115,133,140–143,157] The bleeding or clotting disorder may be primary or

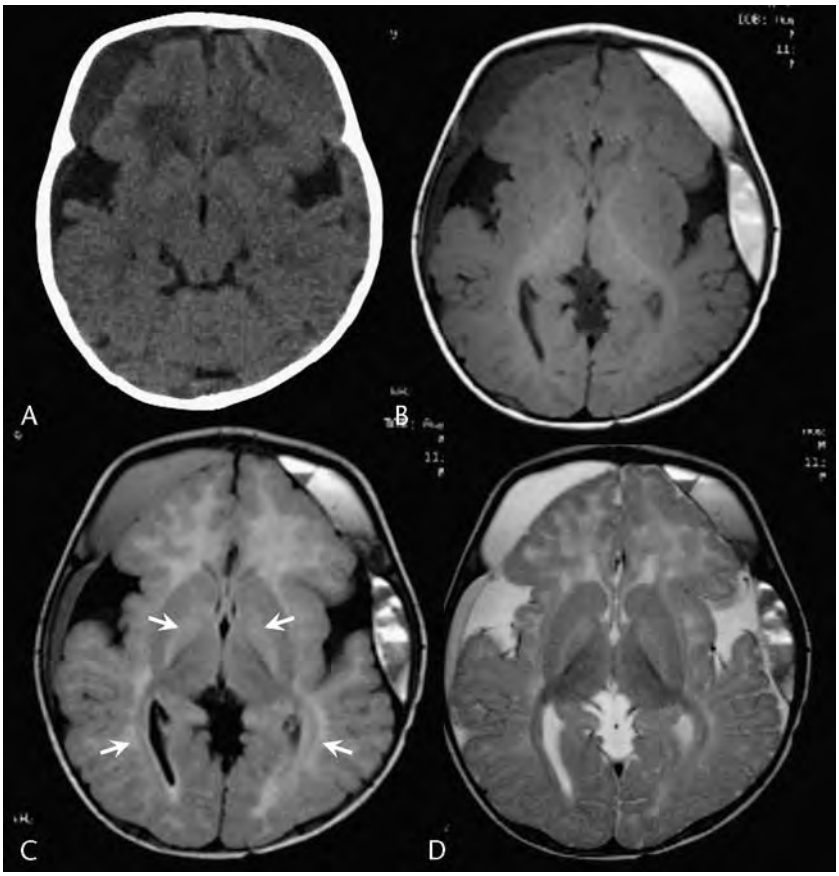

FIGURE 19. Images obtained from a 9-month-old male infant with glutaric aciduria type 1, SDHs, and RHs. CT (A), T1 (B), FLAIR (C), and T2 (D) MRI images show bilateral mixed-density and mixed-intensity extracerebral collections with fluid levels and septations, especially on the left side. Other characteristic findings for glutaric aciduria type 1 include bilaterally wide sylvian fissures (arachnoid cysts) plus abnormal basal ganglia (globus pallidus) and cerebral white matter intensities (arrows).

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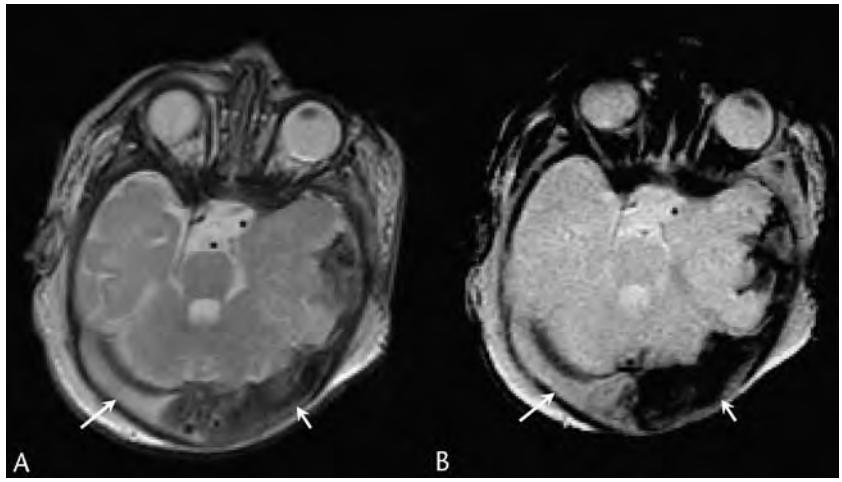

FIGURE 20. Images obtained from a 1-week-old male neonate with seizures, thrombocytopenia, antithrombin III deficiency, and ECMO for pulmonary hypertension. Axial T2 FSE (A) and GRE (B) MRI images show bilateral, mixed-intensity SDHs (arrows).

secondary (Figs. 7, 14–16, 18, 20, 21). In some cases, a more extensive workup beyond the usual *screening* tests will be needed, including a hematology consultation. Included in this category are the anemias, hemoglobinopathies (eg, sickle cell disease), hemorrhagic disease of the newborn (vitamin K deficiency Fig. 21), hemophilia A and B, factor V and XII deficiencies, von Willebrand disease, idiopathic thrombocytopenic purpura, disseminated intravascular coagulation and consumption coagulopathy associated with other conditions (eg, trauma, infection), liver disease, nephrotic syndrome, hemophagocytic lymphohistiocytosis, anticoagulant therapy, and others. Venous thrombosis may involve the dural venous sinuses (ie, dural venous sinus thrombosis [DVST]) and/or the cerebral veins (ie, cerebral vein thrombosis [CVT]) and be associated with primary or secondary hematologic or coagulopathic state.[10,123,124,133,158–161] Risk factors include acute systemic illness, dehydration (fluid-electrolyte imbalance), sepsis, perinatal complications, chronic systemic disease, cardiac disease, connective tissue disorder, hematologic disorder, oncological disease and therapy, head and neck infection, and hypercoagulable states. Seizure and/or neurological deficit are common, and hemorrhagic infarction is characteristic. Subarachnoid hemorrhage, SDH, or RH may also be observed, especially in infants (Figs. 15, 16, 18, 22). Relative high densities anywhere along the dural venous sinuses, tentorium, and falx (interhemispheric fissure and inferior sagittal sinus) may be seen on initial CT. Linear high densities may also be present along the distribution of the cortical ("cord sign"), subependymal, or medullary veins and give the impression of SAH, SDH, or intracerebral

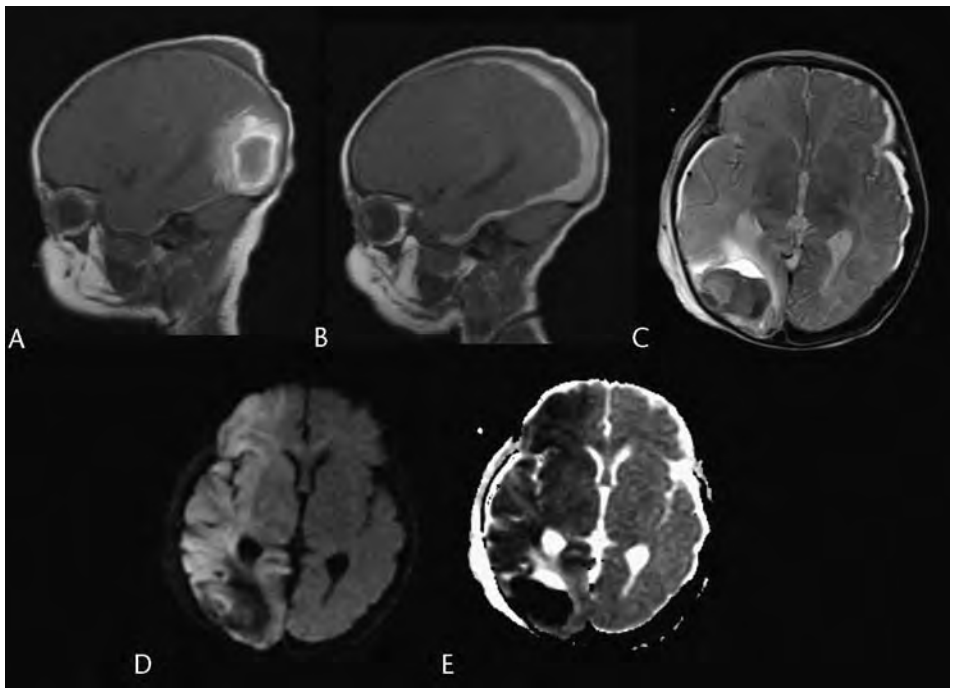

FIGURE 21. Images obtained from a 1-week-old male neonate who had seizures after delivery at home (no vitamin K administered). After surgical evacuation of large, right-side SDH, sagittal T1 (A, B), axial T2 (C), ADC (D), and DWI (E) images show bilateral mixed-intensity extracerebral and intracerebral hemorrhages and right-side cerebral hemispheric restricted diffusion (likely infarction) (diagnosis, hemorrhagic disease of the newborn [vitamin K deficiency]).

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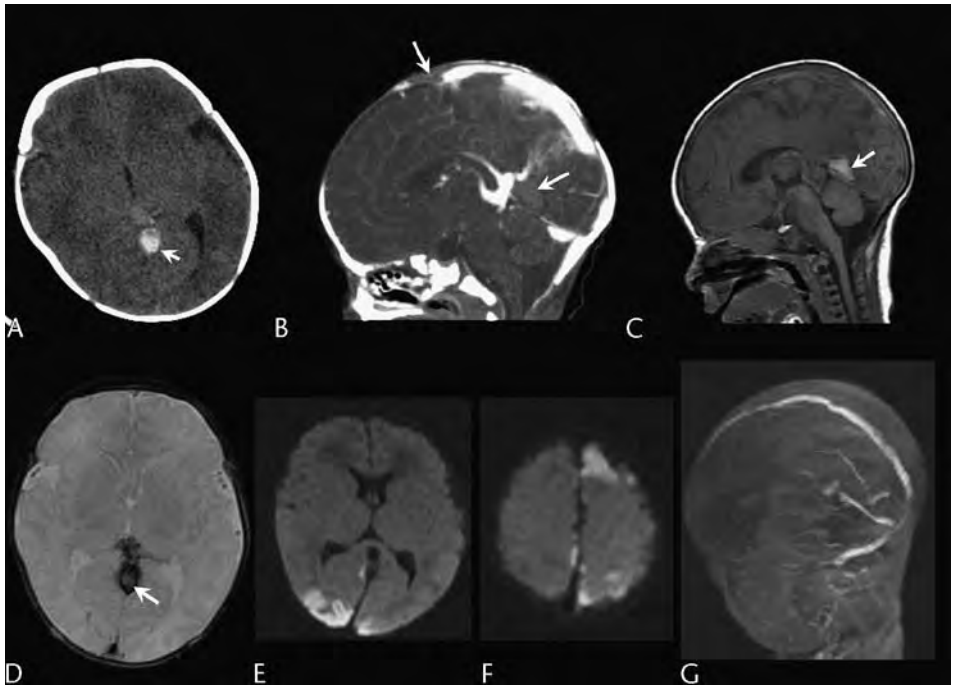

FIGURE 22. Images obtained from a 2-week-old male neonate with lethargy in emergency room (ER). Computed tomographic image (A) shows a focal midline hyperdensity at the level of the straight sinus (arrowhead). Sagittal CTV image (B) shows luminal masses along the straight and superior sagittal sinuses (arrows). Sagittal T1 (C) and axial GRE (D) images show the thrombus within the straight sinus (arrows). Axial DWI images (E–F) show restricted diffusion in multiple cortical areas (likely infarction vs suppuration). Magnetic resonance venography (G) is of poor diagnostic quality as compared with CTV (diagnosis, group B streptococcal meningitis with DVST).

hemorrhage. The "empty delta" sign may be seen within the superior sagittal sinus on contrast-enhanced CT. There may be multifocal infarctions (hemorrhagic or nonhemorrhagic) or intraventricular hemorrhage. With extensive dural venous sinus or cerebral venous thrombosis, there may be massive, focal, or diffuse edema. Orbit, paranasal sinus, or otomastoid disease may be associated with basal venous sinus thrombosis (eg, cavernous, petrosal, sphenoparietal). The thromboses and associated hemorrhages have variable MRI appearance depending on their age (see Image Analysis–Magnetic Resonance Imaging section and Table 1). Computed tomographic venography or MRV may readily detect DVST but not cerebral vein thrombosis, which may be suspected because of the characteristic distribution of hemorrhage or thromboses along venous structures, as demonstrated on susceptibility-weighted sequences (eg, GRE hypointensity). Depending on the clinical context, treatment may be directed only to the specific cause (eg, infection) or may also include anticoagulation or thrombolysis.

Infectious and Postinfectious Conditions

Meningitis, encephalitis, or sepsis (eg, bacterial, viral, granulomatous, parasitic) may involve vascular structures resulting in vasculitis, arterial or venous thrombosis, mycotic aneurysm, infarction, and hemorrhage (Figs. 3, 14–17, 22, 23). Subdural hemorrhage and RH may also be observed.

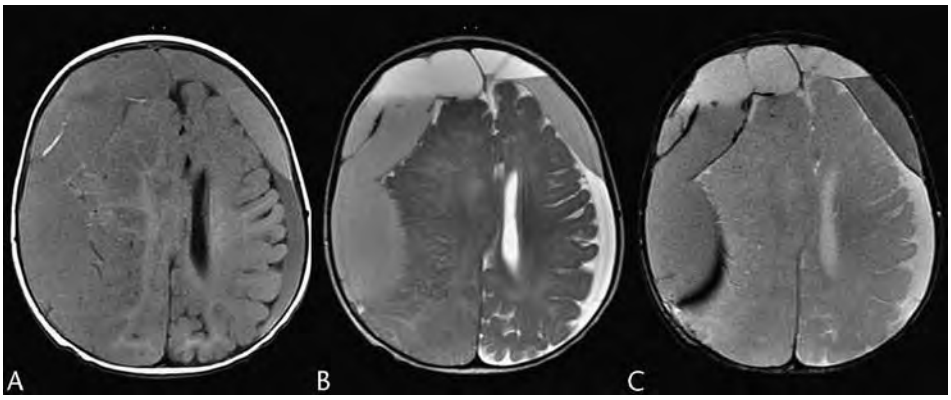

FIGURE 23. Images obtained from a 5-month-old male infant who had macrocephaly and seizures after having group D streptococcal (nonenterococcal) meningitis at the age of 3 days. Axial T1 (A), T2 (B), and GRE (C) images show bilaterally large and mixed-intensity extracerebral collections with septations and asymmetrical mass effect (likely chronic subdural effusions or hygromas with rehemorrhage).

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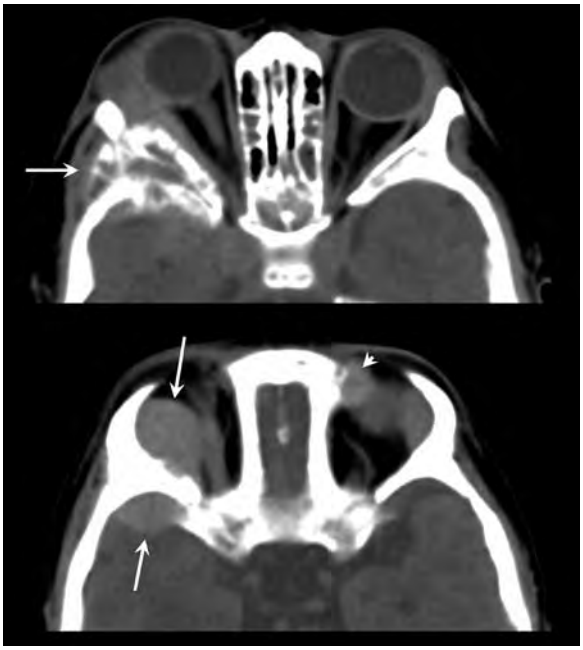

FIGURE 24. Images obtained from an 18-month-old girl with periorbital and facial ecchymoses in ER, evaluated for NAI. Computed tomographic image shows bilateral iso-high-density orbital soft tissue masses with bone destruction (arrows) and extension into the right-side middle cranial fossa (diagnosis, neuroblastoma).

Postinfectious illnesses (eg, postvaccinial) may also be associated with these findings.[139] Included in this category are the *encephalopathies of infancy and childhood* and *hemorrhagic shock and encephalopathy syndrome*.[115,133]

Autoimmune and Vasculitic Conditions

These include Kawasaki disease, systemic lupus erythematosis, moyamoya disease, Wegener granulomatosis, and Behçet syndrome.[115,133]

Oncological Disease

Hematologic malignancies, solid tumors of childhood, and their attendant therapies (including transplantation) are commonly associated with a variety of sequelae or complications that predispose to hemorrhage (eg, SDH and RH).[115,133] This includes vascular invasion by tumor, immunocompromise, infection, and coagulopathy. The clinical presentation and image findings may be mistaken for NAI (eg, leukemia, neuroblastoma) (Fig. 24).

Toxins, Poisons, and Nutritional Deficiencies

This category includes lead poisoning, cocaine, anticoagulants, and vitamin deficiencies (eg, vitamins K, C, D) (Figs. 21, 25). Preterm neonates and other chronically ill infants are particularly vulnerable to nutritional deficiencies and complications of prolonged immobilization that often primarily affect bone development. Such infants may have skeletal imaging findings (eg, multiple healing fractures) that are misinterpreted as NAI, particularly if they present with AI that is complicated by SDH and RH (Fig. 25).[162–174]

Medical and Surgical Complications

This category includes (1) anticoagulant therapy or treatment-induced coagulopathy and (2) morbidity from medical or surgical interventions.[115,133]

CONCLUSIONS

In view of the currently available data, it is clear that we do not have an established EBM platform from which to

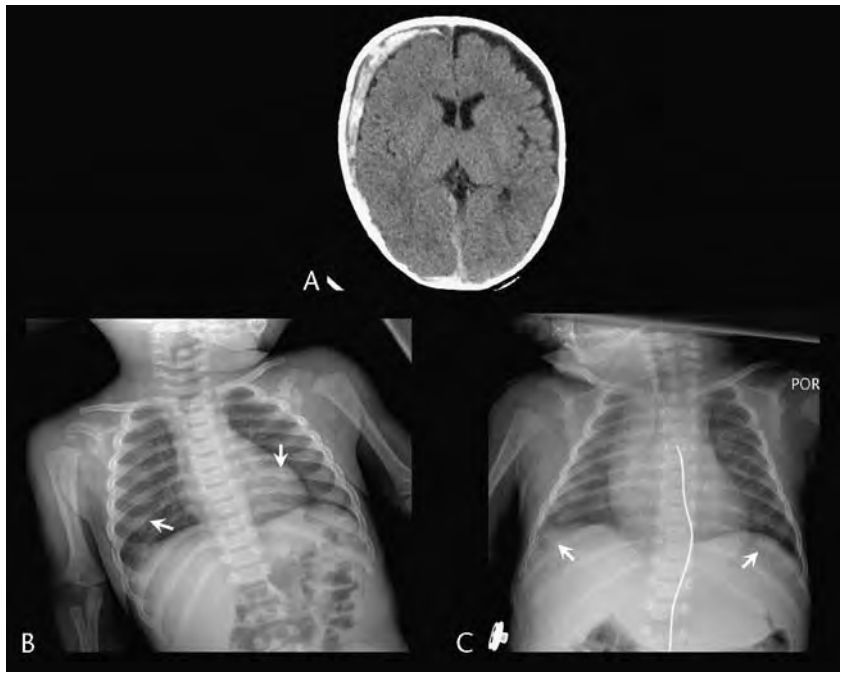

FIGURE 25. Images obtained from a 7-month-old male infant (25-week preterm birth) dropped with head impact to floor, RHs, evaluated in ER. Computed tomographic image (A) shows right-side mixed high-density extracerebral collection, left-side low-density extracerebral collection, posterior interhemispheric high-density hemorrhage, and right-side cerebral low-density edema. Chest radiograph in ER (B) shows bilateral anterior and posterior old, healing rib fractures. Comparison with earlier chest radiograph (C) at discharge from neonatal intensive care unit shows diffuse osteopenia and anterior rib flaring (arrows). Diagnosis: rickets of prematurity vs NAI?; AI with acute SDH superimposed on BECC vs NAI?

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distinguish NAI from AI and, in some cases, traumatic from nontraumatic CNS injury. More reliable research is needed to establish a sound scientific foundation for CNS injury in NAI. The young infant is assumed more vulnerable to traumatic CNS injury, whether accidental or not, as compared with the older child or adult, and relies on the attention of caretakers for safety. However, as the infant becomes more mobile (rolling, crawling, walking, etc), the risk of AI (eg, from falls) increases. Furthermore, the interaction with older siblings or other children becomes a factor. The medical and imaging findings cannot diagnose intentional injury. Only the child protection investigation may provide the basis for inflicted injury in the context of supportive medical, imaging, or pathological findings. Furthermore, biomechanical factors must be taken into consideration regarding the mechanism of trauma.

The radiologist should describe the imaging findings in detail, including the pattern, distribution, and severity of injury. A DDX is given, and timing ranges are provided if possible. If NAI is at issue, then the radiologist must directly communicate the imaging findings to the primary care team and be available to consult with child protection services and other medical or surgical consultants, including the pathologist or biomechanical specialist, law enforcement investigators, and attorneys for all parties, as appropriate.[1–5] The pattern of injury and the timing parameters, as may be provided by MRI, are particularly important with regard to correlation of events as reported by witnesses and potential suspects. The radiologist must also be aware of certain conditions that are known to have clinical and imaging features that may mimic abuse.[1–5] These should be properly ruled out, and the possibility of combined or multifactorial mechanisms with synergistic effects should also be considered (eg, predisposing condition plus trauma). A timely and thorough multidisciplinary evaluation may be the difference between an appropriate child protection and an improper breakup of the family or a wrongful indictment and conviction.

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Abraham Jacobi Award Address

On the Theory and Practice of Shaking Infants

Its Potential Residual Effects of Permanent Brain Damage and Mental Retardation

John Caffey, MD, Pittsburgh

In the first modern discussion in 1946 of the parent-infant stress syndrome (PITS), or battered baby syndrome, I described six infants, 13 months or younger, who suffered from the combination of subdural hematomas and characteristic bone lesions.[1] During the last 25 years[2-5] substantial evidence, both manifest and circumstantial, has gradually accumulated which suggests that the whiplash-shaking and jerking of abused infants are common causes of the skeletal as well as the cerebrovascular lesions; the latter is the most serious acute complication and by far the most common cause of early death.[6]

Today we invite your attention to the evidence which supports our concept that the whiplash-shaking and jerking of infants are frequently pathogenic and often result in grave permanent damage to infantile brains and eyes. We shall also point out that potentially pathogenic whiplash-shaking is practiced commonly in a wide variety of ways, under a wide variety of circumstances, by a wide variety of persons, for a wide variety of reasons. The most common motive for repeated whiplash-shaking of infants and young children is to correct minor misbehavior. Such shakings are generally considered innocuous by both parents and physicians. If our concept of the pathogenic significance of whiplash-shaking is valid, it follows that the prevention of such shaking and jerking might substantially reduce the incidence of brain damage and mental retardation. The line of demarcation between pathogenic and nonpathogenic shaking is often vague.

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The nature and distribution of the bone lesions in the PITS must be interpreted from the radiographic changes exclusively because they have not been studied systematically at either surgical exploration or necropsy. The metaphyseal avulsions are the most common of these lesions. Some are small fragments of cortical bone torn off the external edge of the cortical wall at the metaphyseal levels where the periosteum is most tightly bound down to the cortex. In most cases, however, they appear to be small chunks of calcified cartilage which have been broken off the edges of the provisional zones of calcification at or near the sites of the attachments of the articular capsules (Fig 1 to 3). Often bones on both the proximal and distal sides of a single joint are affected, especially at the knee. All of these metaphyseal avulsion fragments appear to result from *indirect, traction, stretching, and shearing, acceleration-deceleration stresses on the periosteum and articular capsules*, rather than *direct, impact stresses such as smashing blows on the bone itself.*

Traumatic involucra (Fig 4 to 6) commonly accompany the metaphyseal avulsions and involve the same terminal segment of the same shaft.

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Fig 1.—Unexplained metaphyseal fragments (arrows) of the provisional zone of calcification (pzc) on the proximal side of the knee in the femur and the distal side of the knee in the tibia of a patient 4 months of age whose parents denied trauma.

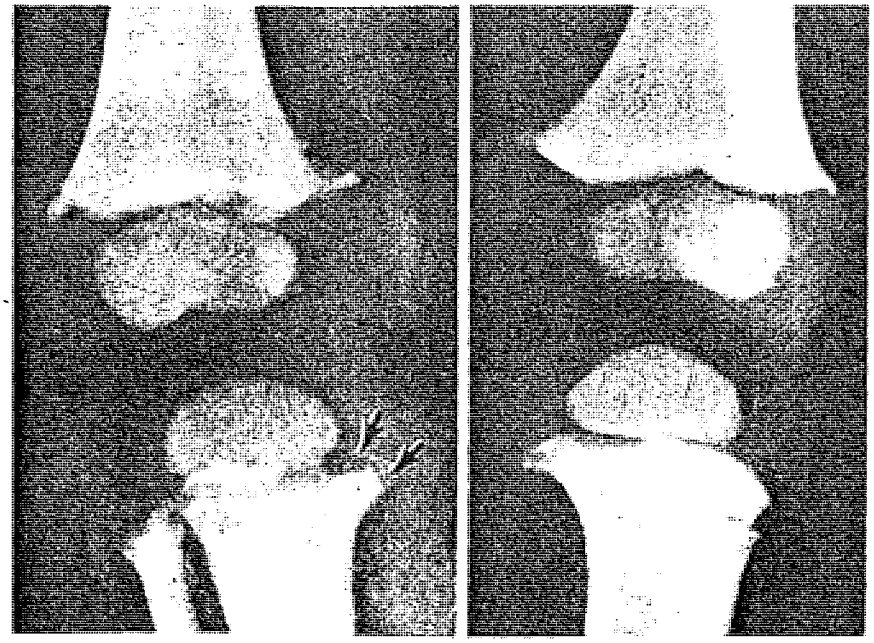
They are the largest and most conspicuous of all traumatic lesions in growing bones. Usually they are not fracture-dependent. They develop due to traction-rupture of the abundant normal perforating blood vessels, which course through the cortical wall between the periosteum and the medullary cavity and which are severed at the junction of the internal edge of the periosteum with the external edge of the cortex. The accumulation of blood internal to the periosteum, but external to the cortical wall, lifts the periosteum off the wall for variable distances and forms subperiosteal hematomas of variable sizes and shapes. Often these are symmetrical in analogous bones in the two arms or legs, or they affect bones in the arms and forearms only, or sometimes in the thighs and shanks only. Frequently they involve bones on both the distal and proximal sides of a single joint, especially the knees. At first they appear radiographically as masses of water density superimposed on the shaft, but after four to ten days a thin opaque shell of new fibrous bone begins to form around the external edge of the hematoma. The entrapped subperiosteal blood is then gradually resorbed. The nature and distribution of these lesions can be best explained on the basis of *indirect, primary, acceleration-deceleration traction drags* on the periosteum, its vessels, and the joint capsules from manual seizing and gripping the extremities and whiplash-shaking the head. Neither their nature nor their distribution can be satisfactorily explained on the basis of *direct impact stresses (blows) on the bone itself.*

Several observers have noted associated diffuse sclerosis of the shafts of some of the affected bones of some abused infants. This has suggested excessively fragile, brittle, chalk-like bones to some radiologists. In recent biopsies, however, the microscopic examination disclosed the lamellae to be laid down in an irregular woven pattern. This, in my opinion, indicates that the sclerosis is caused by excessive newly formed primitive fibrous or woven bone, which forms regularly under the periosteum following traumatic subperiosteal edema or bleeding or both. Traumatic thickenings of the external subperiosteal edge of the cortical wall are the cause of the sclerosis. The epiphyseal ossification centers and round bones are not sclerotic. These sclerotic shafts are probably stronger than normal shafts.

Traumatic metaphyseal cupping is due to traumatic obstructive injury to the epiphyseal arterioles in the neighboring cartilage plate[1]; and the metaphyseal "loop" deformities[2] are due to stretching and extension of the traumatic involucra terminally. Both of these lesions are best explained on the causal basis of the grabbing, squeezing the extremities by the assailant's hands, and whiplash-shaking the infant's head.

Despite the failure of parents and physicians to appreciate the grave significance of whiplash-shaking younger infants, and to record shaking in medical histories, there are

Fig 2.—Symmetrical metaphyseal "angle" fragments in the edges of the pzc of both femurs with fragments in the medial edges of the tibial pzc. A faint "loop" deformity, a fine opaque curved line (arrows), is superimposed on the epiphysis of the right tibia.



several convincing recorded examples of pathogenic and even fatal shaking.

EXAMPLE 1 (15 CASES).—The most gruesome and, at the same time, the most significant examples of proved pathogenic whiplash-shakings and of burpings are recounted in the story of an infant-nurse (*Newsweek* 48(pt 1):90, 1956). She is reported to have killed three infants and maimed 12 others during a period of eight years, largely by shaking and jolting infantile brains and their blood vessels. These assaults all occurred in the homes of upper-middle-class, well-educated, well-to-do parents, who provided expert medical care by highly trained pediatricians. Her services were said to have been much in demand because she had built up a reputation for being "extravagantly kind" to her infant charges. Both parents and doctors apparently had full confidence in her; in one instance she was invited back by the parents to care for their second infant after she had shaken their first child to death. After one infant was found dead without satisfactory explanation, the nurse explained that she had only tried to get the bubble up. "I picked him up and tried to get the bubble up. I didn't do anything wrong." After another of her patients had succumbed unexpectedly, she admitted that she "had given him a good shaking." Necropsy disclosed traumatic brain injury. Eventually she admitted killing three infants and maiming two others. When these admissions became known to the community, parents disclosed that ten additional infants had been significantly injured by this nurse.

The primary assaulting force in all of the nurse's attacks on infants appears to have been violent whiplash-shaking and excessive whiplash–pounding on the back during burping. One is amazed that she was able to continue her murderous assaults in this much better-than-average social and medical milieu for eight years. It is likely that she did not realize the full power and violence generated in her shaking of small infants. She weighed 105.32 kg (233 lb) and was said to have had very large hands. The last infant she shook to death weighed 4.97 kg (11 lb). Had the nurse been shaken by a caretaker of corresponding weight and strength, the monster would have weighed 21 times her weight or about 2,226 kg (5,000 lb) and would have had the strength of 21 strong women.

EXAMPLE 2 (FOUR CASES).—Weston[5] and Steele and Pollock[7] report four cases of pathogenic shaking in battered babies. (1) A girl, 2½ years old, was shaken violently because she whined. (2) A girl 4 years of age was shaken violently because of incessant crying. (3) A girl 4 months of age was shaken fatally, during which her head was banged against the crib. (4) A girl, 2 years old, was shaken and beaten to death with a stick.

EXAMPLE 3 (THREE CASES[8]).—A boy of 6 months had unexplained convulsions and fever. His mother was too shocked to speak. A subdural hematoma was excised and he died three days later. Eventually the mother admitted that she had shaken him several times to save him from "choking to death" during a violent paroxysm of coughing. She had attempted to "clear his throat" and he went into convulsions following the shaking. (2) A boy, 6 months old, began to vomit and convulse. There were no external signs of head injury but retinal hemorrhages were seen in both ocular fundi. Subdural hematomas were treated surgically. (3) A few days later, this patient's twin brother came to the hospital with an unexplained broken femur. Shortly thereafter, the first twin returned with persistent subdural hematoma. By this time, bruises had appeared on both his forearms, which fitted the pads in the fingers and thumbs of the examiner's hands, where the boy's forearms had been gripped by his assailant during the shaking. Eventually, the mother admitted that she and her husband "might have shaken him when he cried at night." Guthkelch opines that a "good shaking" is felt by British parents to be more socially acceptable, and physically less dangerous, than blows or punches to the head.

EXAMPLE 4 (ONE CASE).—Kempe et al[9] described radiographic bone lesions caused by the repeated parental shaking of a prematurely born twin girl of 7 months, who had been abused by the mother since the second month of life. She was shaken while gripped by the legs and held inverted. Multiple massive involucra and several metaphyseal avulsions developed in her femurs and tibias.

EXAMPLE 5 (ONE CASE).—Swischuk[10] described compression fractures of vertebral bodies in one infant, which were believed to be due to whiplash-shaking.

EXAMPLE 6 (THREE CASES).—We have found two examples of protective jerking and one of repeated sibling whiplash-shaking. We have encountered two patients in whom a single, sudden, violent jerk of one extremity apparently produced metaphyseal avulsions and traumatic involucra. (1) A boy, 4½ months old, had always been well until his mother grabbed him by one forearm and yanked him upward to prevent his falling to the floor, off a bassinet. Six weeks later, massive involucra of the radius and ulna in the seized forearm were demonstrated radiographically. (2) A girl of 3 weeks was said to have suddenly developed unexplained swelling of the knee and fever. Twelve hours later, a pediatrician diagnosed osteomyelitis of the tibia because of point tenderness at the knee and slight fever and leukocytosis. There were no bruises. Treatment with penicillin was begun. Radiographs showed avulsion fracture fragments at the level of one proximal tibial metaphysis which indicated trauma. Later films showed the evolution of a long traumatic involucrum on this tibia, when the fever was subsiding. On the 19th day, the mother admitted that, just prior to the appearance of the swelling of the knee, she had grabbed the baby by the leg and jerked her upward to protect her from falling onto a hard wood floor. At the same time she had fallen forward with her and onto her. The pediatrician was still reluctant to accept the primary diagnosis of a trauma because he was unfamiliar with the frequency of fever and leukocytosis after traumatic internal hemorrhage.[11] (3) This boy of 8 months had had

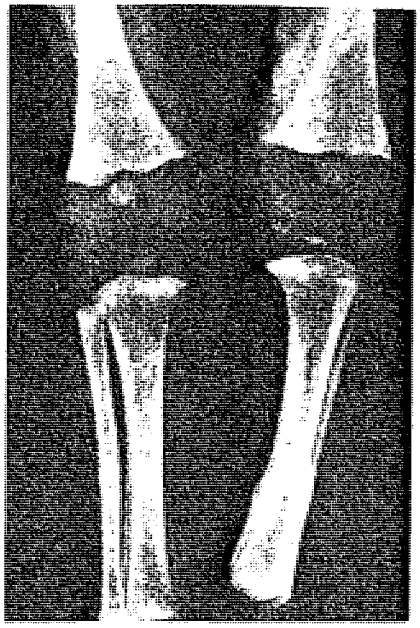


Fig 3.—Unexplained traumatic fracture fragments at both ends of pzc (angle fractures) of the left femur with fine, thin, external thickenings of its cortex which represent early traumatic involucrum. Two thicker, older, traumatic involucra have formed in the distal thirds of the two tibial shafts; these lesions indicate that the infant (6 months) had been seized by both ankles.

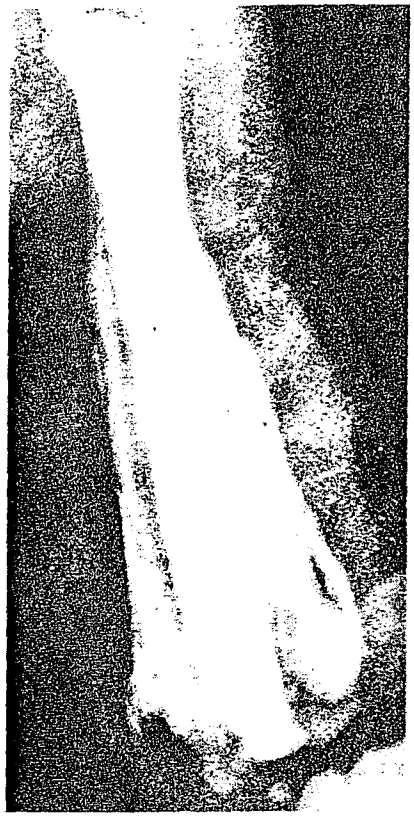


Fig 4.—Unexplained massive traumatic involucra of the left humerus of an infant 13 months of age whose parents denied traumatic abuse. The external edges of the new cortical shell are uneven and there are large and small blackish patches which indicate uneven resorption of blood or repeated traumatic episodes with recurrent subperiosteal bleeding.

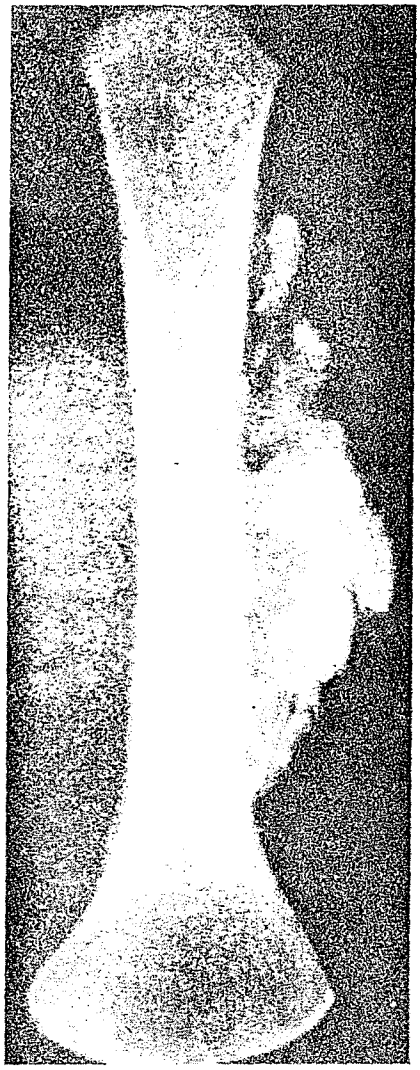


Fig 5.—Unexplained large centrally placed traumatic involucra on the anterolateral aspects of the left humerus of a boy 13 months of age whose parents denied traumatic abuse. This arm had been wrapped in a bandage which bound him in restraint during the performance of a cerebral angiogram, eight weeks earlier.

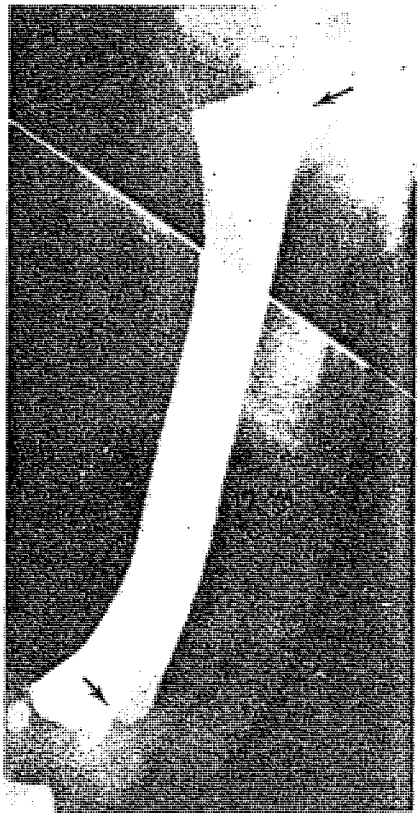


Fig 6.—Metaphyseal avulsion at the proximal and distal ends of the humeral shaft of a boy 12 months old. The distal third of the shaft is thickened and sclerotic due to an old and fused traumatic involucrum. The parents denied physical injury.

unexplained swellings and tenderness and limitation of motion in both legs for several weeks when radiographs of the skeleton disclosed traumatic involucra at the proximal ends of both femurs. The parents rejected the diagnosis of trauma because, other than themselves, no one had been alone with the infant except his 8-year-old brother. He, however, had frequently acted as a lone babysitter when the parents went out socially. They found, at the first trial, by secretly watching their son as lone babysitter, that soon after their departure he seized the infant by the legs and shook him violently, and swung him, and flung him onto a bed.

EXAMPLE 7 (TWO CASES).—These cases involve oculovascular lesions. Gilkes[12] mentions the case of Wallis in which subdural hematoma and retinal hemorrhages resulted from seizing an infant by the legs and swinging him in a circle about the parent's head. He also refers to Breinin's infant patient who developed retinal lesions after a parent had gripped him by the thorax and shaken him violently.

These 27 examples of recorded pathogenic shaking represents only an infinitesimal portion of the uncounted thousands of moderate unadmitted undetected and unrecorded whiplash-shakings which probably occur every day in the United States. The actual daily incidence of pathogenic shakings in the United States is unknown, but it is undoubtedly substantial. The admitted pathogenic whiplash-shakings by the infant-

nurse, cited above, have a special relevance because they demonstrate conclusively that even repeated murderous whiplash-shakings can be concealed for years without arousing the suspicions of educated parents and well-trained pediatricians, in an optimal medical and socioeconomic environment. This long concealment was due in large part to the fact that shakings, which caused fatal brain damage and intracranial bleedings in the nurse's patients, did not cause suggestive signs of head injury such as bruising of the scalp and face, or cephalhematoma, or bulging of the anterior fontanel, or fractures of the calvarium.

The whiplash-shaking of infants and younger children are precarious, pervasive, prevalent, and pernicious practices which can be observed wherever parents, parent-substitutes, infants, and small children congregate: in the home, on the street, in buses, nurseries, kindergartens, day-care centers, orphanages, "preschool" schools, in parks, playgrounds, shopping centers, and even in the waiting rooms of pediatric clinics. Many well-intentioned, responsible parents, who think nothing of giving a small child a "good shaking" or a series of such shakings, would not dream of giving their child a series of "good" blows or punches on the head. Yet, the cumulative pathogenic effects of repeated mild or moderate whiplash-shakings of the head, though inapparent clinically, may be far more grave than single, even if heavy, blows or punches to the head. The history of trauma and the nature of the traumatic force used cannot usually be elicited, sometimes owing to traumatic amnesia of the perpetrators who may not remember the traumatic episode or the kind and amount of violence inflicted on the infant. Steele and Pollock call this kind of amnesia "more or less unconscious defensive forgetting."[7]

The grabbing and gripping of an infant or younger child by the extremities or by one leg or arm and then shaking him seem to be instinctive, almost reflex, violent actions by angry adults in the commission of wilful assault or for ordinary discipline of minor misbehavior. Many infants are whipped, beaten, and spanked, as well as shaken. The frequency of whiplash-shaking varies inversely with the age of the infant; apparently, many infants are shaken and jerked, but few are spanked prior to the seventh month of life. Overvigorous pathogenic shaking may be resorted to unwittingly, by a frightened parent or nurse, to dislodge a suspected foreign body from the mouth or pharynx, or to stop violent coughing, or protracted crying and whining. Even overvigorous "burping" may be fatal, as demonstrated in the story of the shake-prone infant-nurse. All curative and prophylactic medical procedures which require repeated whiplash-shaking and jerking of the head are potentially pathogenic to the brain and should be banned or used with proper caution. Artificial respiration may induce excessively high intracranial and intraocular venous pressures which lead to hemorrhagic brain and eye damage and pneumomediastinum.

There are several apparently innocent, accepted, habitual practices, other than intentional shaking and jerking, which whiplash the head and brain, and which could lead to permanent brain damage. The infantile head is subjected to some of these stresses during such playful practices as repeated vigorous "tossing the baby into the air"; "riding the horse," in which the infant faces the parent while sitting on his pendulant shin, which is swung ventrodorsally; "cracking the whip," or gripping the infant by his ankles and swinging him in a circle around the parent's head; or spinning him on his own longitudinal axis; and "skinning the cat," in which the younger child is suddenly somersaulted forward, after being gripped by the wrists which have been inserted backward between his thighs. Infants and younger children are often handled too roughly in play by older siblings as well as parents, in such procedures as "shaking or spinning him dizzy." It is obvious that younger, more supple, calvaria of smaller children, of 4 to 8 years, although less pliable than the calvaria of the first weeks and months, were not designed for the habitual jolting of repeatedly diving headfirst into water, waterskiing, protracted gymnastic tumbling, and the inevitable brain-jolting of such adversary sports as boxing, wrestling, football, and basketball when indulged in repeatedly for several years. In some of these, the training sessions of several hours each week are more hazardous than the games and performances themselves.

Many of the toys and recreational contraptions which generate whiplash-shakes and jolts to the head should be more carefully assayed for their pathogenicity, and banned from infantile and early juvenile use if it can be shown that their cumulative effects over protracted periods are pathogenic. These items should include baby bouncers and infant jumpers and, for younger children, the swings, seesaws, and playslides in amusement parks, the powered vibratory training and practice equipment in gymnasia; powered cradles and powered rocking horses; trampolines; skateboards, and sled jumping. The same studious consideration should be given to jolting transport-vehicles which carry infants and small children—bicycles and such powered vehicles as motorcycles and motor cars driven rapidly and habitually over rough roads. The recurrent exposure to the snowmobile probably offers the greatest hazard to infantile brains and hearing. The pathogenic, brain-jolting, whiplash potentials for infants and younger children who are carried habitually in speedboats over rough water and in small airplanes in rough weather are manifest. Noise and other vibratory stimuli may be peculiarly pathogenic to infants when they are subjected to them continuously 24 hours a day at home. Mental and emotional disorders are said to be more prevalent around airports with high noise and air-vibration levels. It is possible that the noises and air-vibrations induced by radio, television,

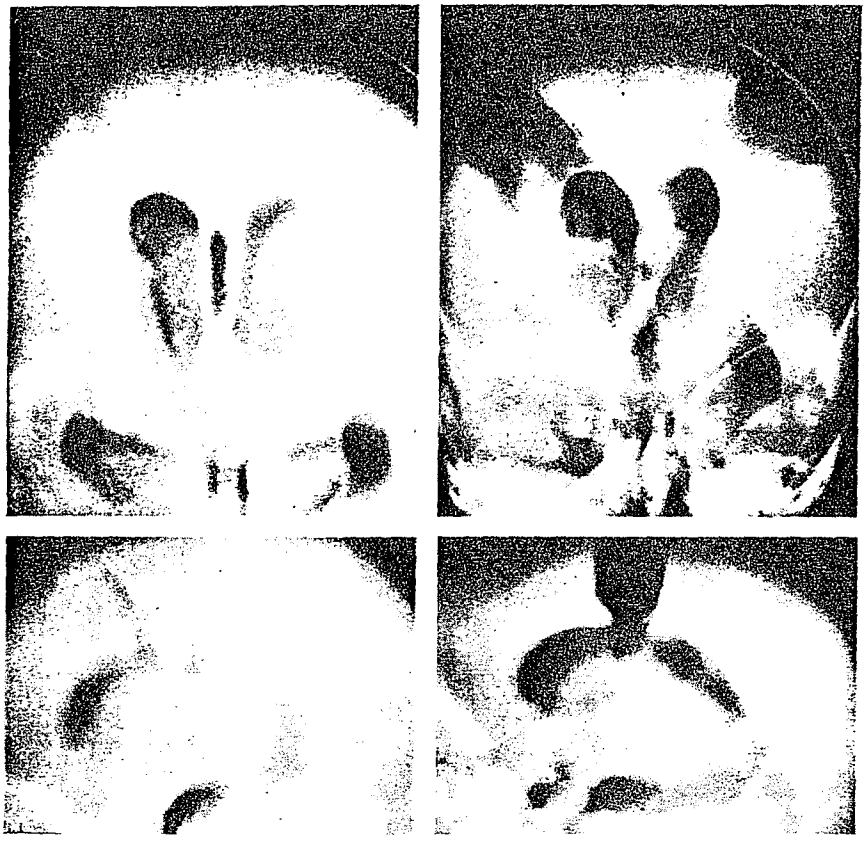


Fig 7.—Large bilateral porencephalic cavities. Top left and top right, Frontal projections at the seventh week. Bottom left and bottom right, Lateral projections two weeks later at the sites of needle tracks in the cerebral hemispheres of an infant who had suffered from meningitis at 7 weeks of age and who was subjected to "needling" of the brain to get the causal organism for optimal treatment.

amplified hi-fi sets, air-conditioners, telephones, vacuum cleaners, blenders, and garbage disposal and dishwashing units may interfere with the basic rest needs of the infant in his own home when he is overexposed to them continuously for long periods. In small thin-walled apartments, excessive exposure of an infant to pathogenically high-level noises is practically guaranteed.

Some of the more violent forms of endogenous trauma, such as the repeated convulsions in tetanus, epilepsy, or of meningoencephalitis caused by viral infections or by lead poisoning, may induce traumatic whiplash-brain damage and mental retardation. The grave, frequently fatal encephalopathy of infantile pertussis results, in part, from increased intracranial venous pressures caused by severe paroxysms of coughing, which in turn leads to focal cerebral hemorrhages and residual cerebral damage. Rhythmic whiplash habits of the infant himself during the first months of life, such as *head-rolling*, *body-rocking*, and *head-banging* may be traumatically pathogenic to his brain and its veins. Protracted, repeated severe *breath-holding spells* may be similarly damaging to the brain.

Theoretically, the heads of infants who suffer disorders characterized by immaturity of their calvaria such as osteogenesis imperfecta, familial hyperphosphatasemia, hypophosphatasia, lacunar skull, and cleidocranial dysostosis should all be specially vulnerable to whiplash shaking. This is also true for the infantile disorders associated with weakness of the cervical muscles.

Subdural hematomas are practically always traumatic in origin. They are found commonly in infants younger than 24 months with a peak incidence during the sixth month. This high vulnerability of the neonate and the younger infant to traumatic intracranial bleeding is due to the combination of heavy head and weak neck muscles, which renders his brain especially susceptible to whiplash stresses. Also his thin, partly membranous calvarium is supple and permits easy stretching of the brain and its veins by the postnatal indirect whiplash-traction stresses of shaking and jerking. The softness of his immature unmyelinated brain adds to its vulnerability. The actual time of onset of the subdural hematoma is usually uncertain during the first weeks and months of life. Much postnatal trauma has long been depreciated because it has been undetected and ascribed to birth injury. Premature infants are very vulnerable, and the vulnerability of full-term infants varies inversely with age. Male infants are twice as vulnerable as females; this is probably due to the relative immaturity of male heads and brains. The hydrocephalic premature infant is maximally vulnerable.

Infantile subdural hematoma frequently remains undiagnosed owing to the customary lack of distinctive diagnostic signs and symptoms. Ingraham and Matson[13] obtained a history of birth injury in only 26% of their 319 patients, and postnatal injury in only 20%. In 54%, the source and nature of the trauma was never determined. Fractures of the skull were identified in only 9%. Regional bruises of the scalp and face were not sufficiently frequent to be included in their tables on clinical findings. The clinical picture was not characteristic; it was made up mainly of fever, convulsions, vomiting, and hyperirritability. These signs are all common to many ordinary infantile disorders. It is probable that practically all of the small cumulative subdural hematomas which result from habitual shaking remain undiagnosed permanently, and go on to become chron-

c subdural hematomas.

Ingraham and Matson[13] state that their mentally retarded and deficient patients, for the most part, were those who suffered from large chronic hematomas associated with marked atrophy of the cerebral hemispheres. The incidence of mental retardation in their pretreated and nontreated patients is not reported. They did find, however, that incomplete removal of the marginal membranes of the hematoma restricted the normal growth of the underlying cerebral cortex and impaired mental growth. This statement implies that the persistence of the membranes in all undetected, untreated patients would impair mental growth in a far greater number in cases of undiagnosed, untreated chronic subdural hematoma. Ford[14] found cerebral damage and mental deficiency and motor deficits in a large majority of the children who survive after aspiration treatment alone. One can reasonably conclude that whiplash-shaking and jerkings, which are the prime causes of subdural hematoma in diagnosed cases, are also substantial causes of later mental retardation in the countless cases of unrecognized chronic subdural hematoma, in far greater numbers. The frequent bilaterality of subdural hematomas also supports whiplash-shaking as the probable cause.

The retinal lesions caused by shaking will undoubtedly become valuable signs in the diagnosis of subclinical inapparent chronic subdural hematoma, and also become a productive screening test for the prevalence of whiplash-dependent mental retardation and other types of so-called idiopathic brain damage. Kiffney[15] found bilateral retinal detachments in a battered girl of 7 months. Maroteaux and associates[16] found permanent, stable retinal lesions in the peripheries of the ocular fundi of previously battered children. In five battered children, Friendly[17] found retinal hemorrhages associated with intracranial bleedings.

It should be borne in mind that the "needling" of the brain to get diagnostic, subdural, subarachnoid, or intraventricular fluid may in itself be a dangerous procedure[18] and may lead to extensive porencephaly and brain destruction (Fig 7). Smith and Crothers[19] have shown that the injection of air into the lumbar subarachnoid space may cause intracranial bleeding and intracranial subdural hematomas. Overvigorous passive movements of the infantile head—to displace the intracranial gas and place it in optimal positions for diagnosis—expose the bridging veins to additional pathogenic stresses.

Chronic infantile hydrocephalus has not been reported as a residual in the follow-up studies of abused infants but has been found in many cases of chronic subdural hematoma.[14] Since subdural hematoma is a common complication of infant abuse, it is possible that residual hydrocephalus has been present but missed because it was not adequately searched for in the currently available inadequate studies of the late sequels in shaken infants. Cephalic enlargement of idiopathic hydrocephalus may not be noted until several weeks or months after birth, which raises the high probability that some, or even much, of the hydrocephalus which has been attributed to birth injury actually resulted from early postnatal whiplash-shaking. The studies of Laurie and Berne[20] and several others have demonstrated that subarachnoid hemorrhage is an important cause of the hydrocephalus engendered by birth injury; this may be equally true for the postnatal injuries caused by whiplash-shaking.

Russel[21] states that the hydrocephalus-producing gliosis of the brain which follows hemorrhages is so similar microscopically to the hydrocephalus-producing gliosis which follows infections that the two cannot be satisfactorily differentiated. The obstructing subependymal gliosis which surrounds the aqueductus cerebri and causes the most common type of ventricular hydrocephalus in infants may be either infection-dependent or hemorrhage-dependent. The same is true for the neuroglial membranes in the fourth ventricle which block the foramens of Luschka and Magendie and may cause the cystic hydrocephalus of the fourth ventricle (Dandy-Walker syndrome).

According to Russel, the meninges react similarly to several kinds of particulate matter in the spinal fluid and produce an inflammatory exudate, followed later by a localized gliosis (fibrosis). Blood extravasated into the ventricular and meningeal spaces can, thus, cause obstructions to the flow of cerebrospinal fluid and back pressure dilatations at any and all levels. Russel also cites examples of hydrocephalus due to hemorrhages incidental to brain surgery. There is, therefore, good evidence that hydrocephalus is frequently caused by intracranial hemorrhage and also good evidence that intracranial hemorrhage is a common feature of the whiplash-shaken infant. Hydrocephalus, however, has not been reported as a significant sequel of abused infants (shaken infants). More adequate late studies of large populations of previously traumatized infants are needed for a satisfactory solution of this contradiction.

Mental retardation occurred in high incidence in two small follow-up studies of abused infants. In Elmer's[22] group of 22 infants, 12 had intelligence quotients of less than 80. This incidence of 12 in 22 becomes increased to 17 in 22 (77%), if one adds five more retarded children who were not available for interviews because they had already been admitted to state institutions for the mentally retarded. None of 67 nonabused children in the control group had similarly low IQs. Morse et al[23] found the incidence of mental retardation to be 60% in 15 children who had been followed for three years after the original injury. Kempe et al reported brain damage in 25 of 45 fatal cases in 1962.[9] These high incidences in three reports suggest that permanent brain damage and mental retardation develop in a surprisingly high number of infants in whom traumatic abuse has been detected. More detailed studies of large populations with

valid controls are needed for determination of the true incidence.

Characteristic, consistent structural changes in the brain have not been demonstrated in so-called idiopathic juvenile mental retardation, which includes 80% to 90% of all cases. In the case of whiplash-shaking, cerebral injuries, it is possible that the original, early traumatic cerebral edema, the small cumulative chronic subdural hematomas, the petechial hemorrhages, and even the larger cerebral contusions have all disappeared completely by the time, years later, the brains are available for autopsy. Owing to the frequent lack of comparable degrees of structural changes in the brains of children who are severely retarded mentally, it has been suggested that the original injuries caused irreversible change in the neurons themselves—changes which are not detectable microscopically or chemically.

The cerebral lesions of whiplash-shaken children have not been studied systematically. In some studies of blunt traumatic injuries to the head, the changes in the brain have differed significantly in younger infants from those in older infants, children, and adults. Diffuse gliosis was common in the youngest brains and may have been the residual of earlier traumatic cerebral edema and hemorrhage. The authors point out that mental deficiency and motor defects may be due to the destruction of the matrix cells around the lateral ventricle.

The most striking documented clinical and pathologic example of mental deficiency, induced by repeated whiplash jolts to the head and which are associated with profuse petechial hemorrhages in the brain, is the so-called punch-drunk or slap-happy veteran fist-fighter. He develops severe losses of memory and impairments of judgment with speech and gait disturbances from being repeatedly jabbed and beaten over the head during years of exposure in the boxing ring. He may never suffer detectable acute brain injury from a single heavy blow; he does suffer, however, from the cumulative effects of numerous, milder, repeated jabs which jolt his brain. If one cared to paraphrase the cruel but realistic lingo of the fight game, one could accurately describe some mentally retarded children as "jolt-dolts" or whiplash-silly.

The exact prevalence of idiopathic juvenile mental retardation in the general child population is not known but all responsible estimates indicate that it is woefully high—as high as 2 million cases in persons younger than 18 years.[24] The rate during infancy is set at 0.5% to 1%. Ten percent to 15% of children in large metropolitan centers have been classified as mentally subnormal. It is obvious that, if the whiplash-shaking of infants is even a minor cause of such a pervasive and devastating disorder, its prevention would eliminate substantial amounts of massive human suffering and misery, as well as monumental socioeconomic wastage. If one assumes that the average family unit for each retarded child includes two parents and two siblings, we can then appreciate that the lives of 8 to 10 million additional humans are dwarfed and darkened by this most baneful of all chronic human scourges—juvenile mental retardation.

Cerebral palsies and idiopathic epilepsy appear to be due to postnatal trauma in many cases, especially in those cases in which the clinical signs first appear weeks, months, and years after birth. It is probable that postnatal whiplash-shaking plays an important causal role in some or many of these patients. Neither of these disorders has yet been reported as residual effects in the PITS. More, and more comprehensive, studies are needed to determine the causal significance of whiplash injuries to the infantile brain, in residual cerebral palsies and idiopathic epilepsy.

Summary and Conclusions

Shaking is generally disregarded as a type of causal violence in the PITS by both parents and physicians and it is rarely mentioned in medical histories. Thus, both its frequency and potential pathogenicity are consistently depreciated and ignored.

The radiographic bone changes in the PITS, both their nature and distribution, indicate that they are usually caused largely by rough manhandling of the infantile arms and legs, such as grabbing and then grasping, squeezing, wringing, and jerking, and then whiplash-shaking of the head; all usually occur in the absence of bruises in the skin of the arms and legs.

Several examples of pathogenic jerking of the extremities and whiplash-shaking of the head have now been recorded in the PITS; six of these were fatal.

Whiplash-shaking is widely practiced in all levels of society, by a wide variety of persons, in a wide variety of ways, for a wide variety of motives.

The pathogenicity of ordinary, casual, habitual, customary, repeated shaking of infants is generally unrecognized by physicians and parents.

The infantile head is especially vulnerable to whiplash injuries owing to a combination of the normal relatively heavy head and weak neck muscles, to the plasticity of an immature, partially membranous calvarium, and to the softness of an immature, unmyelinated brain.

Some, perhaps many, of the cerebrovascular injuries which are currently attributed, both clinically and microscopically, to prenatal infections, congenital malformations, birth injuries, and genetic metabolic diseases are undoubtedly caused by undetected, depreciated, and inapparent whiplash-shakings during the first weeks and months of life.

In the follow-up studies of two small groups of previously traumatized children, mental retardation was found to have a surprisingly high incidence. The mental status of these patients was not determined prior to their traumatic abuse.

The evidence on which our concepts of the pathogenicity of infant-shaking is based does not lend itself to satisfactory statistical analysis; "universal" samples of a total population of shaken infants have not been ob-

tained, nor have adequate matching controls of unshaken infants from the same socioeconomic milieu.

1. Whiplash-shaking of the infantile head is always potentially pathogenic to some degree. Its actual incidence is unknown and cannot be even estimated satisfactorily.

2. Whiplash-shaking appears to be practiced widely in all levels of society for many different reasons. It is generally ignored by physicians.

3. The habitual, repeated, relatively mild whiplash-shakings which are inflicted in the ordinary training and disciplining of infants, and which may induce undetected cumulative chronic subdural hematomas and other undetected chronic types of brain damage, are probably more pathogenic than the less frequent but more violent and conspicuous shakings during wilful assault, because they are consistently unrecognized and may persist to generate mental retardation and permanent brain damage.

4. There are several features of infantile subdural hematomas which indicate that they are not usually caused by *direct impact injuries* to the head, but are caused by *indirect acceleration-deceleration traction stresses* such as whiplash-shaking of the head. These features include bilaterality of subdural hematomas in 85% of infants[13] and frequent bilateral retinal hemorrhages. There is a striking lack of such signs of impact injuries such as blows to the head. Usually there are no bruises to the face or scalp, no subperiosteal cephalhematomas, and no fractures of the calvarium.

5. There is considerable manifest and much circumstantial evidence which indicates that whiplash-shaking and jolting of infantile heads may be major, unrecognized causes of mental retardation and permanent brain damage. The wide practice of habitual whiplash-shaking for trivial reasons warrants a massive nationwide educational campaign to alert everyone responsible for the welfare of infants on its potential and actual pathogenicity.

6. Trauma is the most important killer and crippler of infants and children and it warrants aggressive study to insure effective preventive and optimal diagnoses and management. It seems certain that a new pediatric subspecialist will soon emerge, a pediatric traumatologist, who will head trauma teams in the larger medical clinics and attack the problem of traumatic diseases with the same success as other pediatric subspecialists have already achieved in the infectious, neoplasms, metabolics, deficiency, and genetically determined diseases.

The problem and the prevention of whiplash injuries are summarized in the following quatrain:

Hark ye, good parents, to my words true and plain,
When you are shaking your baby, you could be bruising his brain.
So, save the limbs, the brain, even the life of your tot;
By shaking him never; never and not.

COMMENT.—Dr. C. Henry Kempe, University of Colorado, made available data from his new book *Helping the Battered Child and Its Family* (J. B. Lippincott Co). In chapter 7, by Harold Martin, 42 abused children were followed for three years. He found 33% functionally retarded; 93% of these had a history of trauma to the head. Subdural hematomas or skull fractures were found in 31% and 43% had neurologic residuals. Dr. Martin points out the complexities and interfering factors in attempting to establish exactly the amount of permanent brain damage and mental retardation which results directly from traumatic abuse.

A comprehensive and authoritative biography of Abraham Jacobi by J. S. Leopold can be found in Borden Veeder's *Pediatric Profiles* (C. V. Mosby Co, 1957).

Drs. Bertram Girdany and Albert B. Ferguson generously provided much excellent radiographic and clinical material from the departments of radiology (Dr. Girdany) and orthopedic surgery (Dr. Ferguson), Children's Hospital of Pittsburgh.

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ARTICLES

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

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The theme of this report is fourfold: (1) it presents the essential clinical manifestations of the *whiplash shaken infant syndrome;* (2) it presents evidence which indicates that many so-called *battered babies* are really *shaken babies;* (3) it emphasizes the high vulnerability of the infantile head, brain and eyes to habitual, manual, whiplash stresses of ordinary shaking by the extremities; and (4) it supports the hypothesis that casual, habitual, manual whiplash shaking (WLS) of infants is a substantial primary, frequent cause of later mental retardation and permanent brain damage.

Subdural hematomas, intraocular bleedings and multiple traction changes in the long bones were the essential elements in our first descriptions of the *original six battered babies* in 1945.[1] In 1972 we reported that shaking was the probable major cause of traction stresses in the periosteums of the long bones, and also of intracranial and intraocular bleedings.[2]

Today we direct your attention to some important new evidence on the pathogenicity of WLS; some new examples of admitted WLS; necropsy findings in two cases; two cases of residual mental retardation at 12 months, which became evident several months after admitted WLS during early infancy; and one case of fatal cerebral palsy in an infant 10 months of age, who had been manually shaken at 2 months. We shall also evaluate the diagnostic significance of manual whiplash shaking, of the findings in the history, physical examination, necropsies and fundoscopic examinations. The pathogenic significance of subdural hematomas, the excessive pliability of the immature skull and brain, and the cumulative, progressive intracranial bleedings engendered by habitual but mild WLS will be correlated.

EVIDENCE FOR THE PATHOGENICITY OF WLS DERIVED FROM THE RADIOGRAPHIC CHANGES IN THE LONG BONES OF SO-CALLED BATTERED BABIES

The first suggestions that many supposedly *battered infants* were actually *shaken infants* came from radiographic studies of scores of supposedly battered babies during several decades. Finally, it became conclusively clear that a reasonable explanation for the pathogenesis of these common lesions—metaphyseal avulsions and subperiosteal hemorrhages—was traction-stretching stresses on the periosteums, induced by grabbing the infants by the extremities or by the thorax, and then shaking them, which in turn induced whiplashing of the head onto the thorax. WLS of the head was the reasonable explanation for the presence of bilateral subdural hematomas and bilateral intraocular hemorrhages, combined with concurrent absence of external signs of trauma to the head and neck

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and to the extremities in more than half of our early cases.

EVIDENCE DERIVED FROM ADMISSION BY THE ASSAILANT IN THE HISTORIES OF SO-CALLED BATTERED BABIES

Direct evidence of trauma through admission by the parent-assailant or the statement of a witness is rarely obtained or obtainable. Usually there are no witnesses. The medical history of manual WLS is practically never obtained because it is considered innocuous by both the parent-assailant and the questioning physician. Although our evidence, based on admission by the assailant, is meager, it is valuable because it is reliable.

By far the most extensive anecdotal proof of pathogenic manual WLS comes from the confessions to the savage shakings of dozens of infants by an infant-nurse[3,4] who whiplashed three infants to death, maimed two others, and shook uncounted others during a period of nine years. She stated that "one of her babies" died after she had "pounded it on the back to get a bubble up." Prior to a coroner's inquest she broke down and confessed that "baby K refused to drink her bottle so I seized her by both arms and shook her until her head bobbed and she became faint. Then I quickly put her down." After a preliminary investigation, the Coroner charged that this infant-nurse had caused the infants' deaths during her "uncontrollable fits of anger and uncontrollable urge to grasp infants between the elbows and shoulders and shake them as long as they persisted in crying." Two others of the whiplashed babies were said to have been manhandled so severely that they became painfully injured. One year after the Coroner's inquest, two fathers, who had employed this same nurse to care for their two infants earlier, reported that each of the infants had become retarded mentally. As soon as the Coroner's report became known to the community, "dozens" of mothers reported that their infants had been significantly injured by this same nurse, and a review of the office records of several pediatricians confirmed additional injuries to many other infants from the same source. It is amazing that this infant-nurse was able to continue her brutal WLS practices during the long period of nine years in an enlightened academic community, on the infants of well-educated parents and on the patients of well-trained expert pediatricians. This was due in large part to the absence of external signs of trauma after violent often repeated pathogenic shaking.

Necropsies were done in two of the three fatal cases. The protocols are dated 1948 and 1956 for babies H and K, respectively.

Baby H, 12 days of age, premature girl was well until tonight, when she awakened crying as if in pain. There were no external signs of trauma. Nutrition good, respirations deep and gasping, anterior fontanel bulged slightly, diffuse hemorrhages in the ocular fundi. Death three hours after admission. No history of trauma, no fracture of calvaria. Necropsy findings: skin normal, thymus large. Microscopic focal hemorrhages in the myocardium; pinkish cellular exudate in the pulmonary alveoli; small subcapsular laceration of the liver filled with fresh blood, liver capsule intact. Brain and head: bulging anterior fontanel, bilateral subdural hematomas, bilateral subarachnoid bleedings, subpial bleeding, lacerations of the cerebral parenchyma, pyknosis and death of ganglion cells and large perivascular bleedings. Eyes: optic nerves congested and edematous; external bleeding throughout the fibrous layer of the retina with scattered hemorrhages in its inner nuclear layer.

Baby K, girl 11 weeks of age. Chief complaint: bulging of anterior fontanel. Fell asleep well but awakened crying and lethargic; semicomatose on arrival, tachypneic, fontanel bulging, reflexes hyperactive, ocular fundi invisible (bleeding?), moderate generalized cyanosis. Cerebrospinal fluid was bloody, gross fresh blood. Infant turned greyish and died 2 hours after admission. No external signs of trauma on the face or head. There was a small "sheet" burn at the left knee and a short superficial scratch in the abdominal wall. No evidence of fractures. Several small foci of atelectasis in the lungs. Brain: no external signs of trauma to the head; bilateral subdural hematomas with subarachnoid hemorrhages, as well, over both cerebral hemispheres; extensive bleedings at the sites of attachment of the bridging cerebral veins to the superior sagittal sinus. Microscopic findings: extensive subarachnoid but no intracerebral bleedings; eyes not examined.

The findings in these two necropsies demonstrate that the manual WLS by an adult assailant was pathogenic, especially to the brains and eyes. The harmful effects of manual shaking are probably maximal at the tender ages of these two shaken infants. The absence of any external signs of trauma to the head notwithstanding the presence of massive intracranial and intraocular hemorrhages is especially noteworthy and significant in the pathogenesis of the intracranial and intraocular bleedings.

Guthkelch[5] found subdural bleedings in 13 of 23 abused infants, 22 of whom were younger than 18 months. Five of the 13 had no external signs of head trauma or cranial fracture. At necropsy, in one case bleedings over the cerebral cortex, and in another extensive cortical bruisings were found at

surgical exploration. In two additional cases, not included above, the clinical histories indicated that shaking rather than beating (battering) was the cause of subdural hematomas. In one infant 6 months of age, convulsions and a bulging fontanel, without a history of trauma or external physical signs of trauma, suggested the presence of subdural hematomas. This infant died three days later. At necropsy, several of the bridging cerebral veins were torn from their attachments at the falx cerebri. Also the edge of the underlying brain was torn and bruised. Trauma of any kind was denied at first by the mother, but later she admitted that she "held him up and shook him several times, to clear his throat and stop his coughing." The second patient, 6 months of age, was admitted to the hospital with complaints of vomiting and convulsions which led to a provisional diagnosis of meningitis. There were no external signs of trauma to the head, trunk or extremities and no fractures of the skull or long bones. The anterior fontanel, however, bulged. Subdural hemorrhages were found and treated. Soon after discharge of this patient from the hospital, his twin brother was admitted with a broken femur. The first twin returned shortly thereafter with recurrence of subdural hematomas. He now showed oval bruises on the skin of the arms which fitted exactly the sites where the pads of the thumbs and fingers of an assailant might have been applied, when he seized and shook him. At first the mother denied traumatic injury of any kind but later she admitted that the father "might have shaken him when he cried at night." Guthkelch thus discovered two cases: one fatal, with intracranial and intraocular hemorrhages, induced by admitted manual WLS; and in one case intracranial hemorrhages and ruptures of the cerebral bridging veins were demonstrated at necropsy.

In Helfer and Kempe's monograph, *The Battered Child*,[6] Weston reported two infants who had been shaken to death and who had subdural hematomas at necropsy. In the same monograph, Steele and Pollock describe two infants who were "shaken, roughed up and spanked," but who apparently did not develop intracranial hemorrhages.

Silverman[7] found massive traumatic involucrums and metaphyseal avulsions in the bones of the legs of a premature infant 7 months of age who apparently had been shaken manually many times since the second month. The mother shook her after grabbing her by the legs and inverting her. We have observed two infants who have developed typical bone changes after a single, violent jerk of that extremity; and in a third, 8 months of age, after manual shaking by an enraged and jealous sibling brother 8 years of age. Subdural hematoma was not detected in any of these three.

Fractures of the spine with local injuries to the spinal cord of one infant were attributed by Swischuk[8] to manual WLS.

A bizarre example of pathogenic manual WLS was reported by Guarnaschelli and associates.[9] An infant 2 months of age was treated for "sunken fontanelle" (Caida de Mollera) by his Mexican grandmother. Two days before admission to the hospital, she had attempted to raise the sunken fontanel by a series of therapeutic maneuvers which terminated in holding the infant topsy-turvy by its ankles, with its head over a pan of water, and then shaking the infant up and down while an assistant slapped and pounded on the soles of its feet. The sunken fontanel did rise and had become bulgy when admitted to the hospital. Subhyaloid hemorrhages were found in the ocular fundi and the pupils were fixed. There were no signs of external trauma to the head, trunk or extremities. Clonic seizures developed and the cerebrospinal fluid from the cranial subdural space and the lumbar subarachnoidal space contained fresh blood. The clinical signs subsided after treatment of the subdural hematomas. The infant died 8 months later at 10 months of age from pneumonia and quadriplegia. In this case, manual longitudinal WLS of the inverted infant with concurrent pounding of the soles of its feet induced longitudinal whiplash and jerking stresses to the head which resulted in permanent severe generalized brain damage—with cerebral palsies. It is possible that when the head is in the dependent position, while being shaken, the vulnerability to intracranial and intraocular bleeding is increased.

Mushin and Morgan[10] record the story of an infant 3 months of age who appears to have been subjected to a prolonged session of WLS. The father at first attempted to "strangle the infant with a blanket," but when the infant convulsed during this assault and became stuporous, the father apparently repented, and then with the mother spent the rest of the night manually shaking the comatose infant in a belated effort to revive it. The next morning the infant was admitted to the hospital with extensive bruising of the skin and bilateral intraocular bleedings. It died 24 hours later. Necropsy findings included bilateral large subdural hematomas and widely scattered intraocular hemorrhages in the retinas. Microscopic examinations disclosed extensive intraretinal subhyaloid and small vitreous hemorrhages. In this case it is likely that the stresses of strangling as well as those of manual WLS contributed to the intraocular changes and possibly the subdural hematomas. In view of the fact that both parents spelled each other during several hours in vigorous

paroxysmal manual shaking, the probabilities are high that the head of the infant was subjected to several hundred individual whiplash stresses from shaking alone.

EVIDENCE FROM THE PHYSICAL EXAMINATION ON THE PATHOGENICITY OF MANUAL SHAKING

The most characteristic pattern of physical findings in the whiplashed infant is the absence of external signs of trauma to the head and the soft tissues of the face and neck, and of the facial bones and calvaria, in the presence of massive traumatic intracranial and intraocular bleedings. This is an extraordinary diagnostic contradiction. It is, in large part, responsible for the frequent failures to diagnose subdural hematoma and retinal hemorrhage, and the failure to attribute them to manual shaking and whiplashing of the head. It is obvious that in such cases, routine examinations of the ocular fundi in both sick and well infants would provide a convenient and accurate method for the early diagnosis of bleedings in the eyes, and more effective treatment and prevention of WLS. Fundoscopic visualization should become a routine practice in the examination of infants for the detection of the pathogenic whiplash shaking.

EVIDENCE FROM OTHER TESTS ON THE PATHOGENICITY OF WHIPLASH SHAKING

In selected cases cerebral pneumography, cerebral angiography and isotopic scanning are all valuable procedures in the diagnosis of subdural hematoma and intracerebral hemorrhages. In the mass routine detection of subdural hematomas, less hazardous and more convenient complementary procedures such as electroencephalography, sonography and perhaps electroretinography could be employed in selected cases after the fundoscopic changes were demonstrated.

EVIDENCE OF PATHOGENIC WLS IN NECROPSIES

In two cases the most significant findings were limited to bleedings in the brains and eyes, in the subdural, subarachnoid and subpial spaces and in the cerebral substance itself. In one necropsy the optic nerve was congested and edematous. Extensive hemorrhages were demonstrated in both the fibrous and nuclear layers of the retina. The walls of the inferior and superior sagittal sinuses were lacerated at the sites of the attachments of the bridging cerebral veins. Clots of blood covered parts of the falx cerebri. Ganglion cells were sparse and some were pyknotic. The only significant finding outside the head was a subcapsular hemorrhage in the liver in one case. Microscopic focal bleedings in the myocardium were also found in one case. There were no external signs of trauma to the head in either of the infants.

THE PATHOGENIC SIGNIFICANCE OF OCULAR LESIONS IN BATTERED AND WLS INFANTS

In the first six reported *so-called battered babies,*[1] retinal hemorrhages were present in two, both of whom had subdural hematomas. Intraocular hemorrhages have been reported in several *so-called battered infants* since. Similar ocular lesions have been reported in *shaken infants* by Mushin and Morgan in one case; in two infants, victims of the "shaking infant-nurse" (see above); in two cases by Guthkelch[5] and one by Guarnaschelli.[9] Mushin[12] found ocular changes in 12 of 19 *battered infants,* all 12 of whom had permanent impairment of vision in one or both eyes.

In 1964 Kiffney[11] found bilateral retinal detachments behind incomplete cataracts in one so-called battered infant 7 months of age, which were originally diagnosed as retinoblastomas. In follow-up studies of 16 battered infants, Maroteaux and associates[13] found plaques in the ocular fundi which tended to be located in the periphery of the temporal segments of the retinas. The authors state that these lesions cannot be satisfactorily explained on the basis of battering and they question the validity of the term "battered child" for all abused infants, and its worldwide use in English. They propose that some of the affected infants are the victims of overvigorous *manipulations,* not battering. We agree with them and believe that many of these infants are whiplash-shaken rather than beaten, especially those with intracranial and intraocular bleedings. They also report resorption (lysis) of the nasal septum of several abused infants in France, a lesion which I have never seen personally, nor have I seen reported in an abused infant in America. Aron and associates[14] found similar retinal spots located in the peripheries of the temporal segments of the fundi, some with retinal detachments in all 18 abused infants. More than half of these lesions had persisted for more than ten years and one as long as 19 years.

In 1967 Gilkes and Mann[15] stated that they had found only one reference, that of Kiffney, on the status of the eyes of abused infants. In their cases, they were impressed with the extensive spread and the persistence of the signs of ocular hemorrhages, both preretinal and intraretinal, and by the presence of gross papilledema in some cases. These authors cite the patients of Wallis who suffered from subdural hematomas induced by the parents who "gripping the infants by the ankles

swung him in a circle around their head" (so-called cracking the whip); and the infant of Breinin who "had a traumatic retinopathy" after having been gripped by the chest and shaken violently.

Eleven abused infants studied by Harcourt and Hopkins[16] had ocular complications, eight had permanent impairments of visual acuity and ten had intraocular bleedings. In five abused infants Freindly[17] found vitreous hemorrhages, bilateral cataracts, dislocated lenses and retinal detachments. The first case is possibly an example of bilateral cataracts from manual whiplash shaking in view of the lack of a history of trauma and lack of external signs of trauma to the head. In these reports by ophthalmologists manual shaking was admitted in three cases and was probable in several others. In the interesting report of Phelps[18] two infants, 31 days and 2 months of age, respectively, had numerous pale-centered retinal hemorrhages which were totally unexplained; there was no history of trauma and there were no external signs of trauma on the bodies. It is possible that in such cases, manual WLS is the primary causal traumatic factor.

The pathogenesis of retinal hemorrhages in the manual WLS of infants and children cannot be evaluated satisfactorily without a consideration of the incidence, nature, and persistence of idiopathic retinal hemorrhages of the newborn. Sezen[19] found retinal hemorrhages in 14% of 1,238 newly born infants immediately after birth. Between the third and fifth day this incidence of 14% had diminished to 2.6%. This indicates that most of the idiopathic retinal hemorrhages of the newborn infant disappear during the first weeks of life, in contrast to the observations of Aron[14] who found that the retinal hemorrhages in abused infants persisted for ten years and in one case for 19 years. Planten and Schaaf[20] concluded that idiopathic retinal hemorrhages of the newborn infant appear in 20% to 30%, but that they cannot be causally related to increased intracranial pressure during labor, they rarely occur during breech deliveries or cesarean section, and are rarely associated with subdural hematomas or other signs of brain damage. Schlaeder and others[21] detected idiopathic retinal hemorrhages, directly after delivery by the vacuum method (ventouse), to be about three times as frequent as after normal delivery (41/100 compared to 15/100). Baum and Bullpit[22] compared the incidence of idiopathic retinal hemorrhages and idiopathic conjunctival hemorrhages in the newly born infant. Retinal hemorrhages seemed to result from several causes: increase in the blood viscosity and polycythemia (RBC) appeared to be the major causal factors. Conjunctival hemorrhages on the other hand appeared to result principally from increases in the cephalic pressures. The preponderance of the evidence from several sources indicates that the idiopathic retinal hemorrhages of the newborn infant are not due to trauma at time of birth and the retinal hemorrhages found in battered and shaken infants are probably caused by postnatal manual shaking.

SIGNIFICANCE OF SUBDURAL HEMATOMA IN PATHOGENESIS OF THE SHAKEN INFANT

Subdural hematoma is the most common, most injurious and the least understood lesion in the *shaken infant*, and it is also the most frequently undetected. It is by far the most frequent cause of death of so-called battered infants. Infantile subdural hematomas are caused by trauma in practically all cases, and they are bilateral in more than 80% of cases. They remain undiagnosed in most cases owing to the lack of a history of trauma, the vagueness of the clinical picture and the usual diagnostic paradox of massive intracranial bleedings in the absence of external signs of trauma to the head and face. Diagnostic changes of bleedings in the ocular fundi are also commonly present. These are missed of course when the fundi are not adequately examined. Blood in the subdural and cerebrospinal fluid often confirms the diagnosis. Blood apparently is rarely identified in the urine.

According to Ingraham and Matson[23] the frequency and accuracy of diagnosis depends largely on the intensity of the search for subdural hematomas. General practitioners and pediatricians ordinarily do not have a high index of suspicion for subdural bleedings owing to the vagueness of the clinical findings and the failure of parents to offer the history of trauma voluntarily when questioned by the physician. Ingraham and Matson found subdural hematomas from all causes to be lesions essentially of the first year of life with a peak-age incidence of 6 months. In their study of 319 cases, diagnosis was made during the second through the fifth months in 32, 30, 27 and 28 cases, respectively, and during the 20th through the 24th months in only three, one, one and six cases, respectively. Their histories disclosed a high incidence of such common infantile complaints as vomiting, 48%; hyperirritability, 41%; infections, 39%; stupor, 32%; history of birth trauma, 26%; and of postnatal trauma, 20%. The absence of a history of trauma of any kind in 54% is significant and suggests that whiplash shaking may be the cause in many patients. Their common physical findings included fever, 57%; hyperactive reflexes, 52%; bulging fontanel, 36%; anemia, 31%; enlarged head, 29%; abnormal ocular fundi, 22%; paralyses,

19%; and fractures of the skull, 9%. The absence of bulging fontanel in 64%, of enlarged head in 71%, of fundus changes in 78%, of paralyses in 81% and of fractures of the skull in 91% is noteworthy. Signs of external trauma to the soft tissues of the head and neck were apparently so rare that they were not included in the records. It is manifest from these results in a relatively large, carefully studied group of infants suffering from proved subdural hematoma, that subdural hematoma should never be excluded because of an absence of a history of trauma, or absence of external signs of trauma to the head or eyes in the physical examination.

These facts indicate that many features of post-traumatic subdural hematomas are not satisfactorily explained or understood; namely, the nature of the primary causal trauma in more than half of the cases, the exact causal mechanisms of the combination of subdural and intraocular bleedings, or vulnerability of the affected structures and tissues to whiplash stresses. The usual presumption that the chronic subdural hematomas are caused by a single acute traumatic episode which produces immediate and massive hematomas which then maintain their volume or increase in volume due to oncotic pressures is far from proved. Many of these facts are better explained theoretically on the basis of repeated subdural bleedings induced by repeated whiplash shaking which causes progressive cumulative changes in the hematomas over several weeks or months.

THE NATURE OF THE WHIPLASH STRESSES AND THE RESISTANCE OF THE INFANTILE HEAD TO THESE STRESSES

The normal infantile brain and its blood vessels are highly vulnerable to whiplash stresses owing to several normal structural features. The infantile head is relatively heavier and the neck muscles of infancy are weaker than at any other age level. The whiplash stresses on the head from shaking the infant by the extremities and trunk are thus maximized in the infant, in comparison with children whose heads are relatively less in weight and whose cervical muscles relatively and absolutely stronger. The pliable sutures and fontanels are relatively larger and more stretchable in the infantile calvaria, which induces excessive tearing forces at the attachments of vessels to the more rigid fixed soft tissues, such as the falx cerebri, when the head is shaken. The infantile brain case is more supple, so that less forceful whiplash stresses can stretch the brain and its blood vessels more easily. Special lacerating stresses are thus applied to the cerebral bridging veins at the fixed sites of their attachments to the walls of the sagittal sinuses. The infantile brain is unmyelinated and is softer than older myelinated brains. This permits excessive stretching of both brain and vessels. The relatively greater volume of cerebrospinal fluid in the infantile ventricles and in the subarachnoid spaces shift farther and faster during whiplash shaking stresses, and thus increase their stretching effects on the more resistant brain parenchyma and attachments of the blood vessels.

Thus the heavy infantile head with its soft brain, supported poorly by normally weak cervical muscles, fulcrumed on the atlas through the occipital condyles and pivoted on the axis is highly vulnerable to many kinds of whiplash stresses on the head such as shaking, jolting and jerking, especially when they are repeated during long periods.

Shaking of the infant trunk causes a two-phase cycle of rapid, repeated, to-and-fro, alternating, acceleration-deceleration flexions of the head ventrad until the chin strikes the anterior chest wall (sternum), followed immediately by similar but reverse companion extensions of the head on the neck dorsad until the occiput strikes the back (upper thoracic spine). Infants are rarely subjected to a single manual shake. Commonly they are shaken in paroxysms which may be repeated frequently or infrequently over periods of days or weeks, or months in the case of habitual shaking. Many infants receive dozens and scores of whiplash stresses, some hundreds. It is obvious that although the single manual shake of an infant may be less forceful and pathogenic than the single whiplash in an automobile accident, the summation of the injurious effects of the many repeated but less forceful manual shakings may be much more harmful to the brain and the intracranial blood vessels and also to the veins in the eyes. The extremities of the shaker thus may become handles and levers for mishandling (whiplashing) the head; and the hands of an angry parent or jealous older sibling may become "deadly weapons" in either violent whiplash shaking assaults or in protracted habitual less violent casual shakings. Violent or habitual milder slapping or cuffing of the infant's head with the open hand could cause similar but probably less frequent intracranial and intraocular bleedings.

Ommaya, an experienced investigator of whiplash stresses on the brains of small experimental animals, wrote me recently in a personal communication that he agreed with me on the high risk of the whiplash shaking mechanism owing to the high vulnerability of the human infantile calvaria and brain. He and his associates[24] found that the crucial factor in whiplash injuries to the mature head is the inertial effect of the easily deformable brain moving with a time lag after rotating dis-

placement of its much less deformable mature container, the skull. Attachments to the outer surfaces of the brain and the inner surfaces of the skull are thus subjected to powerful tensile and shearing forces. He and his colleagues[25] also found that experimental cerebral concussion as well as gross bruises of the brain and upper cervical cord could be produced by rotational displacement *alone* of the head on the neck. In 1971 Ommaya and Hirsch[26] supported the hypothesis that approximately one half of the potential for brain injury during impact on the head is causally related to head rotation. Rotation of the head is of course a consistent additional stress in the manual ventrodorsal shaking of infants.

Manual whiplash shaking of the head may be a major causal factor in sudden unexplained infantile deaths ("cot" or "crib" death) associated with epidural hemorrhages of cervical spine reported by Towbin,[27] who found such lesions in four infants who were apparently in good health and died suddenly without apparent cause. There was no history of birth trauma or postnatal trauma. They may have suffered unadmitted manual shaking which caused no physical signs of external trauma. Unfortunately the status of the ocular fundi is not mentioned and the possibility of manual shaking was not considered.

THE LATENT WLS INFANT SYNDROME

In 1972[2] we raised questions on the probabilities of WLS during early infancy being one of the primary causes of permanent brain damage and mental retardation. In this paper, we have already reported three cases which establish a direct linkage between admitted manual WLS and mental retardation in two,[4] and severe motor deficits (cerebral palsy) in the third.[9] In follow-up studies of three groups of so-called *battered infants,*[28-30] many of whom may have been shaken infants, the incidences of mental retardation were surprisingly high in two groups (77% and 62%). In the third 33% were functionally retarded.

Habitual, moderate, casual manual whiplash shaking appears to be practiced to some degree nearly everywhere by many types of parents or parent-surrogates for a wide variety of reasons. The common motives for habitual casual whiplash shaking are punitive for minor misbehavior and disciplinary in normal training. The exact frequency, violence and pathogenicity of this type of infantile "mild" assault have never been studied and are not known, even approximately. However, in view of the high vulnerability of all normal infantile brains to several kinds of whiplash stresses, and the usual repetition of these casual milder shakings over protracted periods, it seems reasonable to hypothecate that habitual whiplash shakings are pathogenic to some degree in many such cases. It follows that whiplash shaking may be responsible for repeated, small but cumulative intracranial and intraocular bleedings which slowly engender progressive, cumulative permanent disorders of the brain and eyes such as permanent cerebral palsies, mental retardation and permanent impairments of vision. These facts being true, it is highly probable that the routine regular examinations of the ocular fundi in all, even apparently healthy babies, would detect the residues of retinal hemorrhages and make possible the early stoppage of habitual casual shaking. More important, a massive educational campaign on the potential hazards of the habitual casual manual shaking of infants might prevent the development of "mild" mental retardation and "mild" cerebral palsies in thousands of otherwise normal healthy infants. It is possible that many slow-learning and clumsy children with IQs of 90 might have been intelligent and normally mobile children with IQs of 120, had they not been habitually shaken and whiplashed during infancy.

SUMMARY

Our evidence, both direct and circumstantial, indicates that manual whiplash shaking of infants is a common primary type of trauma in the so-called *battered infant syndrome.* It appears to be the major cause in these infants who suffer from subdural hematomas and intraocular bleedings. The label *"battered infant"* is a misnomer in many cases which may interfere with early diagnosis and proper preventive treatment.

The essential elements in the *infantile whiplash shaking syndrome* present an extraordinary diagnostic contradiction. They include intracranial and intraocular hemorrhages, in the absence of signs of external trauma to the head or fractures of the calvaria, and are associated with traction lesions of the periosteums of the long bones in the absence of fractures and traumatic changes in the overlying skin of the extremities. Usually there is no history of trauma of any kind.

Habitual, prolonged, casual whiplash shakings may produce an insidious progressive clinical picture, the *latent whiplash shaken infant syndrome,* which is often inapparent to both parents and physicians. It usually first becomes evident at school age when minor *idiopathic* cerebral motor defects are first detected along with mild idiopathic mental retardation. Permanent impairments of vision and hearing may also be identified at this time for the first time when the children are 5 to 6 years of age. The actual number of such cases is incalculable from current evidence but it

appears to be substantial.

This concept of the *whiplash shaken infant syndrome* warrants careful diagnostic consideration in all infants with unexplained convulsions, hyperirritability, bulging fontanel, paralyses, and forceful vomiting singly or in combination. The routine careful examination of the ocular fundi of all infants should provide a superior screening method for early and massive detection of pathogenic whiplash shakings along with radiographic examination of the long bones for confirmation in appropriate cases.

Current evidence, though manifestly incomplete and largely circumstantial, warrants a nationwide educational campaign on the potential pathogenicity of habitual, manual, casual whiplash shaking of infants, and on all other habits, practices and procedures in which the heads of infants are habitually jerked and jolted (whiplashed).

The proposed nationwide educational campaign against the shaking, slapping, jerking, and jolting of infants' heads is summarized in the following stanza:

Guard well your baby's precious head,
Shake, jerk and slap it never,
Lest you bruise his brain and twist his mind,
Or whiplash him dead, forever.

J.C.

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Position Paper on Fatal Abusive Head Injuries in Infants and Young Children

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This article represents the work of the National Association of Medical Examiners Ad Hoc Committee on shaken baby syndrome. Abusive head injuries include injuries caused by shaking as well as impact to the head, either by directly striking the head or by causing the head to strike another object or surface. Because of anatomic and developmental differences in the brain and skull of the young child, the mechanisms and types of injuries that affect the head differ from those that affect the older child or adult. The mechanism of injury produced by inflicted head injuries in these children is most often rotational movement of the brain within the cranial cavity. Rotational movement of the brain damages the nervous system by creating shearing forces, which cause diffuse axonal injury with disruption of axons and tearing of bridging veins, which causes subdural and subarachnoid hemorrhages, and is very commonly associated with retinal schisis and hemorrhages. Recognition of this mechanism of injury may be helpful in severe acute rotational brain injuries because it facilitates understanding of such clinical features as the decrease in the level of consciousness and respiratory distress seen in these injured children. The pathologic findings of subdural hemorrhage, subarachnoid hemorrhage, and retinal hemorrhages are offered as "markers" to assist in the recognition of the presence of shearing brain injury in young children.

Key Words: Abusive head injury—Shaken baby syndrome—Head injury—Inflicted injuries.

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Editor's note: The Board of Directors of the National Association of Medical Examiners charged the authors of this article with writing a position paper on the shaken baby syndrome. This article was the result. The manuscript was reviewed by three reviewers on the Board of Editors of the *American Journal of Forensic Medicine and Pathology.* They believed that while it was worthy of publication, it should not be published as a position paper because of the controversial nature of the subject. The Board of Directors responded to this opinion by stating that position papers always deal with controversial subjects.

The original charge to this ad hoc committee was to produce a position paper on shaken baby syndrome. This terminology was taken by the committee to refer generally to the area of abusive head injury in young children. However, because the term *shaken baby syndrome* has taken on such controversy, this article will address the topic of abusive head injury in young children. In several areas of this article, the term *marker* is used when describing the importance of identifying the presence of subdural, subarachnoid, and retinal hemorrhages. The term *marker* indicates a grossly observable sign to signify the possible existence of diffuse axonal injury that is not grossly evident. Use of the term *marker* does not imply that such hemorrhages cannot exist without such an association but is intended to remind us to be alert to the possibility.

Head injuries account for up to 80% of fatal child abuse injuries at the youngest ages (1). Blunt force impact as well as vigorous shaking may play a role in the pathogenesis of these injuries (2). This article describes the state of knowledge concerning the pathogenesis, clinical features, and pathologic changes of fatal abusive head injuries in young children. The intent is to inform the practicing pathologist about the proper recognition, interpretation, and clinical correlation of these injuries.

Caffey's description of whiplash shaking of infants in the early 1970s introduced the concept that serious and even fatal head injury could be inflicted by a caretaker through shaking (3,4). Caffey described injuries characterized by subdural and/or subarachnoid hemorrhages, brain swelling, and reti-

nal hemorrhage without injuries that would indicate impact, such as facial bruises, scalp bruises, or skull fractures. Since that time, experts in many scientific fields have investigated whether such apparently innocent practices as tossing a baby into the air and other playful maneuvers might cause brain damage by a similar shaking mechanism. Currently, it is generally accepted that such playful practices do not result in injuries to the young child's brain. The type of shaking that is thought to result in significant brain injury involves holding the child by the thorax or an extremity and violently shaking the child back and forth, causing the head to forcefully whiplash forward and backward with repeated accelerations and decelerations in each direction.

Abusive head injury commonly occurs in response to prolonged crying and often is inflicted by a caregiver with limited patience or experience in handling a child. Some individuals who admitted to shaking children as a mechanism of injury have stated that shaking would stop the babies from crying. These assailants have actually used this practice to stop babies from crying on previous occasions without any visible adverse result.

When shaking is the mechanism of injury, the arms and legs of the child may also be violently flung about during the shaking, causing injuries to the long bones. Most frequently affected are the tibia, distal femora, and proximal humeri. These lesions were originally described as bucket-handle and corner fractures and were thought to be caused by the indirect forces of the shaking. These injuries are currently recognized as fractures through the most immature portion of the metaphyseal primary spongiosa and appear radiographically as separation of portions (corner) or of the entire disk (bucket handle) of metaphyseal bone, depending on the radiographic plane (5,6). Many babies with fatal abusive head injuries do not demonstrate any external injury, although in about 25% to 50% of cases, such injuries are evident on external examination (7–10). It is important that a careful search is made to identify any injury to the body, such as a bruise or abrasion. Grasping the child by the arms or thorax may result in bruises in these locations, but children may be grasped in this manner without leaving bruises. Likewise, ribs may be fractured while grasping the child around the thorax. At autopsy, close attention should be directed to the rib cage. Fractured ribs should be removed, decalcified, and examined microscopically. Posterior rib fractures are highly specific for abuse. Ribs are the most common bones fractured in association with other abusive injuries of children who die of fatal child abuse (11,12). Infants may sustain abusive head injury of less than fatal outcome and may sustain injuries to the brain that will later be reflected in degrees of mental retardation or slowness, learning disorders, seizures, blindness, or irritability. Of infants who receive abusive head injuries, approximately 7% to 30% die; 30% to 50% have significant cognitive or neurologic deficits; and 30% have a chance of full recovery (13–15). Lethal abusive head injury is not confined to infants. Children as old as 4 or 5 years can be fatally head injured by abuse, although the great majority are under 2 years of age, and most are under 12 months of age (16). Adults may also sustain head injuries by shaking, with findings identical to those found in shaken infants. Pounder described a slightly built, short, 30-year-old prisoner who was grasped by the shoulders and violently shaken. When he died 3 days later, his autopsy demonstrated subdural, subarachnoid, and retinal hemorrhages with diffuse axonal injury (17).

MECHANISMS OF INJURY

Appreciation of the unique characteristics of young children's head injuries requires an understanding of the developmental differences in the skull, brain, and neck before the age of about 4 years. Injuries to the young child's brain are unique in that the trauma occurs to an organ that is in the process of maturing; the mechanisms, the thresholds of injury, and the types of injuries differ from those that affect the older child or adult. The primary features unique to the young child include the thinness and pliability of the skull; the rapid growth of the brain and skull, resulting in a large heavy head; the softness of the brain, which is composed primarily of neurons without dendritic connections; the paucity of the myelin sheath of axons; the relative flatness of the skull base; the undeveloped neck muscles; and the subarachnoid space, which is large in its extent but shallow in depth (18,19). Because of the unique characteristics of the developing skull and brain, children under the age of 4 or 5 years are particularly vulnerable to a type of brain injury that is best described as shearing injury. Shearing injury implies a distortion of the brain shape that elongates it in an anterior–posterior dimension with resulting shifting apart of adjoining brain structures. Impact to the immature brain is more likely to produce shearing injury rather than the typical brain contusions that might occur in older children and adults (19). Impact force is more effectively transferred through the thin pliant skull and across

the large and shallow subarachnoid space of a young child's head. The paucity of myelination, the large number of neurons without glial or dendritic connections, and the small axonal size predispose the young brain to shearing injury by creating a soft consistency. The large heavy head mounted on the weak neck of the young child produces instability of the head, which allows greater movement of the head and brain when acted on by acceleration–deceleration forces. Last, the shallow skull base allows the young child's brain to rotate more readily in response to head acceleration or deceleration than occurs after the skull base has developed more prominent bony ridges and concavities (7,19,20). The mechanical forces that are operative in head injury are primarily translational and rotational (angular). Translational forces produce linear movement of the brain, a type of movement that is quite benign (21,22). The trivial falls that children sustain in falling from furniture and even down stairs primarily involve translational forces. Although such falls may occasionally result in a skull fracture, these incidents are generally very benign and do not result in loss of consciousness, neurologic deficit, or death (23–26). Rotational forces are generated by either impact or nonimpact inertial mechanisms, such as whiplash shaking, which produce sudden acceleration or deceleration of the head. Rotational forces applied to the head cause the brain to turn abruptly on its central axis or its attachment at the brainstem–cerebral junction. Extensive clinical and experimental data have demonstrated that such rotational movements of the brain result in a type of injury referred to as *shearing injury* or *diffuse axonal injury* (23–29). To keep pace with the accelerating skull responding to rotational forces, the brain moves within the cranial cavity, and because of the nonuniformity and different consistencies of the brain structures, strains occur at the junctures between cortex and subcortical white matter, white matter and deep gray matter, and lateral extensions across the midline of the brain (corpus callosum and superior and middle cerebellar peduncles). Disruptions of the axonal processes occur at these junctures as the peripheral parts of the brain rotate farther or more rapidly than the inner, deeper, more relatively fixed parts of the brain. Lesser forces disrupt the most external junctures, whereas greater forces disrupt deeper junctures down to the deep gray matter of the basal ganglia, thalamus, and finally the rostral brainstem. In young children, either an impact or a shaking mechanism may result in diffuse axonal injury, when significant, because both impart rotation to the brain (7,19,20). After the age of 4 or 5 years, the most common cause of diffuse axonal injury is the motor vehicle crash.

Some experimental evidence suggests that shaking alone may not be sufficient to produce the angular acceleration necessary to create fatal shear injury (7). The particular model used in the latter study to determine how much force could be transmitted by shaking to an infant's head utilized as a model a doll with a rubber neck, and the force considered necessary to produce shear injury was derived from studies of adult primates. Neither of these experimental circumstances necessarily truly resembles the immature human infant skull and brain (29). In favor of shaking as a possible mechanism, many forensic pathologists have experience based on confessions by perpetrators or witnesses of how these injuries were inflicted, as well as on autopsies in which no impact site is found on the scalp or skull. However, this experience must be received with some caution. Perpetrators may not remember, or later may not be willing, to fully describe their actions. Children may be violently shaken, then forcefully thrown rather than just placed down gently. Impacts may not be reflected on the scalp if the striking surface is padded or if it is broad and firm. A child's scalp is very elastic and stretches on impact. Not all impacts are registered as hemorrhage in the galea. In the vast majority of cases, it is not possible to definitely characterize children's head injuries as being caused by either pure impact or pure shaking because the pathologic changes in the brain are identical in cases in which either of these two mechanisms has been suggested (7). If there are focal injuries, such as skull fractures, scalp bruises, or subgaleal hemorrhage, an impact can be assumed, but coexistent shaking cannot be excluded. In the absence of signs of an impact, however, shaking alone should not be presumed because there may well have been an impact that cannot be identified (30). Subarachnoid and subdural hemorrhages should be appreciated as markers of brain displacement by angular force and the possibility of accompanying diffuse axonal injury (24,31). In young children, both impact and shaking produce these pathologic findings, which should be appreciated as markers for the underlying problem in the brain: the diffuse axonal injury (32).

SUBDURAL HEMORRHAGE

The grossly and microscopically identifiable pathologic changes in young children's rotation or acceleration–deceleration head injuries include subdural hemorrhage, subarachnoid hemorrhage,

and retinal lesions, including hemorrhages and schisis. Subdural hemorrhage results from tearing of bridging veins, which extend from the cortical surface to the dural venous sinuses (Fig. 1). These rather transparent veins tear when they are stretched as the brain moves within the subdural space of the cranial cavity (23,33–35). Subdural hemorrhages occur most frequently over the convex cerebral surfaces, especially posteriorly within the interhemispheric sulcus, and may be either unilateral or bilateral, although more commonly they are bilateral (Fig. 2). If the subdural hemorrhage is associated with a skull fracture, it need not be on the same side as the fracture. Subdural hemorrhage is probably uniformly present in cases of shearing injury but is evident at autopsy in about 90% to 98% of cases (7,36). Small amounts of interhemispheric blood that can be detected by computed tomography (CT) may not be seen at autopsy (5,37). Very thin layers of subdural blood over the cerebral convexities may not be visible on CT but can be found at autopsy. Magnetic resonance imaging (MRI) is able to detect a much greater number of subdural hemorrhages than can CT; however, many critically ill young children are not able to undergo MRI (19). As a result, studies that report data on nonfatal cases of abusive head injury find that about 80% to 85% of patients have subdural hemorrhage (38). At autopsy, the subdural hemorrhage may consist of only 2 to 3 ml of blood and may not be observed if the prosector does not personally inspect the subdural space as the calvarium is being removed (Fig. 3). Extreme caution should be taken to not misinterpret as premortem subdural hemorrhage the blood draining from the dural sinuses when these are incised at autopsy. The importance of subdural hemorrhage is typically not that of a space-occupying mass lesion producing increased intracranial pressure and the consequences of tentorial herniation, although some hemorrhages are large enough to bring about these complications. Rather, it is important as a marker of brain movement within the cranial cavity and may accompany shearing injury. Subdural bleeding may continue and accumulate to some extent if the child experiences postinjury survival. At autopsy, large subdural hemorrhages resulting in part from postinjury accumulation have been observed. Even a small amount of subdural hemorrhage indicates that brain displacement has been produced, which may have caused some shearing brain injury.

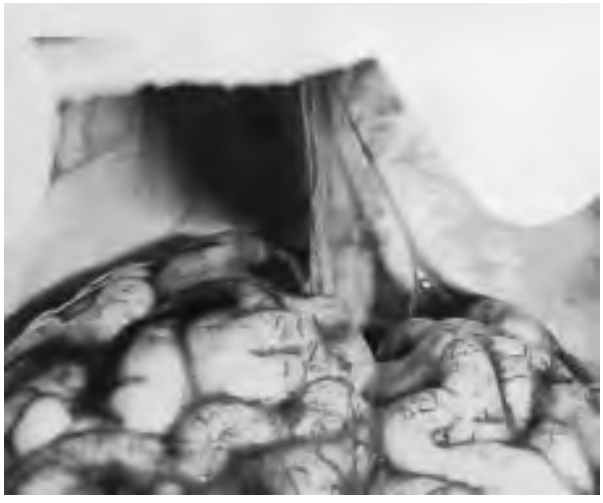

FIG. 1. Thirteen-week-old infant with normal brain and intracranial spaces demonstrating bridging vein (arrow) arising from left cerebral convexity (right).

FIG. 2. Nine-month-old infant with fracture of right parietal calvarium showing bilateral acute subdural hemorrhages over the cerebral convexities.

SUBARACHNOID HEMORRHAGE

Subarachnoid hemorrhage occurs in patches over the cerebrum, especially over the parasagittal cerebral convexities (Fig. 4). It is present in virtually all

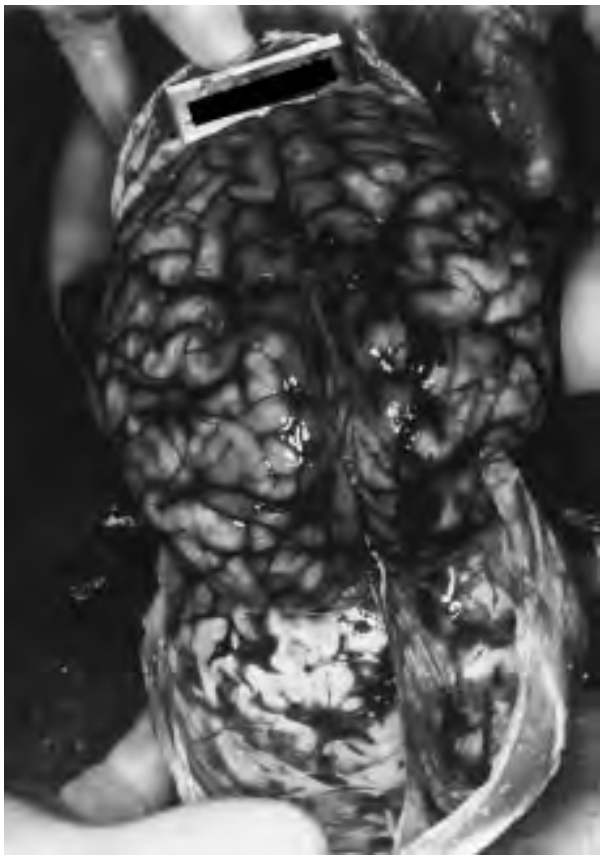

FIG. 3. Eleven-week-old infant with small amounts of acute subdural hemorrhage over both cerebral convexities.

fatal cases, although it may be very small and difficult to identify, especially on the interhemispheric surfaces (Fig. 5). Subarachnoid hemorrhage arises from tearing of arachnoid vessels at the same time bridging veins are torn, because the bridging veins are surrounded by an arachnoid sheath as they cross the subdural space to enter the inner dural layer and finally the dural sinuses. Tearing of bridging veins usually produces both subdural and subarachnoid hemorrhages.

RETINAL HEMORRHAGES

Retinal lesions are observed in 70% to 85% of young children with severe rotational brain injuries (32,39). Currently, their pathogenesis is not precisely understood. Their presence highly correlates with rotational head injury, and they are greatly overrepresented among cases of nonaccidental trauma in young children. Possible mechanisms to account for retinal hemorrhages include increased pressure transmitted to the central retinal vein from increased intrathoracic or intracranial pressure, direct trauma to the retina from being struck by the vitreous moving within the eye, and traction on the retina by the movement of the vitreous pulling away from the retina. The retinal hemorrhages seen in abusive head injuries are similar to those that are frequently observed in full-term neonates after vaginal delivery. In neonates, the hemorrhages appear to be consequent to increased intrathoracic or intracranial pressure from squeezing of the thorax during the passage through the birth canal. Most of the neonatal retinal hemorrhages completely resolve by 5 or 6 days, although a few persist longer (40,41). In children older than 30 days who have retinal hemorrhages, the great majority have abusive head injuries.

Ophthalmologic findings in abused children include peripheral retinal hemorrhages associated with retinal detachments, retinal tears, and large numbers of retinal hemorrhages (39). There may be other internal eye injuries in these children, consisting of vitreous bleeding and retinal folds. There is

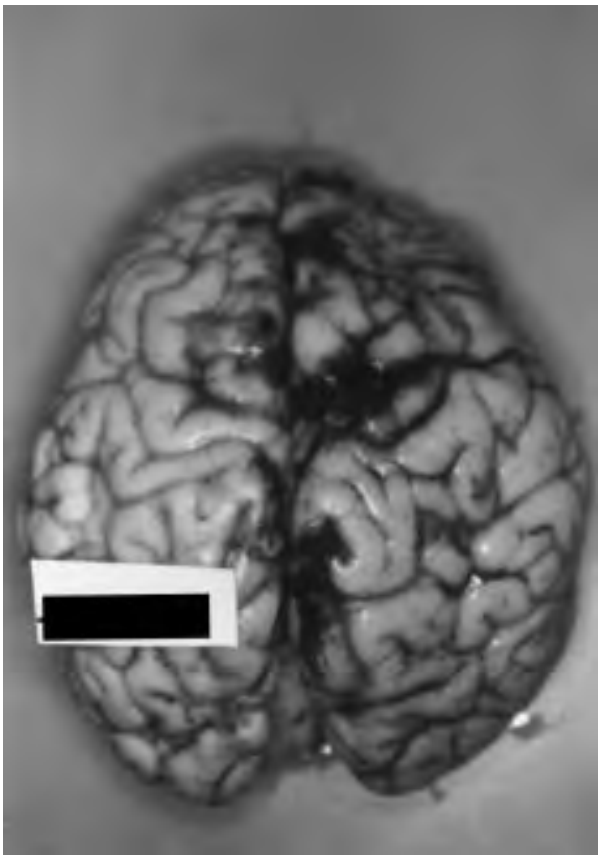

FIG. 4. Seventeen-week-old infant with large (70–80 ml) acute subdural hemorrhage over right cerebral convexity demonstrating patches of subarachnoid hemorrhage over both parasagittal regions, greater on the right than on the left.

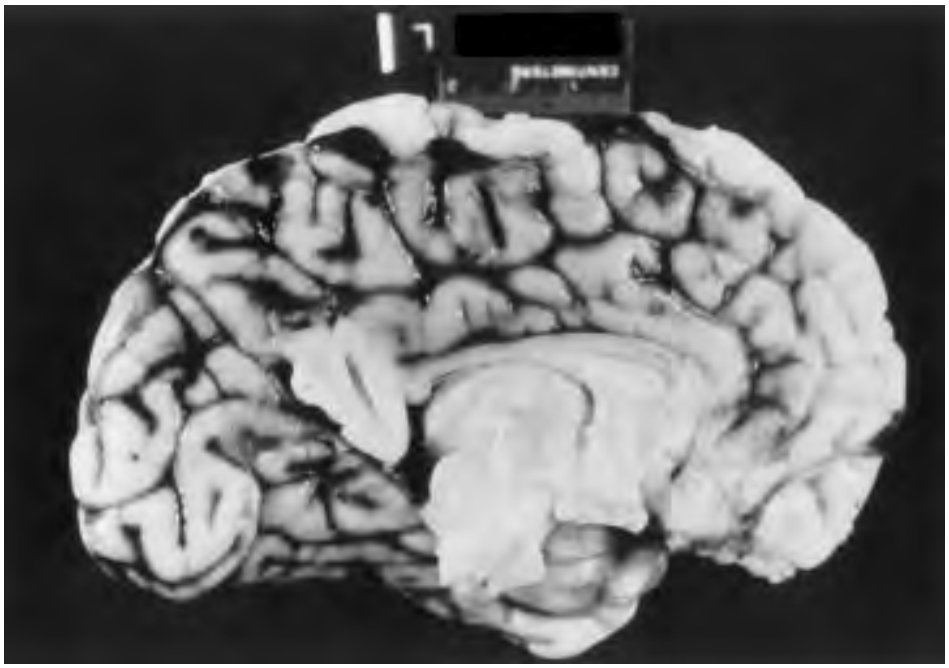

FIG. 5. Five-month-old infant with large bilateral acute subdural hemorrhages over cerebral convexities showing patches of subarachnoid hemorrhage on the mesial surface of the right cerebral hemisphere.

evidence that increasing severity of trauma to the head directly correlates to severe eye damage, beginning with subhyloid and intraretinal hemorrhages and progressing to retinal detachment and finally choroidal and vitreous hemorrhage (42). In children with very severe accidental head injury, (e.g., from a car accident), retinal hemorrhage is occasionally found (26). The retinal hemorrhages associated with nonaccidental head injuries tend to be bilateral, although they may be unilateral, multiple, and extensive and reach far into the periphery of the retina (39). Nontraumatic causes of retinal hemorrhages include bleeding disorders, sepsis, meningitis, vasculopathies, increased intracranial pressure, and, very rarely if ever, cardiopulmonary resuscitation (43–47). Retinal hemorrhages that occur in association with increased intracranial pressure are found at the posterior pole of the retina around the optic disc and are accompanied by papilledema (39).

Optic nerve hemorrhage is observed in association with inflicted head injuries in children but is not specific for those injuries. Optic nerve hemorrhage is hemorrhage in the perineural area. These hemorrhages are seen commonly whenever subdural hemorrhage is found in the cranial cavity, although there is not necessarily a direct connection between the subdural compartment of the orbital sheath and the subdural compartment of the intracranial cavity (42). Optic nerve hemorrhage can be seen in some cases of increased intracranial pressure that are not related to any form of trauma (48).

DIFFUSE BRAIN INJURY

Diffuse brain injury consists of tears of axonal processes and small blood vessels and, rarely, more extensive tissue tears (49,50). The areas of predilection are the corpus callosum; the subcortical white matter, especially of the superior frontal gyri; the periventricular areas; and the dorsolateral quadrants of the rostral brainstem. The axonal disruptions result in microscopic lesions that may be visible by light microscopy after 18 to 24 hours as retraction bulbs or varicosities. Retraction bulbs are accumulations of axoplasm, which appear on hematoxylin and eosin staining as pink bulbs. They are observed as the axoplasm of the disrupted axons accumulates at the damaged end and creates a bulbous enlargement. These axonal lesions are very difficult to see in young children because of the small size of the axonal processes. Immunohistochemical stains for β-amyloid precursor protein may allow demonstration of axonal injury as early after survival as 2 hours (51,52). The blood vessel tears of diffuse brain injury may be visible grossly as linear streaks or punctate hemorrhages, which vary from less than 1 mm up to many centimeters if bleeding continues for several days (31). However, these hemorrhages are very seldom seen in young children with diffuse brain injury because the blood vessels in young children are very elastic and do not readily tear even when adjacent axonal processes are torn.

In some (rare) cases of diffuse axonal injury in children under 1 year of age, parenchymal tears are

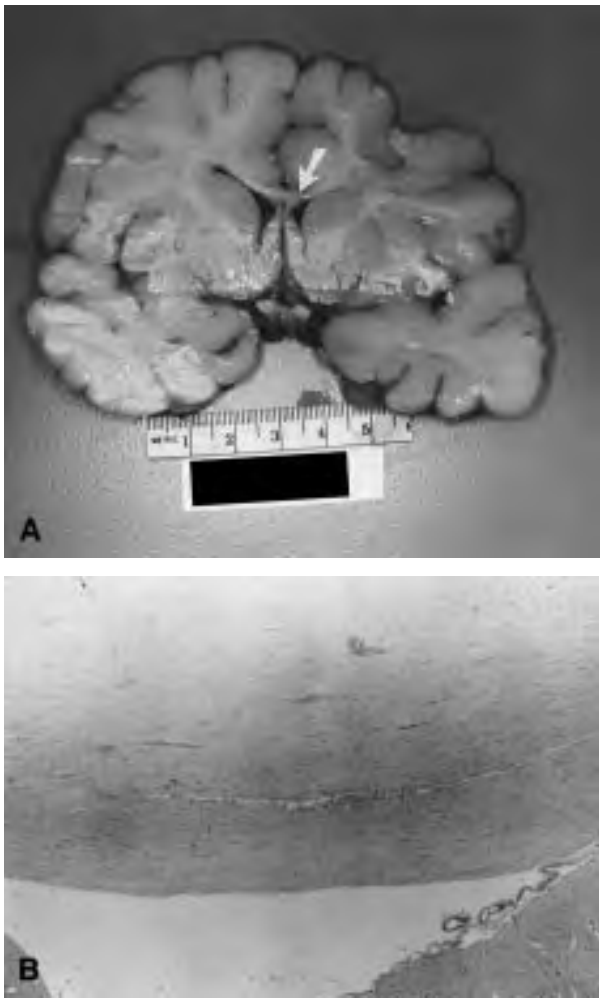

FIG. 6. Seventeen-week-old infant (same infant as in Fig. 4). (**A**) Right side of corpus callosum has a 6-mm linear tear (arrow). (**B**) Photomicrograph of linear tear in corpus callosum with split in the tissue and fresh hemorrhage.

also grossly observable. This is the lesion Lindenberg described as the contusion tear (53). These are slitlike tears that occur at the cortex-white matter junction or within the layers of the cortex and are caused by the differential movements within the brain while some portions of the brain shear or slide apart during differential rotation of the brain tissues (Figs. 6 and 7). Contusion tears are rare, and when seen they are accompanied by the usual markers of diffuse axonal injury, the subarachnoid and subdural hemorrhages in the usual locations (33). Care must be taken not to misinterpret a cross-section through the depth of a sulcus as a contusion tear. It is also prudent to take care to not mistake artifacts created by the process of handling or cutting the brain as true tears. Contusion tears should not be diagnosed on the basis of finding only a microscopic tear without other evidence of diffuse axonal injury.

It is not usually possible to morphologically establish the existence of diffuse axonal injury in young children by demonstrating the classic pathologic changes of retraction bulbs, tissue tears, or intraparenchymal hemorrhages, although these findings may be demonstrated on occasion (33,54). Many of these children die too soon after injury for these pathologic changes to be established. For this reason, it is important to appreciate the markers of shearing injury to identify these cases as diffuse axonal injury.

BRAIN SWELLING

Shearing injuries in young children are accompanied by various degrees of brain swelling. The swelling may not be apparent at autopsy in infants with brief survival intervals. Initially, CT may demonstrate progressive brain swelling and decreased ventricular size without other lesions being visible. The swelling is probably related both to direct injury to the axonal processes, causing localized edema, and to generalized swelling caused by changes in vascular permeability and autoregulation (5). Some investigators have postulated that hypoxia occurring when a child is shaken and becomes apneic accounts for the underlying cerebral insult and brain swelling (55). However, hypoxia does not explain why the injury is sometimes more unilateral than bilateral, the atrophy that develops in the brains in children who survive, or that the appearance of these brains at autopsy is not typical of hypoxic injury.

TIMING OF INJURIES

Timing of the head injury is often an important issue because most abusive injuries occur only in the presence of the individual who injured the child and who may not provide an accurate history. Studies in children dying of accidental head injuries indicate that children with diffuse injury show an immediate decrease in the level of consciousness (16,56). Studies in children with nonaccidental head injuries also indicate that they show an immediate decrease in their level of consciousness at injury (36). Individuals sustaining diffuse brain injury of moderate to severe degree become symptomatic immediately (24,31,49). Young children with moderate to severe degrees of diffuse brain injury would certainly include those in whom there is a significant neurologic outcome or death. Correlations of clinical and experimental observations on

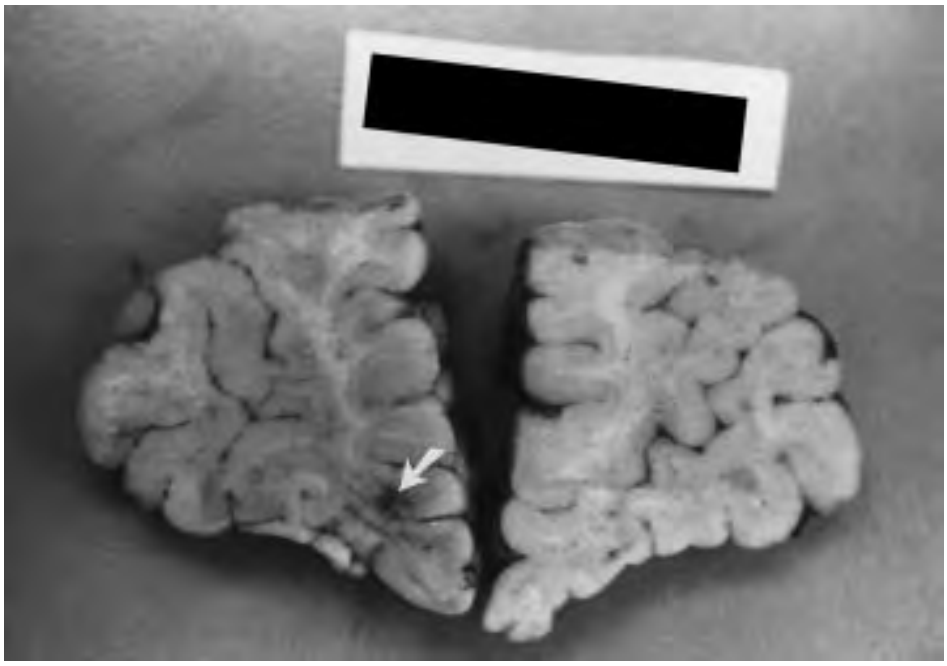

FIG. 7. Seven-month-old infant with depressed skull fracture of left parietooccipital calvarium with small acute subdural hemorrhage over the posterior aspects of both cerebral convexities demonstrating a 3- to 4-mm contusion tear (arrow) in left orbital subcortical white matter.

cerebral concussion and traumatic unconsciousness demonstrate that progressively deeper disconnections of axonal processes affecting the deep gray matter and rostral brainstem are the cause of the unconsciousness in these children, not that the unconsciousness is the result of increasing intracranial pressure or hypoxia (24). Symptoms experienced by these severely injured children include an immediate decrease in the level of consciousness (either lethargy or unconsciousness); respiratory irregularity, difficulty, or apnea; and frequently seizures. The respiratory difficulty in these children may be related to damage to the lower brainstem (medullary) centers of respiratory control. The timing with which respiratory difficulty develops is not very precise, but it is concurrent with or follows the decrease in level of consciousness in these children, who cannot survive for many hours without ventilatory support. It is not currently possible to predict the exact length of time such an injured child may survive.

Children who sustain repetitive episodes of mild diffuse brain injury may gradually accumulate brain damage and acquire neurologic deficiencies (14). The exact timing of such mild additive injuries is not possible. There is no reason to believe that remote shearing lesions would make the brain more susceptible to new shearing injury. Mild injuries unaccompanied by loss of consciousness are not usually brought to medical attention. Some mild shearing injuries are manifested as seizures and clinically present difficult diagnostic problems, because there is no currently available method to demonstrate the underlying pathologic changes of the shear injury until more severe degrees of diffuse axonal damage have been sustained and can be recognized by the markers of subdural or subarachnoid hemorrhage on CT or MRI (38).

CHRONIC SUBDURAL HEMATOMA

Rebleeding after trivial injury or spontaneous rebleeding from a preexisting chronic subdural hematoma should not be offered as an explanation for the presence of acute subdural blood lacking obvious demonstration of such an old subdural membrane (57). The pathogenesis of subdural bleeding has become better delineated after more than 20 years' experience with CT. The classic multilayered chronic subdural hematoma is currently considered a unique type of hemorrhage for several reasons. A chronic subdural hematoma very rarely follows severe head injury in a previously normal person, in whom an acute subdural hemorrhage transforms by aging to become a chronic subdural membrane. Instead, the blood of the acute subdural hemorrhage in these head injuries is readily resolved or rapidly organized (58–60). The resorption of subdural blood tends to be even more rapid and more complete in children than in adults (19). The development of the classic multilayered chronic subdural hematoma results from venous bleeding under low pressure and requires the potential for the subdural space to enlarge without a significant increase in pressure. The

factors that promote such a development within a low-pressure intracranial space exist only in specific categories of people, such as those with brain atrophy (i.e., the elderly and those with alcoholism), those with hydrocephalus who have been treated by placement of a ventricular shunt, or those with traumatic encephalomalacia (59). In children with glutaric aciduria type 1, frontotemporal atrophy develops, and occasionally subdural hemorrhage without trauma develops on that basis (61). Minor trauma in these specific categories of patients may result in tearing of bridging veins and small amounts of subdural hemorrhage, which induces an ingrowth of granulation tissue from the dura. This granulation tissue contains fragile capillaries, which may produce microbleeds leading to enlargement of the hematoma. The further evolution of these hematomas is determined mostly by the nature of the vascular neomembrane formed in these patients. The expansion of these hematomas also appears to be related to the excessive activation of both the clotting and the fibrinolytic systems in the subdural fluid (62–64). A young child whose subdural hemorrhage subsequently organizes into a membrane composed of large vascular channels at risk for rebleeding would have been symptomatic before the time of rebleeding, because there would have been a preexisting brain abnormality. The signs and symptoms that would be expected before rebleeding include seizures, macrocephaly, anorexia, lethargy, headache, and apnea (60).

About 20% to 30% of asymptomatic neonates have small amounts of subarachnoid and subdural hemorrhage during delivery. The resolution of this blood may result in the presence of small numbers of dural macrophages containing hemosiderin and, sometimes, small fibrous patches consisting of a few layers of granulation tissue on the dura. These patches of thin membrane or scattered macrophages are not at risk of rebleeding with trivial trauma. Proof of rebleeding of a chronic subdural membrane should be based on the demonstration of a chronic subdural membrane that is grossly evident at autopsy, followed by microscopic confirmation of the vascularized membrane, and should not be based solely on the microscopic finding of fragments of fibrous tissue or a few macrophages containing hemosiderin.

The dura is a tough, fibrous, bilayered membrane overlying the arachnoid. It consists of an inner layer (menigeal) and an outer layer (periosteal). The periosteal layer serves as the periosteum of the inner table of the skull. The dura of young children, particularly along the basilar skull sutures, is a very cellular structure, which contains growing fibrous tissue along with numerous hematopoietic cells, including macrophages, many of which normally contain hemosiderin. The appearance of the normal young dura may be misinterpreted as having a thin chronic subdural membrane by microscopists who are not familiar with looking at these young duras, who may not be able to tell the inner from the outer dural surface, and who may incorrectly believe a chronic membrane to be the cause of acute subdural hemorrhage.

INTERPRETATION OF INJURY

The distinction between nonaccidental and accidental head injury in children is an area of concern for pathologists as well as other medical specialties. Fatal accidental shearing or diffuse brain injuries require such extremes of rotational force that they occur only in obvious incidents such as motor vehicle accidents. Besides vehicular accidents, other fatal accidental childhood head injuries tend to involve crushing or penetrating trauma, which is readily evident. These injuries tend to be the result of falling from considerable heights (greater than 10 feet) or having some object penetrate the head. There are distinctions between head injuries that are truly accidental and those that are abusive. The incidence of isolated subdural/subarachnoid hemorrhage as the only gross finding in fatal accidental head injuries in young children is less than 2%, compared with the 90% to 98% incidence of these hemorrhages associated with abusive head injuries (Case ME, unpublished autopsy studies). The trivial home accidents that children so frequently sustain are associated with primarily translational forces and not with the rotational forces necessary to develop tearing of bridging veins, which would produce subdural hemorrhage or other shearing injury (26,32,65–71). In low falls of less than about 8 feet, witnessed by uninvolved and nonbiased individuals, about 1% to 2% of children sustain a narrow simple linear skull fracture. In a small proportion of the children who sustain these skull fractures, an epidural hemorrhage will develop. These epidural hemorrhages are not accompanied by a decrease in the level of consciousness at the time of injury. If the epidural bleeding continues and produces significant increased intracranial pressure, there may be a subsequent decrease in the level of consciousness attributable to tentorial herniation.

It is essential that a meticulous autopsy examination be performed in all cases of possible injury to children. When subdural and/or subarachnoid hemorrhage is found at autopsy, the brain must be thor-

oughly examined to exclude the possibility of other causes of bleeding in these spaces. Although berry aneurysms are uncommon in young children, they may occur. Vascular malformations may also occur in young children and cause hemorrhage in intracerebral and intracerebellar subarachnoid and subdural areas. The distribution of bleeding in aneurysms and arteriovenous malformations is unlikely to resemble that of head injury, but these malformations need to be excluded by careful examination of the brain (72,73).

CONCLUSION

Anatomic and developmental differences of the brain and skull of children under the age 4 or 5 years make the head injuries and mechanisms of injury that affect these children different in certain respects from those occurring after that age. Inflicted head injuries in these young children usually create shearing injuries of the brain and blood vessels, resulting in diffuse axonal injury and subdural, subarachnoid, and retinal hemorrhages. The pathologic findings of subdural and subarachnoid hemorrhages and very, frequently, retinal hemorrhages are the most common findings by which these rotational head injuries in young children are identified at autopsy. Recognition of the underlying mechanism of the rotational brain or shearing injury is important to an understanding of the clinical course of these children, particularly with respect to the decrease in the level of consciousness and respiratory distress demonstrated after injury.

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Symptoms Following Head Injury

To the Editor:

A recent proposed position paper regarding pediatric nonaccidental abuse head injury would allege that we may depend on symptoms to appear immediately upon injury (1). I have a concern about that claim, which I will explain with the following case. CASE REPORT

A 13-month-old Hispanic girl was brought to the University of Wisconsin Hospital on the morning of September 18, 1999. The complaint was of vomiting that had lasted for 24 hours. She was described as irritable, sleepy, and vomiting. In our emergency room she was noted to have extensive bruises on the cheeks, chest, back, and arms; the mother attributed these to bites by a 3-year-old housemate. She was admitted and given intravenous fluids. She was sedated with pentothal followed by head computed tomography, which was negative. She was then admitted to the pediatric ward. The resident who saw her described her in the chart and in discussion as being fussy and clingy, but interactive and responsive. Because of the numerous bruises, the police were notified and took pictures.

At about 2:00 the following morning, a nurse coming in to care for the child noted that she had decreased respirations. It was then shown that she was unresponsive and had a right dilated unreactive pupil with a sluggish left pupil. She was taken emergently to the pediatric intensive care unit, where she was intubated and given mannitol. A subsequent computed tomography scan showed very poor differentiation of gray/white matter interface. A Codman catheter was placed and then replaced with a ventriculostomy tube after an intracranial pressure of 21 mm Hg was noted.

On the evening of the day after admission, a cerebral blood flow study showed no cerebral blood flow. She was pronounced brain dead.

In the interim, her mother had fled town and has not been found since.

An autopsy was done on September 20, 1999. This showed hemorrhage in the left optic nerve sheath and left retinal hemorrhages as well as marked cerebral edema and thin widespread subdural hemorrhage. Diffuse axon injury was demonstrated with amyloid precursor protein antibody.

My point is that the child did have some symptoms, but clearly the severe intracranial injury symptoms, which were confirmed on repeat computed tomography and autopsy, were delayed for several hours, during which time she was under our view and review in the hospital. Others have noted similar problems (2).

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Author's Response

To the Editor:

In response to Dr. Robert Huntington's letter in which he expresses concern about the Position Paper's position on interval from injury to symptoms in young children with abusive head injuries, I would make the following comments. Dr. Huntington describes a child who is noted to be "irritable, sleepy, and vomiting" on admission. The Position Paper states, "Symptoms demonstrated by these severely injured children include an immediate decrease in the level of consciousness (either lethargy or unconsciousness)." Lethargy is defined as a condition of drowsiness or indifference *(Dorland's Illustrated Medical Dictionary)*. The sleepiness in this child is the neurologic symptom that marks the time at which the injury to this child occurred. It indicates a decrease in the level of consciousness. After the child was in the hospital several hours, she showed signs of increased intracranial pressure (right dilated pupil and sluggish left pupil) and went on to brain death. Dr. Huntington remarks that the severe intracranial injury symptoms were delayed for several hours. The symptoms to which he is referring were not the initial symptoms of injury but those related to increased intracranial pressure. The brain injury that precipitated this course of events (diffuse axonal injury) occurred when the child first became neurologically symptomatic. Brain swelling followed the diffuse axonal injury and resulted in increasing intracranial pressure and eventually brain death. Certainly, the child's neurologic symptoms changed with time, reflecting this changing neuropathology. It is the initial neurologic change that marks the time of injury.

The article by Gilliland that Dr. Huntington mentions is not helpful in elucidating the interval from injury to symptoms. The cases reported in that article relied on histories from possibly biased witnesses (caregivers who

might have injured a child) and took at face value the time intervals provided in each case. Dr. Gilliland noted this problem in her article, stating, "It should be noted that in all of the cases where information was supplied by someone other than the perpetrator, the child was not normal during the interval."

The claim that a young child has been fine for hours after a fatal head injury was inflicted and then suddenly developed symptoms is a claim that has no support from legitimate or mainstream medicine. When a child has suffered a serious acceleration injury to the brain that will result in long-term neurologic impairment or cause death, the so-called lucid interval is a fiction. The change from "fine" to "not fine" may be lethargy or it may be unresponsiveness, but it is a neurologic change, and it occurs at the time of injury.

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Deaths from Falls in Children: How Far is Fatal?

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The outcome of injury was determined in 317 children who were brought to a children's trauma center with a history from the caretaker that the child had fallen. Cases in which the clinicians' judgment was that an incorrect history had been given were included along with cases in which the history was not questioned. Seven deaths occurred in 100 children who fell 4 feet or less. One death occurred in 117 children who fell 10 feet to 45 feet. The 7 children who died in short falls all had other factors in their cases which suggested false histories. When children incur fatal injuries in falls of less than 4 feet, the history is incorrect. Long falls with an outdoor component are likely to be reliable data points for studies of children's injuribility.

Precise and certain differentiation of nonintentional injuries from inflicted injuries is extremely important in the management of injured children with histories discrepant from the injuries they show. Errors in diagnosis may be very costly. If inflicted injury cannot be proven to exist when it is present, children may be left in the care of persons who may injure them again, sometimes fatally. If the diagnosis is made incorrectly children may be removed from the care of their parents when that is not needed, and, conceivably, innocent persons could be convicted of crimes. Diagnosing child abuse medically is analogous to diagnosing cancer in that the risks of overlooking the problem may be fatal and the interventions available after the diagnosis is made are likely to be invasive and hazardous.

Whether a child's fatal injury is abusive often rests solely on the medical determination that the injury effects could or could not have been produced by the event described in the case history. While a number of studies[1-6] provide knowledge that can be applied to this determination, there is no comprehensive database on children's injuribility which supports highly reliable diagnosis, and the existing literature conflicts on the question of whether or not infants and children can receive fatal injuries in short falls.

The present study examines fatal outcome of injury in 317 infants and children who were seen at a children's trauma center with injuries and a history of having fallen. The purpose of the study was to determine the relationship of historical fall height to mortality, to assess the reliability of historical fall height, and to determine what types of fall histories might be used in establishing a database for children's injuribility.

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METHODS

The complete medical records of all children who were admitted to the Trauma Center at Children's Hospital-San Diego between August 1984 and March 1988 and for whom a mechanism of injury of "fall" had been recorded were reviewed to determine the fall height, the surface on which the child fell, the nature of the fall (whether free or interrupted), the person observing the fall, the diagnosis, and the outcome. The records included the prehospital care notes by first professional responders, emergency department notes, and hospital records from admission to discharge for the injury admission episode. In 23 of the cases the clinical staff had determined that the history given was unlikely to be true.

RESULTS

Table 1 displays the age and gender distribution of the 317 cases. The predominance of male toddlers and preschool children in this set of children who fell conforms to a pattern seen in many previous fall studies.

Table 2 displays the heights of the falls recorded for 283 cases. The remaining records provided no estimate of the height of the fall.

Table 3 displays the frequency of fatal outcome by the

TABLE 1
Gender and age of all children

	Number	Percentage
Gender		
Male	199	62.8
Female	118	37.2
Total	317	
Age (years)		
>1	30	9.5
1-3	145	45.7
4-6	61	19.2
7-12	65	20.5
13+	16	5.0
Total	317	

TABLE 2
Estimated fall heights

Fall Height (feet)	Number of Cases	Percentage of Total
1–4	100	35.3
5–9	65	23.0
10–14	75	26.5
15–19	24	8.5
20–29	17	6.0
30–45	2	0.7
Total	283	

TABLE 3
Case fatality rate by fall height

Fall Height (feet)	Number Died	Total	Case Fatality Rate (%)
1–4	7	100	7.0
5–9	0	65	0.0
10–45	1	118	0.8
Total	8	283	2.8

TABLE 4
Seven fatal cases with short falls; Type of fall

Standing Fall	2
Fall from Bed or Table	2
Fall Down Stairs	1
Fall in Arms of Adult	2

height of fall found in the history. Seven of 100 children died whose caretakers gave a history of a fall of 0 to 4 feet. No deaths occurred in 65 children who fell from 5 to 9 feet, and one death occurred in 118 children who fell from 10 to 45 feet. The fall that produced this single death in the children with "long" falls was not observed. The parents stated that their first knowledge that the 11-month-old child had fallen was when a neighbor brought the child to their door saying he had found the child beneath an open second story window. No detailed report of a scene investigation could be found for this case.

All of the children who died had head injuries as a cause of death consisting of subdural hematoma (mostly thin), cerebral contusion with brain swelling, or both, except for the child who presumably fell from the second story. This child had a markedly depressed parietal skull fracture with underlying bleeding, but survived his head injury and died 6 weeks later from sepsis complicating very severe and prolonged adult respiratory distress syndrome.

The types of falls given in the histories for the 7 children who died following short falls are shown in Table 4.

In the case with a history of a stairway fall the 11-month-old child was found unconscious at the foot of the stairs by a babysitter, but no one claimed to have witnessed the fall. This case was classified as a short fall because of the sitter's statement and because falls down stairs are believed to be series of short free falls.[7] In addition to a massive head injury this infant had small round bruises on both arms and bruising on the labia majora and one inner thigh. Two patients had histories of falling in the arms of an adult. In one the father stated that he fell against a crib while holding the 6-week-old infant 6 days before bringing her for care unconscious with agonal respirations. This infant had interhemispheric bleeding without a skull fracture and had conjunctival hemorrhages, bruising of the scalp, bruising on one ear, and retinal hemorrhages. In the other fall with an adult, the sitter stated that she had fallen on the child while going up stairs. The 13-month-old child had thin frontal and occipital subdural hematomas without a skull fracture, retinal hemorrhages, and an older healing tibial fracture. The head injury pathologic findings found in the seven cases by autopsy and clinical studies are shown in Table 5.

Although all seven patients had fresh subdural bleeding and cerebral edema, only one had a skull fracture.

Five of the seven fatal cases with short fall histories had "associated injuries" including old fractures, bruising on the trunk and extremities, genital injury, or more than one impact site on the head.

DISCUSSION

Diagnosis of classical "battered children" who are presented for care with multiple injuries in differing stages of healing is relatively simple for experienced physicians.[8,9] It is more difficult to be certain about children with a single "discrepant injury." Inflicted injury is often diagnosed when the clinician can state with a high level of certainty that the single injury seen in a child could not possibly have been produced by the event described by the caretaker. For the most part this discrimination is based upon the physician's clinical experience of children's "injuribility" and the limited empirical studies in the literature. Wissow and Wilson[10] stated the need for

TABLE 5
Seven fatal cases with short falls; head injury findings

Skull Fracture	1
Subdural Hematoma	7
Subarachnoid Blood	5
Cerebral Edema	7
Retinal Hemorrhage	5

TABLE 6
Seven fatal cases with short falls; associated injuries

Old Fractures	2
Bruises on Trunk or Extremities	3
Genital Injury	2
Two Head Impact Sites	2
No Associated Injury	2

a database of known accidents of various sorts which provide knowledge of children's injuribility. They suggested that the National Electronic Injury Study (NEISS) of the Consumer Product Safety Commission (CPSC) would be useful for this purpose. Chadwick[11] pointed out that the NEISS database led Sweeney[12] to conclude that children may die in falls as short as 1 foot, and that it may be seriously contaminated with inflicted injuries that are not screened out in the data collection process.

Hall et al.[2] reviewed records in the Medical Examiner's Office of Cook County, Illinois and found 18 cases in which a history of a fall of 3 feet or less was associated with fatal head injury. Their work omits the detailed information necessary to exclude inflicted injury, and many of those cases might be in that category.

Helfer's[3] classical study of children who fell while in the hospital provides a very useful and reliable set of events and effects and was recently replicated by Nimityongskul.[4] These two studies record the injuries noted in about 180 total small children who fell while in hospitals. The falls were all in the 3–4-foot range and the children had very minor injuries or were uninjured. The studies of Smith et al.[5] and of Barlow et al.[1] of long free falls of children from buildings found that the shortest falls that resulted in death were from the four-story level (or perhaps 30 feet). Snyder et al.[6] studied 100 falls of children and adults with personal scene investigation by the senior author and found one death in an apparent 10-foot fall of a child which was unobserved, but otherwise found that life-threatening injury required at least a 15-foot fall.

The data in the present study show an astonishing concentration of risk of death in the group with the shortest falls. Only 1 of 118 children died who were reported to have fallen from 10 to 45 feet. 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 1 to 4 feet than for those who fall from 10 to 45 feet. Since this conclusion appears absurd, it is necessary to seek another explanation for the observed relationship.

In children whose injuries are inflicted, parents typically invent accident histories which they hope will be accepted by health care providers. Since most falls of over 10 feet usually require that the fall occur outdoors (from a window, balcony, or other such location), caretakers may not wish to risk the possibility that a history could be proven false by a neighbor or passerby. It is also very possible that many lay persons believe that short falls may be fatal for children and are surprised to encounter skepticism. The best explanation of the findings is that for the seven children who died following short falls the history was falsified.

The low case fatality rate in the 118 children who fell 10 to 45 feet is in keeping with the observations of Smith et al.[5] and of Barlow et al.[1] but not with the conclusions of Hall et al.[2] Since the current study was done in an area with a county-wide trauma system that provides for quick and sophisticated first responses and designated hospitals for all trauma cases, a low case fatality rate for children's falls would be expected. However, delay in care is a common feature in inflicted injuries to children, and was probably a factor in all seven children who died with histories of short falls. The delay in care eliminates much of the advantage provided by an organized trauma system.

Many of the records lacked details that would be useful in determining impact sites and impact energy in any very precise way. Hospital records typically lack this sort of detail, which can only be captured if skilled investigators examine fall scenes and interview all witnesses fairly soon after the event.

CONCLUSIONS

Falls of less than 4 feet are often reported in association with children's head injuries that prove to be fatal, but such histories are inaccurate in all or most such cases. Long falls outside of buildings are more likely to provide accurate data points for studies of children's injuribility, and research on children's injuribility should utilize these longer falls rather than short indoor falls witnessed by just one person.

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Retinal hemorrhages caused by accidental household trauma

Cindy W. Christian, MD, Alexandra A. Taylor, MD, Richard W. Hertle, MD, and Ann-Christine Duhaime, MD

Traumatic retinal hemorrhages in young children are considered pathognomonic of child abuse. We identified 3 children with unilateral retinal hemorrhages caused by accidental household trauma. The hemorrhages were ipsilateral to intracranial hemorrhage and isolated to the posterior retinal pole. (J Pediatr 1999;135:125-7)

Traumatic retinal hemorrhages in infants and young children are considered by some to be pathognomonic of child abuse.[1,2] Retinal hemorrhages have been reported with severe accidental injury but have not generally been associated with minor or moderate head trauma.[1-3] The recognition that retinal hemorrhage can rarely result from accidental household injuries may influence decisions regarding suspected child abuse. We present 3 cases of unintentional household trauma (one of which has been previously reported)[4] that resulted in head injury and retinal hemorrhages.

Case Reports

Case 1

A 13-month-old boy fell down 13 concrete basement stairs in a walker; the fall resulted in loss of consciousness and irregular respiration. On arrival to the emergency department, he was awake and intermittently irritable, with a Glasgow Coma Scale score of 15. Initial blood pressure was elevated (145/114 mm Hg) but normalized within minutes. His head circumference was at the 90th percentile for age. Physical examination showed a small frontal scalp contusion, nasal abrasions, and a right thigh bruise; a neurologic examination revealed no focal findings. Head computed tomography scan revealed an acute subdural hemorrhage in an enlarged extra-axial space in the right parieto-temporal region, with minor mass effect (Fig 1). A skeletal survey revealed no acute or healing bony injuries, and no coagulopathy was present. A social work evaluation by a hospital trauma specialist revealed no risk factors for or concerns about child abuse. Clinically, the child remained well except for intermittent irritability and occasional vomiting. A second head CT scan 48 hours later showed resolution of the brain hemorrhage and a normal-appearing brain. He was discharged home after 4 days.

Initial ophthalmic evaluation revealed unilateral, pre-retinal, and intraretinal hemorrhage and localized retinal edema in the posterior pole of the right eye. There were no other ophthalmic abnormalities. The child had normal findings on subsequent ophthalmic examinations 3, 11, and 24 months after discharge.

CT	Computed tomography
GCS	Glasgow Coma Scale

Case 2

A 9-month-old boy was being supported on the forearms of his father and swung in play, when his father lost his grip. The child fell 1 to 2 feet, hitting the back of his head on the floor. The event was witnessed and described by friends visiting with the family at the time. Immediately after the fall, the child cried, then became transiently unresponsive with abnormal arm and leg movements. At the local emergency department he was noted to have extension of the arms and a right lateral gaze. Initial blood pressure was 118/97 mm Hg, which normalized quickly. His initial GCS score was 4, and an endotracheal tube was placed. CT scan showed acute subdural and/or subarachnoid hemorrhage over the left parieto-occipital region with a minimal midline shift. He was transported to The Children's Hospital of Philadelphia for further care.

On admission to our hospital, the infant had spontaneous eye opening and extremity movement, and he grimaced

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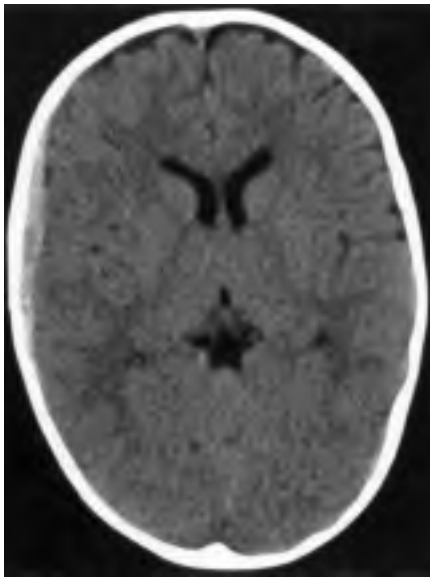

Fig 1. Case 1. Non-contrast CT scan shows an acute subdural hemorrhage in right parieto-temporal region, with minimal mass effect. Note also enlarged extra-axial cerebrospinal fluid space.

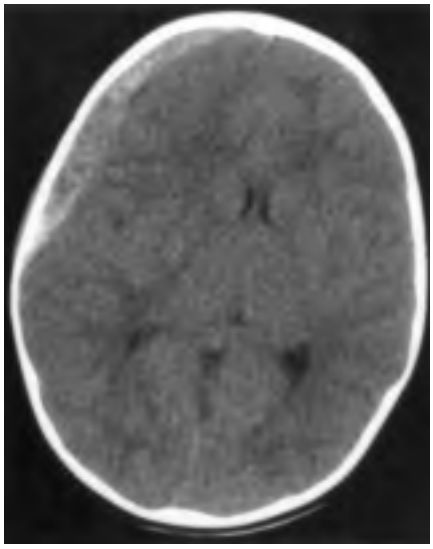

Fig 2. Case 3. Non-contrast CT scan demonstrates a large acute right fronto-parietal subdural hematoma associated with midline shift.

in response to noxious stimuli. No bruises or soft tissue swellings were noted. The endotracheal tube was removed, and administration of anticonvulsants was begun. The initial movements and extensor tone were thought to most likely represent seizure activity. Over the next 3 hospital days, the child experienced no further seizure activity, had no focal neurologic deficits, and had resolution of mild irritability and hypoactivity. A skeletal survey revealed no bony injuries, and follow-up head CT scan done the day after admission showed resolution of the hemorrhage and a normal-appearing brain.[4] No coagulopathy was found. A hospital social work evaluation and a child abuse team evaluation did not find risk factors for child abuse. In addition, a neighbor who witnessed the injury was called and verified the mechanism that was described.

Initial ophthalmic examination revealed multiple intraretinal flame-shaped hemorrhages and round intraretinal hemorrhages in the posterior pole of the left eye. The left macula showed foveal edema without exudates. In addition, there were 2 small, posteriorly located vitreous hemorrhages arising from the superficial retinal vasculature along the posterior pole arcade. Findings on examination of the right eye were normal. The infant was discharged home after 3 days and was found to be developmentally and neurologically normal at 3- and 6-month follow-up visits.

Case 3

A 7-month-old girl fell through a stair rail onto a concrete basement floor, landing on her head. There was no loss of consciousness. The child was brought directly to the local emergency department, where she was somnolent but arousable with a GCS score of 7. Initial blood pressure was 123/76 mm Hg, which normalized quickly. Her fontanelle was bulging and her right pupil was sluggish in response to light. An endotracheal tube was placed, and she was transferred to Children's Hospital for further care. Head CT scan showed a large, right parieto-frontal subdural hemorrhage with a midline shift (Fig 2).

On arrival, the baby was taken to the operating room for evacuation of the hematoma by means of a right frontoparietal craniotomy. A diastatic fracture of the coronal suture, a linear fracture of the temporal bone, and a right cerebral contusion were noted at surgery. The patient did well postoperatively, regaining normal neurologic function by the second hospital day. Findings on a skeletal survey were normal except for evidence of the previous craniotomy. Evaluation by the hospital trauma social worker did not reveal concerns about non-accidental injury. Initial prothrombin time (14.3 seconds) and partial thromboplastin time (39.7 seconds) were mildly elevated but were corrected without therapy.

By the third hospital day, the baby had right periorbital edema, mild eyelid erythema, and mild eye discharge. Ophthalmic examination at that time revealed unilateral abnormalities alone, consisting of orbital inflammation (edema, cellulitis) and sub-retinal and pre-retinal hemorrhages in the posterior pole of the right eye. The baby received antibiotics to treat possible preseptal cellulitis. The patient was discharged home on the fourth hospital day and had normal findings on neurologic examinations 1 week and 1 month after discharge and normal findings on ophthalmic examinations 3 weeks and 6 months after discharge.

In follow-up averaging 4 years, no reports of physical abuse have been made to child and youth agencies in the counties in which these children reside.

DISCUSSION

Retinal hemorrhages in young children have almost become diagnostic of child abuse. Although most traumatic hemorrhages after birth are related to abuse, the differential diagnosis of retinal hemorrhages is extensive, and qualitative and quantitative differences in hemorrhages from different causes exist.

Retinal hemorrhages seen in cases of child battering are often intraretinal, frequently involving all layers of the retina. The hemorrhages are often bilateral but can be unilateral.[5] Although they can be isolated to the posterior

pole, diffuse hemorrhages to the periphery of the retina are characteristic. In the most severe cases, vitreous hemorrhage, traumatic retinoschisis, perimacular retinal folds, and retinal detachment occur.[6-8] A correlation has been demonstrated between the severity of retinal and cerebral injuries in non-accidental trauma.[9,10]

Retinal hemorrhages after accidental trauma have been reported only occasionally.[2,3,11] These hemorrhages likely result from injury mechanisms similar to those described for intentional injury. Differentiating hemorrhages caused by accidental injury from those caused by abuse may be difficult, because either cause can result in unilateral or bilateral hemorrhages and can involve all layers of the retina.

Our patients had accidental injuries that resulted in subdural and/or subarachnoid hemorrhage and unilateral retinal hemorrhage. In each case the hemorrhages were ipsilateral to the intracranial hemorrhage and were isolated to the posterior retinal pole. Retinal involvement was relatively mild, without peripheral retinal involvement, retinal folds, or detachment. We recognize that differentiating abusive from accidental trauma can be difficult; however, several factors suggest that these cases were accidental. In each case the initial history provided was of significant trauma and did not change over time. Individuals not related to the child witnessed one injury. The neurologic symptoms and rapid recoveries were concordant with the forces described, there was no delay in seeking medical treatment, there was no prior reported history of abuse, and the remaining results of physical examination and skeletal survey were negative. To our knowledge, none of the children have been subsequent victims of physical abuse. Our conclusion of accidental trauma in case 1 is supported by previous reports of vitreous and retinal hemorrhages associated with walker injuries.[11] It is possible but unlikely that mild, transient hypertension and coagulopathy contributed to the retinal hemorrhage. Further research regarding the biomechanical pathophysiology of retinal hemorrhages is needed.

We report these cases to highlight the fact that rare cases of household accidental trauma can result in retinal bleeding. The incidence of retinal hemorrhages caused by household trauma is unknown; ophthalmic examinations in which mydriatics are used are not routine in children with head injury. However, during the 4 years in which these 3 cases were identified, 1617 children with head injury were admitted to our hospital. Although we acknowledge the strong association of traumatic retinal hemorrhages and child abuse, we caution against presuming that all children with traumatic retinal hemorrhages have been abused. Finally, because retinal hemorrhages can affect future vision, we recommend that all infants and children with significant head trauma, especially those with subdural and/or subarachnoid hemorrhage, have an ophthalmic examination.

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Evidence-Based Medicine and Shaken Baby Syndrome

Part I: Literature Review, 1966–1998

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In recent years, there has been a clear move toward basing medical practice and opinions on the best available medical and scientific evidence. This process has been termed *evidence-based medicine* (EBM) and involves a review of the quality of evidence that is available in various diseases and fields of inquiry within medicine.

This is the first of 2 articles that attempts to formally rank the available medical scientific evidence by internationally accepted methods, to determine the degree of confidence that can be held on various claims about the condition termed *shaken baby syndrome* (SBS). Areas with good scientific evidence are identified, and shortcomings in the research and publications on the subject are addressed.

Approximately half of all indexed medical publications on the subjects of SBS and shaken-impact syndrome were published before 1999 and half since that time. Given that 1998/1999 is regarded as the turning point in acceptance of the tenets and practice of EBM, it seemed reasonable to assess the quality of evidence before 1999 and compare it with the quality of evidence on the same subject matter since that time. At the conclusion of Part II, the 2 periods are compared to determine the extent to which EBM has affected the field of SBS in terms of quality of available evidence.

The aim of this review is to be neutral on the subject of SBS. Neutrality is difficult to define in this field, in part because of the polarization of opinions on the highly emotional subject of infant injury and death and in part because of clear data deficiencies arising from difficulties in performing experiments. It is clearly unethical to intentionally shake infants to induce trauma, and there is an obvious problem with studies and reports that rely on either indirect or disputed evidence of the occurrence, severity, or type of trauma.

Many studies lacking these critical data make the obvious logical error of selecting cases by the presence of the very clinical findings and test results they seek to validate as diagnostic. Not surprisingly, such studies tend to find their own case selection criteria pathognomonic of SBS.

Neutrality in this review simply means that there is no selective quotation of the available literature, and literature is not chosen to support any particular view. The assessment is of the methods and quality of the actual research, and until this assessment is complete, the content, findings, and recommendations are irrelevant. At the end of the ranking, those studies that achieve the highest QER scores are reviewed for their content, findings, and recommendations. Their outcomes are collated, and the entire published data set is then reviewed as a whole to determine the summarized recommendations, noting areas of agreement, conflict, or controversy. From this, the problems with the published evidence are noted, and data gaps are identified. Recommendations can then be made according to the summarized data.

In assessment of the quality of the available scientific evidence, the author has taken an approach recently defined worldwide as an appropriate scale for review of quality of evidence. This approach has been described recently in context of setting Australian clinical guidelines.

Genuine hypothesis testing requires use of appropriate research methodologies, including collection of relevant control data, and suitable statistical analysis. The interpretation of individual study findings may be constrained by factors such as whether the cohort examined was adequately representative of the patient population in general. Replication across studies and in independent research centers is a key factor in the reliability of evidence.

Compelling evidence comes from consistent findings in 2 or more well-constructed, controlled trials or population-based epidemiologic studies (i.e., level I or level II evidence). By contrast, clinical practice guidelines with level IV evidence represent consensus statements of the expert panel according to clinical experience and limited scientific data. Although these statements may influence current practice,

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they are likely to be modified in response to further research findings. Data from a single case series without control subjects provide little more than a stimulus for subsequent hypothesis testing.

Quality of Evidence Ratings

I: Consistent evidence obtained from more than 2 independent, randomized, and controlled studies or from 2 independent, population-based epidemiologic studies. Studies included here are characterized by sufficient statistical power, rigorous methodologies, and inclusion of representative patient samples. Meta-analysis of smaller, well-characterized studies may support key findings.

II: Consistent evidence from 2 randomized controlled studies from independent centers, a single multicenter randomized controlled study, or a population-based epidemiologic study. Data included here have sufficient statistical power, rigorous methodologies, and the inclusion of representative patient samples.

III-1: Consistent evidence obtained from 2 or more well-designed and controlled studies performed by a single research group.

III-2: Consistent evidence obtained from more than 1 study but in which such studies have methodologic constraints, such as limited statistical power, or the inclusion of patient samples that may be nonrepresentative.

III-3: Evidence obtained from a single case study or a selected cohort study.

III-4: Conflicting evidence obtained from 2 or more well-designed and controlled studies.

IV: Consensus opinions of authorities according to clinical experience or descriptive reports.

SBS: Literature Review (1966–1998)

Overview and Methods

The entire Biomednet Medline database (http://www.biomednet.com/db/medline) was searched by using the search term *shaken baby syndrome* and Internet Explorer in late November 1998. Other articles identified that had not yet been indexed on MEDLINE but had been published were also included.

The entire set of retrieved articles was reviewed, and those in which SBS was only peripherally mentioned or in which SBS was unrelated to the original article were omitted. Letters and brief correspondence were also discarded, unless they added new information or data on SBS. Articles in non-English journals that lacked an English abstract were also generally excluded from assessment.

These exclusions reduced the initial list of 71 articles to 54, which were reviewed, categorized, and ranked according to the QER above. To these was added the important study by Jayawant et al. from *BMJ* of December 5, 1998. The editorial was omitted because it added nothing to the original article.

It was impossible to review the full original article in many cases, although all of the major articles were reviewed in full. The remainder was assessed for categorization by using the authors' abstracts.

Each article was assigned to 1 of 4 categories: (1) randomized controlled trial; (2) case series with or without controls (with date, series size recorded); (3) single case reports; and (4) other, including review articles, opinion pieces, and articles on social implications.

Results of Quality of Evidence Ratings

Fifty-four articles or abstracts were reviewed. One was a randomized controlled trial.[34] This trial was not relevant to the general topic of SBS because it assessed a diagnostic technique (electroretinograph) that proved unsuccessful in diagnosis. Twenty-six were case series.[1,2,7,9,11,13–15,18,19,26,28–30,33,36,39,42,45–50,52,53] Twenty-five were retrospective studies, and 1 was prospective.

In total, 307 SBS cases were claimed to have been assessed among the 23 articles in which numbers of SBS patients were provided, with a mean study size of 13 cases and a median of 7 cases per series.

Selection criteria for SBS cases were unstated in 12 articles, based on presumption or suspicion in 10, and confirmed in 4 by confession or conviction. Two studies had appropriate control groups, 3 had inappropriate control groups, and 21 were case series without control groups. Twelve studies were case reports:[3–5,8,10,17,22,24,31,32,51,55]

Retinal pathology in suspected SBS, 5 cases

Blunt head injury at autopsy, 1

Subdural hemorrhage (SDH) and retinal hemorrhage (RH) as a result of fall and chest compression, 1 case

Shaking causing traumatic aneurism, 1 case

Arteriovenous malformation, not SBS, as cause, 1 case

Intentional asphyxia and shaking of 15-week-old baby, 1 case

Magnetic resonance imaging value in diagnosis, 1 case

Raised intracranial pressure as cause of RH, 1 case

Fifteen were "other" articles[6,12,16,20,21,23,25,27,37,38,40,41,43,44,54]

Historical reviews of SBS,10 articles

Opinion papers without original material, 3 articles

Social issues and SBS, 1 article

Review of imaging in SBS (computed tomography versus magnetic resonance imaging), 1 article

RESULTS

The randomized controlled trial was unrelated in any aspect of interest in this review and addressed a method of assessment of the retinae that proved to be unsuccessful.

Of the case series, the flaws noted above are relevant. All but 1 was retrospective, and all but 5 had no control population to compare cases with. Three of those that did

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include controls chose insufficient or inappropriate controls (head impact trauma, without healthy controls or other illness unrelated to head injury). This shortcoming would normally have excluded these studies from the literature review (because they do not fulfill criteria for inclusion). Given the difficulties inherent in assessment of SBS and of identifying appropriate control groups, however, these articles have been included as QER III-2.

In studies with confirmed (i.e., admitted or observed) trauma and SBS, there were few common findings, apart from the presence of SDH accompanied by RH in 80% of examined cases. Some articles attempt to measure other risk factor, and the study by Jayawant et al.[42] is noteworthy in this respect.

Finally, the "other" articles do little but summarize opinions and summarize past data. Such articles do not add to the quality of understanding of the condition, nor are they necessarily accurate in what has become a rather emotionally charged area of research and polarized opinion.

In this article, the quality of evidence, rather than the predominance of findings, is being assessed. The issue of the evidence for SBS appears analogous to an inverted pyramid, with a small database (most of it poor-quality original research, retrospective in nature, and without appropriate control groups) spreading to a broad body of somewhat divergent opinions. One may need reminding that repeated opinions based on poor-quality data cannot improve the quality of evidence.

Data Gaps Identified

There exist major data gaps in the medical literature about SBS. There is a very obvious lack of clear definition of cases. For valid studies, some method of determining cases of actual proven shaking must be found, and appropriate control groups (trauma without shaking, other illness, healthy controls) must be defined and assessed blindly. This gold standard has yet to be achieved in even a single study in the field of SBS. There is a lack of useful and specific laboratory or other markers proven to identify SBS. There is poor definition and quantification of the social and family risk factors to provide guidance on likelihood of abuse for a given set of circumstances. Last, there is a strong need for a checklist or other diagnostic or management tool to assess cases and to quantify index of suspicion of shaking.

CONCLUSIONS

There was no evidence on the subject of SBS that exceeded QER III-2 by the end of 1998, which means that there was inadequate scientific evidence to come to a firm conclusion on most aspects of causation, diagnosis, treatment, or any other matters pertaining to SBS.

The majority of evidence achieved only a level of QER IV, opinions that shed no new light upon SBS and did not add to knowledge about SBS. Many of the authors repeated the logical flaw that if RH and SDH are nearly always seen in SBS, the presence of RH and SDH "prove" that a baby was shaken intentionally. Many other studies assumed that the presence of RH and SDH was sufficient to make the diagnosis of SBS in terms of case selection.

The remainder of articles are QER III-3, and as noted above, the inclusion of case series without controls would normally not occur. Thus, the data available in the medical literature by the end of 1998 were inadequate to support *any* standard case definitions, or *any* standards for diagnostic assessment.

Before 1999, there existed serious data gaps, flaws of logic, inconsistency of case definition, and a serious lack of tests capable of discriminating NAI cases from natural injuries. By 1999, there was an urgent need for properly controlled, prospective trials into SBS, using a variety of controls. Without published and replicated studies of that type, the commonly held opinion that the finding of SDH and RH in an infant was strong evidence of SBS was unsustainable, at least from the medical literature.

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The shaken baby syndrome

A clinical, pathological, and biomechanical study

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✓ Because a history of shaking is often lacking in the so-called "shaken baby syndrome," diagnosis is usually based on a constellation of clinical and radiographic findings. Forty-eight cases of infants and young children with this diagnosis seen between 1978 and 1985 at the Children's Hospital of Philadelphia were reviewed. All patients had a presenting history thought to be suspicious for child abuse, and either retinal hemorrhages with subdural or subarachnoid hemorrhages or a computerized tomography scan showing subdural or subarachnoid hemorrhages with interhemispheric blood. The physical examination and presence of associated trauma were analyzed; autopsy findings for the 13 fatalities were reviewed. All fatal cases had signs of blunt impact to the head, although in more than half of them these findings were noted only at autopsy. All deaths were associated with uncontrollably increased intracranial pressure.

Models of 1-month-old infants with various neck and skull parameters were instrumented with accelerometers and shaken and impacted against padded or unpadded surfaces. Angular accelerations for shakes were smaller than those for impacts by a factor of 50. All shakes fell below injury thresholds established for subhuman primates scaled for the same brain mass, while impacts spanned concussion, subdural hematoma, and diffuse axonal injury ranges. It was concluded that severe head injuries commonly diagnosed as shaking injuries require impact to occur and that shaking alone in an otherwise normal baby is unlikely to cause the shaken baby syndrome.

KEY WORDS • shaken baby syndrome • head injury • child abuse

THE term "whiplash shaken baby syndrome" was coined by Caffey[3] to describe a clinicopathological entity occurring in infants characterized by retinal hemorrhages, subdural and/or subarachnoid hemorrhages, and minimal or absent signs of external trauma. Because a nursemaid admitted that she had held several such children by the arms or trunk and shaken them, the mechanism of injury was presumed to be a whiplash-type motion of the head, resulting in tearing of the bridging veins. Such an injury was believed to be frequently associated with fatalities in infantile child abuse and has been postulated as a cause of developmental delay in survivors.[4,15]

While the term "shaken baby syndrome" has become well entrenched in the literature of child abuse, it is characteristic of the syndrome that a history of shaking in such cases is usually lacking.[12] Shaking is often assumed, therefore, on the basis of a constellation of clinical findings and on the computerized tomography (CT) picture of subarachnoid and subdural hematomas, particularly in the posterior interhemispheric fissure.[17] Because of the ambiguous circumstances of such injuries, medicolegal questions are particularly troublesome, and the neurosurgeon is often consulted to give an opinion as to whether the findings are consistent with child abuse or accidental injury.

This paper reviews all cases of the shaken baby syndrome seen at the Children's Hospital of Philadelphia (CHOP) between January, 1978, and March, 1985. To better study the mechanism of injury, autopsy results in all fatal cases were reviewed, and the biomechanics of this injury were studied in a series of infant models. Based on these observations, we believe that shaking alone does not produce the shaken baby syndrome.

Clinical Studies

Clinical Material and Methods

All reports submitted to the Suspected Child Abuse and Neglect team were reviewed. Since house officers

TABLE 1

Initial clinical criteria for diagnosis of shaken baby syndrome

Diagnosis*	Cases		No. of Deaths
	No.	Percent	
retinal hemorrhage + SAH or SDH	29	60	5
retinal hemorrhage + SAH & SDH	10	21	5
bilateral chronic SDH	3	6	0
SAH &/or SDH & interhemispheric blood on CT	6	13	3
total	48	100	13

* SAH = subarachnoid hemorrhage; SDH = subdural hemorrhage; CT = computerized tomography.

TABLE 2

Best history in 48 cases of shaken baby syndrome

Etiology	Cases	
	No.	Percent
shaking only	1	2
fall or accidental blunt trauma	15	31
strike or fall plus shaking	10	21
strike only	3	6
trauma or shaking denied, caretakers in attendance	8	17
history unknown, caretakers not in attendance	10	21
cardiopulmonary resuscitation	1	2

TABLE 3

Trauma associated with shaken baby syndrome in 48 cases

Associated Trauma	Cases	
	No.	Percent
no evidence of blunt impact to head	18	37.5
no extracranial trauma	12	25.0
additional extracranial trauma	6	12.5
acute	3	6.25
old trauma only	3	6.25
evidence of blunt impact to head	30	62.5
skull fractures	12	25.0
cranial soft-tissue contusions	18	37.5
additional extracranial trauma	18	37.5
acute	15	31.25
old trauma only	3	6.25

and emergency room personnel are well trained in recognizing the clinical manifestations associated with this syndrome, it is considered that essentially all cases seen at CHOP are reported to this group.

Suspicion of shaking was based on history, clinical findings, and CT data. All subjects met the following criteria: presence of retinal hemorrhages with subdural and/or subarachnoid hemorrhages, bilateral chronic subdural hematomas, or a CT scan showing subdural or subarachnoid hemorrhages with interhemispheric blood. In addition, all patients were judged to have histories suggestive of child abuse or neglect; well-documented, witnessed accidental trauma was excluded. Histories were obtained from several interviews with caretakers by physicians, social workers, and in some cases law enforcement agents. Caretakers were routinely asked specifically about shaking.

Associated trauma data were obtained from physical examination, skull radiographs, CT scans, and skeletal surveys. All fatal cases were examined by the Philadelphia Medical Examiner, and pathology data were obtained from that office.

Results

Fifty-seven patients with suspected shake injury were identified. Of these, detailed clinical information was available in 48 cases. These patients ranged in age from 1 month to 2 years (mean 7.85 months). Thirty-one patients were male (65%). There were 13 fatalities (27%). Initial clinical criteria for diagnosis of the shaken baby syndrome are listed in Table 1. Thirty-nine patients (81%) had retinal hemorrhages plus subarachnoid and/or subdural hemorrhages. The remainder had bilateral chronic subdural hematomas (6%) or the above-mentioned CT findings without retinal hemorrhages (13%).

The most common presenting complaints were lethargy, breathing difficulty, irritability, poor feeding, and seizures. Best history is listed in Table 2; the most common histories were accidental blunt trauma (usually a fall) in 15 (31%) and blunt trauma plus shaking in 10 (21%); trauma and shaking were denied in eight (17%). In three cases (6%) the child was struck by the caretaker. In eight additional cases the history was unknown, usually because the child was left alone or with a babysitter. There were two cases (4%) with no history to explain the present findings, but both children were known to have been abused previously or subsequently. One case was associated with cardiopulmonary resuscitation (2%). In only one case was a history of shaking alone obtained; this child was reportedly shaken when she appeared to have difficulty in breathing associated with a respiratory infection.

Associated trauma observed clinically, radiographically, or at autopsy is listed in Table 3. The presence of scalp contusion, subgaleal or subperiosteal hemorrhage, and/or skull fracture was considered evidence of blunt impact to the head. Twelve cases (25%) had intracranial findings associated with the shaken baby syndrome alone, with no findings of associated blunt trauma to the head and no extracranial trauma. Six additional cases (13%) had the syndrome without signs of blunt head trauma but did have associated extracranial trauma. Thirty cases (63%) had findings of blunt impact to the head in addition to the intracranial findings of the shaken baby syndrome. Of these, 12 (25%) had skull fractures and 18 (38%) had significant cranial soft-tissue contusions. Most of the fractures were in the occipital or parieto-occipital region.

Clinical history, physical findings, hospital course, intracranial pressure (ICP, when measured), and pathological findings of the 13 fatalities are listed in Tables

TABLE 4

*Clinical and pathological findings in 13 fatal cases of shaken baby syndrome**

Factor	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8	Case 9	Case 10	Case 11	Case 12	Case 13
age (mos)	24	7	3	22	11	9	8	5	10	13	24	4	19
sex	F	M	M	M	F	F	F	M	F	M	M	M	F
history													
fall or hit	+		+	+	+			+	+	+	+	+	+
shaking			+								+		+
trauma denied													
unknown		+				+	+						
initial examination													
unresponsive	+	+	+	+	+	+	+	+	+	+	+	+	+
retinal hemorrhages	+	+		+			+	+	+	+	+	+	
cranial impact	+			+						+	+	+	+
extracranial trauma	+			+	+						+	+	
intracranial pressure	↑↑	NM	↑↑	↑↑	↑↑	NM	↑↑	NM	↑↑	NM	↑↑	↑↑	↑↑
survival time (days)	2	2	7	2	3	2	2	1	1	1	4	1	1
pathology													
cranial contusions	+	+	+	+	+	+	+	+	+	+	+	+	+
skull fracture(s)	++			++	++							+	+
subdural hematoma	+	+	+	+	+	+	+	+	+	+	+	+	+
subarachnoid hemorrhage	+	+	+	+	+	+	+	+	+	+	+	+	+
hemispheric contusions	+	+	+			+		+					+
white matter tears	+					+			+				+
diffuse brain swelling	+	+	+	+	+	+	+	+	+	+	+	+	+

* ↑↑ = increased; NM = not measured; + = factor present; ++ = severe.

4 and 5. Mean age in this group was 12.23 months; 54% were male. All of these children arrived at the hospital in an essentially unresponsive state, and all died from the effects of uncontrollably increased ICP associated with massive brain swelling. In only one case was a subdural hematoma thought to be of significant size to warrant surgical intervention, and drainage was ineffective in controlling elevated ICP.

Pathological examination showed that all of the children who died had evidence of blunt head trauma. Eight had soft-tissue contusions and five had contusions and skull fractures. In seven cases, however, impact findings were noted only at autopsy, and had not been apparent prior to death. All fatal cases had subdural and subarachnoid bleeding. Focal cerebral contusions and lacerations occurred in six. Microscopic examination was performed in three cases and showed corpus callosum hemorrhages, cortical laminar necrosis, or white matter hemorrhages. All children had diffuse and usually massive brain swelling.

Biomechanical Studies

Whole Infant Models

To test the hypothesis that infants are particularly susceptible to injury from shaking because of a relatively large head and weak neck, we constructed models of 1-month-old infants that were implanted with an accelerometer to measure the results of shaking or impact manipulations. Since the mechanical properties of the infant neck have not been studied, three models were built with different neck structures in order to include the range of limiting conditions that might exist in the live infant. Both a fixed center of rotation with zero resistance (hinge model) and moving centers of rotation with low and moderate resistance (rubber neck models) were tested.

Experimental Methods

The heads and bodies of the models were adapted from Just Born dolls. Head circumference was 36 cm, coronal width was 10 cm, anteroposterior diameter was 10.75 cm, and height from vertex to base (calculated from a line drawn from chin to caudal occiput) was 9.0 cm; values were comparable to human infants. Brain weight for an infant of this size was assumed to be 500 gm.[1] The ideal weight of the head was estimated by balance-weight measurements of several infants with an average age of 1 month, and was 770 to 870 gm. The heads of the models were tightly filled with cotton, with water added until the desired weight range was reached. The water was absorbed by the cotton and distributed so that no sloshing of the contents occurred. The heads were reweighed after neck insertion and sealing and at the end of all experiments.

Neck length from the skull base to the T-1 vertebra

TABLE 5

Summary of findings in 13 fatal cases of shaken baby syndrome

Factor	Finding
age (mos)	
mean	12.23
range	3–24
sex M/F	7/6
history	
fall or hit (three with shaking)	10
unknown	3
initial examination	
unresponsive	13
retinal hemorrhages	9
cranial impact	6
extracranial trauma	5
intracranial pressure	
measured, unable to control	9
not measured	4
survival time (days)	
range	1–7
mean	2.2
pathology	
cranial contusions	13
skull fractures(s)	5
subdural hematomas (one requiring surgery)	13
subarachnoid hemorrhage	13
unilateral	3
diffuse	3
multifocal	7
hemispheric contusions	6
diffuse, multiple	3
focal, coup-contrecoup	3
white matter tears	4
gross	2
microscopic	2
diffuse brain swelling (11 with herniation evident)	13

TABLE 6

Mean acceleration and time course of shakes and impacts in all models

Manipulation	No.	Peak Tangential Acceleration (G)	Time (msec)	Angular Velocity (radians/sec)	Angular Acceleration (radians/sec^2)
shakes	69	9.29	106.6	60.68	1138.54
impacts	60	428.18	20.9	548.63	52,475.70

TABLE 7

Effects of neck condition and "skull" on mean peak tangential acceleration and time course of shakes and impacts

Variant	Shakes		Impacts	
	Acceleration (G)	Time (msec)	Acceleration (G)	Time (msec)
hinge neck	13.85	92.7	423.42	18.6
flexible rubber neck	5.70	93.3	427.78	21.4
stiff rubber neck	7.02	130.5	433.33	22.8
skull	9.86	107.4	436.12	20.2
no skull	8.89	103.5	427.04	21.6

TABLE 8

Effect of impact surface on mean peak tangential acceleration and time course

Surface of Impact	Acceleration (G)	Time (msec)
padded surface	380.60	24.22
metal bar	489.51	17.13

was measured from lateral neck films of several normal infants with an average age of 1 month and ranged from 3.5 to 4.5 cm; all models were therefore given neck lengths of 4.0 cm. Necks were embedded in Castolite resin* superiorly, which was also used to seal the head. The interior part of the neck was secured in dental stone.† The stuffed body was then replaced around the dental stone "thorax," with lead weights added as necessary to the thorax to reach a total body weight of 3 to 4 kg. Arms and legs were not weighted, so the slightly low total weight for age reflects an attempt to approximate trunk:head weight ratios.

Model 1 had a hinge neck made from a 360° steel hinge, 3.6 cm in width, placed in the horizontal plane to allow complete anteroposterior angulation of the head. The center of rotation was 3.3 cm below the estimated level of the skull base (approximating at the C-6 vertebral level). Model 2 had a 1.9-cm diameter hollow rubber neck with a 0.8-cm lumen. This neck did not support the weight of the head in the upright position but did not kink when the head was allowed to fall unsupported. Model 3 had a 2.9-cm rubber neck with a 1.2-cm lumen. This neck was able to support the head in the vertical position but allowed full passive movement of the head. In all models, head motion was limited in the anteroposterior direction by the occiput striking the upper back and the chin striking the chest.

To test for the effect of the deformability of the model heads on impact, all models were tested with and without an external "pseudoskull" made from thermoplastic.‡ This "skull" was 1/8 in. thick and was molded to the occipital, parietal, temporal, and posterior frontal areas, with the facial area uncovered. The "skulls" weighed 170 to 200 gm.

Data were recorded from a piezoelectric accelerometer§ embedded in a small piece of thermoplastic and attached to the vertex in a coronal plane through the

* Resin manufactured by Buehler Ltd., Evanston, Illinois.

† Dental stone, Glastone Type IV, manufactured by Ransom and Randolph Co., Toledo, Ohio

‡ Polyform thermoplastic manufactured by Rolyan Medical Products, Menomonee Falls, Wisconsin.

§ Accelerometer manufactured by Endevco Corp., San Juan Capistrano, California.

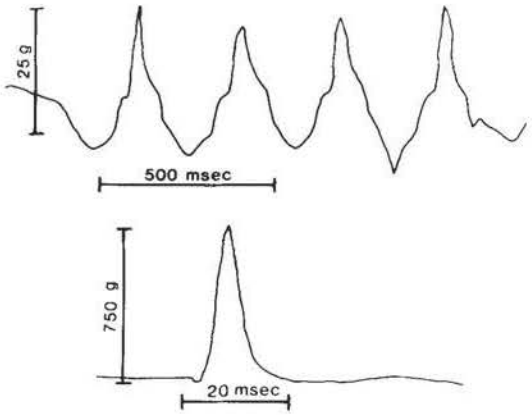

FIG. 1. Representative tangential acceleration traces for infant models undergoing shake *(upper)* and impact *(lower)* manipulations. While manipulations of the infant models were performed as described, with a series of shakes followed by an impact, the magnitude of the impact accelerations was so much greater than that associated with the shakes that different scales are used to display the respective acceleration traces.

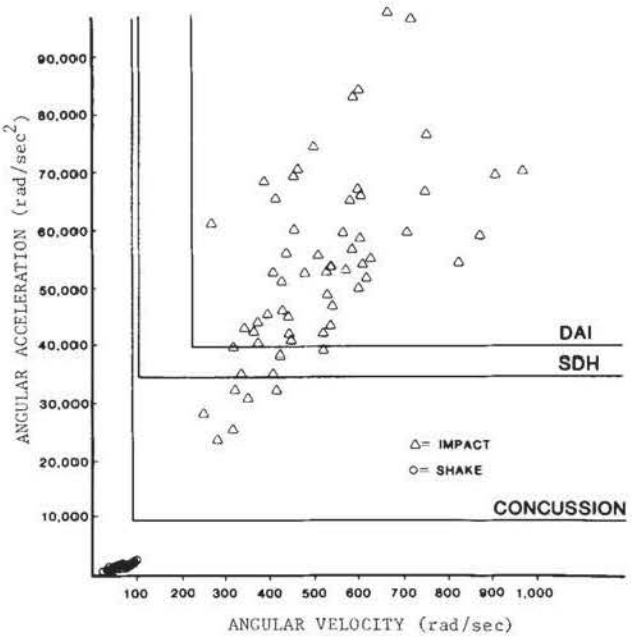

FIG. 2. Angular acceleration versus angular velocity for shakes and impacts, with injury thresholds from primate experiments scaled to 500-gm brain weight. DAI = diffuse axonal injury; SDH = subdural hematoma.

center of the neck. Each model was subjected to repetitive violent shaking, allowing the head to travel its full excursion several times, by adult male and female experimenters. The models were held by the thorax facing the experimenter and were shaken in the anteroposterior plane, since this is the motion most commonly described in the shaken baby syndrome. At the end of each series of shakes the occiput was impacted against either a metal bar or a padded surface. Each model was tested at least 20 times. Acceleration traces were amplified and recorded.‖

Angular accelerations were calculated from the measured peak tangential accelerations by using C-6 as the center of rotation in all cases. Angular velocity was calculated as the time integral of the acceleration curve. Translational forces were assumed to be minimal.

Results

The data were collected from 69 shaking episodes ("shakes") and 60 "impacts." Typical tangential acceleration traces for shake and impact manipulations are shown in Fig. 1. The criterion for significant difference was $p < 0.01$ in all cases.

Shakes Versus Impacts. Angular acceleration and angular velocity for each shake and impact are shown in Fig. 2. Mean peak tangential acceleration for 69 shaking episodes was 9.29 G; mean peak tangential acceleration for 60 impacts was 428.18 G (Table 6). The accelerations due to impact are significantly greater than those obtained by shaking ($p < 0.0001$); on the average, impact accelerations exceed shake accelerations by a factor of nearly 50 times. Mean time interval for shakes was 106.6 msec and for impacts was 20.9 msec. This difference is significant at the $p = 0.001$ level.

Effects of Neck Condition. Mean tangential accelerations and time courses for shakes and impacts for each neck condition are presented in Table 7. There is no significant difference between the hinge neck, the flexible rubber neck, and the stiff rubber neck in the mean acceleration resulting from impacts (423.4, 427.8, and 433.3 G, respectively) or in the mean time course (18.6, 21.4, and 22.8 msec, respectively). With shakes, the more flexible hinge neck is associated with higher accelerations (mean 13.85 G) than the two rubber neck models (mean 5.7 and 7.0 G) ($p < 0.001$). There is an inverse relationship between neck stiffness and time duration of a shake: the stiff rubber neck was associated with a longer time course than the more flexible rubber neck (130.5 msec and 93.3 msec, respectively) ($p < 0.001$).

Effects of "Skull." The presence of a hard thermoplastic "skull" did not change the magnitude or time course of accelerations associated with shaking of the models. The acceleration magnitude and time course were also unchanged when the models were impacted. These data are shown in Table 7.

Effects of Impact Surface. Impact against a padded surface was associated with significantly smaller acceleration (mean 380.6 G) and longer time course (mean 24.22 msec) than that against a metal bar (mean 489.5 G and 17.13 msec) ($p < 0.001$). Data are shown in Table 8.

‖ Shock amplifier, Model 2740 A, and pulse memory unit, Model 2743, manufactured by Endevco Corp., San Juan Capistrano, California.

Discussion

Clinical head injury can be classified into two major categories according to the distribution of pathological damage, whether focal or diffuse.[10] Such a distinction is important for treatment and prognosis, as well as for establishing the biomechanical conditions necessary to produce a given injury type. It has been established both experimentally and clinically that most focal injuries are associated with impact loading, resulting in contact phenomena, while diffuse injuries are associated with impulsive loading conditions resulting from acceleration-deceleration phenomena.[6] Damage to the brain occurs as a result of these biomechanical forces and from the secondary effects of ischemia due to altered autoregulation or brain swelling.

The shaken baby syndrome has been postulated to result from the effects of nonimpact acceleration-deceleration forces. It has been suggested that the back and forth movement of the head alone is sufficient to cause tearing of bridging veins, resultant subdural hematomas, and death.[8,13] The relatively large size of an infant's head, weakness of the neck musculature, softness of the skull, relatively large subarachnoid space, and high water content of the brain have been postulated to contribute to the susceptibility of shaking injuries in infants.[4,14]

While shaking alone has been considered sufficient to cause a fatal injury, the usual lack of history of the true mechanism of injury in these cases has hampered accurate clinicopathological correlations. It is of interest, however, that in a recent series of fatal cases of infantile head injuries from suspected child abuse,[5] white matter tears were found similar to those described by Lindenberg and Freytag[11] in blunt trauma in infancy. In addition, lesions in the distribution typical of diffuse axonal injury, like those found in adult head injury and in subhuman primates subjected to high acceleration-deceleration injury,[7] were described in some cases. In fact, at least one of Caffey's original cases[3] included "lacerations of the cerebral parenchyma." Shaking alone was the presumed mechanism of these injuries.

As experience has accumulated in experimental angular acceleration injury it has become clear that, besides the magnitude of the acceleration, another important biomechanical factor influencing injury type is the time interval over which the acceleration occurs. Thus, large angular accelerations occurring over shorter time periods tend to result in subdural hematoma, while longer intervals are associated with diffuse axonal injury.[6] A tolerance scale relating these two factors to resultant injury has been developed for the subhuman primate by Thibault and Gennarelli.[16] Values above certain critical limits result in a particular type of injury such as concussion, subdural hematoma, or diffuse axonal injury. When such a curve is scaled for the brain mass of an infant the size of our models, it can be seen that the angular acceleration and velocity associated with shaking occurs well below the injury range, while the values for impacts span concussion, subdural, and diffuse axonal injury ranges (Fig. 2). This was true for all neck conditions with and without skulls. A padded surface decreases the magnitude of acceleration and lengthens the time course to some extent, but these impacts also fall in the injury range.

These results are consistent with the observation that the fatal cases of the shaken baby syndrome in this series were all associated with evidence of blunt impact to the head. This preponderance of blunt trauma has also been found in at least one other series of nonaccidental head trauma in childhood in which the mechanism of injury was investigated.[9] It is of interest that in more than half of our fatal cases, no evidence of external trauma was noted on the initial physical examination, which helped to contribute to the diagnosis of "shaken baby syndrome." Skull fractures and scalp contusions were found at autopsy, however, most often in the occipital or parieto-occipital region. In addition, several babies had parenchymal lesions in a distribution consistent with diffuse axonal injury.[11]

While some reports on the shaken baby syndrome mention brain swelling, in most reports the subdural collections themselves have been postulated as the cause of death. In this series, all fatalities were consequent to uncontrollable brain swelling, and it is clear that drainage of the small collections present would have been useless in controlling the ICP. The problem of acute brain swelling is particularly common in the pediatric population, and its cause is poorly understood.[2] Whether high accelerations in the anteroposterior direction have some particular association to this complication remains to be investigated.

It is our conclusion that the shaken baby syndrome, at least in its most severe acute form, is not usually caused by shaking alone. Although shaking may, in fact, be a part of the process, it is more likely that such infants suffer blunt impact. The most common scenario may be a child who is shaken, then thrown into or against a crib or other surface, striking the back of the head and thus undergoing a large, brief deceleration. This child then has both types of injury — impact with its resulting focal damage, and severe acceleration-deceleration effects associated with impact causing shearing forces on the vessels and parenchyma. Unless a child has predisposing factors such as subdural hygromas, brain atrophy, or collagen-vascular disease, fatal cases of the shaken baby syndrome are not likely to occur from the shaking that occurs during play, feeding, or in a swing, or even from the more vigorous shaking given by a caretaker as a means of discipline.

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Ocular Autopsy and Histopathologic Features of Child Abuse

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Purpose: To study the ocular histopathologic features in eyes of children with fatal suspected child abuse.
Design: Retrospective case series.
Participants: One hundred eighteen autopsy cases of known or suspected child abuse.
Methods: The ocular autopsy and histopathologic features of a cohort of consecutive cases of known or presumed child abuse submitted by Maryland's Office of the Chief Medical Examiner or Johns Hopkins Hospital to the Wilmer Eye Pathology Laboratory were tabulated.
Main Outcome Measure: Ocular hemorrhage or structural abnormality.
Results: Retinal hemorrhage was present in 44% of cases. Circumferential folds with macular schisis cavities were present in 23% of cases and were bilateral in half of those cases. Peripapillary scleral hemorrhage was present in 38% of cases, and subdural hemorrhage was present in the distal optic nerve in 46% of cases. Hemosiderin was present in 27% of cases.
Conclusions: Intraretinal hemorrhages, circumferential macular folds with schisis cavities, peripapillary scleral hemorrhages, and subdural hemorrhages are common pathologic findings in cases of fatal known or suspected child abuse. Their presence on autopsy should raise the suspicion of shaking or blunt nonaccidental trauma. *Ophthalmology 2007;114:1384–1394 © 2007 by the American Academy of Ophthalmology.*

Since the first pathologic description of ocular involvement in nonaccidental trauma in 1964,[1] several reports have described various ocular features associated with child abuse. Ocular findings are clinically present in approximately 40% of known cases of child abuse[2,3] and approximately 70% of deaths due to child abuse.[4,5] Ocular findings are the presenting feature in 4% to 6% of cases of child abuse.[2,3] It is therefore important that all physicians recognize the ophthalmic features associated with child abuse.

Autopsy studies of ocular findings in child abuse have generally ranged from single case reports to small case series including 6 to 10 patients.[4–6] Two reports of larger series of autopsy cases include child deaths of all causes, including accidental trauma, nonaccidental trauma, and nontraumatic death.[7,8] Both of these reports present data concerning some ocular features of known or presumed child abuse, including retinal and optic nerve hemorrhages. A report of a systematic evaluation of all gross and histopathologic ocular manifestations of child abuse has not yet been published.

In this study, we characterize the gross and histopathologic features of child abuse and describe their relationship to systemic findings and mechanism, based on history.

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From Wilmer Eye Institute, Johns Hopkins Hospital, Baltimore, Maryland.

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Materials and Methods

This study was exempt from review by the Johns Hopkins Hospital institutional review board. The Wilmer Eye Pathology Laboratory's records were examined for autopsy specimens sent for evaluation of suspected nonaccidental trauma by Maryland's Office of the Chief Medical Examiner or Johns Hopkins Hospital. Cases examined were received over a period spanning 43 years. One case was excluded from this series because the findings were thought to be due to thrombocytopenia and no other ophthalmic signs of abuse were present.

Both eyes of each case were fixed in 10% formalin solution for at least 48 hours. After external examination of the eye and attached optic nerve, the nerve was transected. When the nerve was long, it was cut into 2 portions, proximal and distal. Eyes were opened in the horizontal plane. Data collected included presence of conjunctival hemorrhage, vitreous hemorrhage, retinal hemorrhage, circumferential macular fold, optic nerve head swelling, optic nerve sheath hemorrhage, and orbital hemorrhage. Retinal hemorrhage was graded based on number of hemorrhages: 0 (no hemorrhage), 1+ (1–5 hemorrhages), 2+ (6–10 hemorrhages), 3+ (10–20 hemorrhages), or 4+ (>20 hemorrhages) (Fig 1). Photographs of the external and internal findings were taken of each case.

Microscopic sections through the pupil–optic nerve–macula plane of each eye and cross sections of the distal and proximal portions of each optic nerve were prepared. In cases in which findings were present grossly in the superior or inferior calottes, additional microscopic sections were prepared of these areas. Each specimen was stained with hematoxylin–eosin, periodic acid–Schiff, Prussian blue (for iron), and phosphotungstic acid–hematoxylin (for fibrin). Histologic data collected concerning the presence of anterior segment abnormalities included conjunctival hemorrhage, hyphema, cataract, rubeosis irides, and iris and ciliary body hemorrhage. Posterior segment findings documented included vitreous hemorrhage, vitreous base avulsion, sub–internal limiting lamina hemorrhage,

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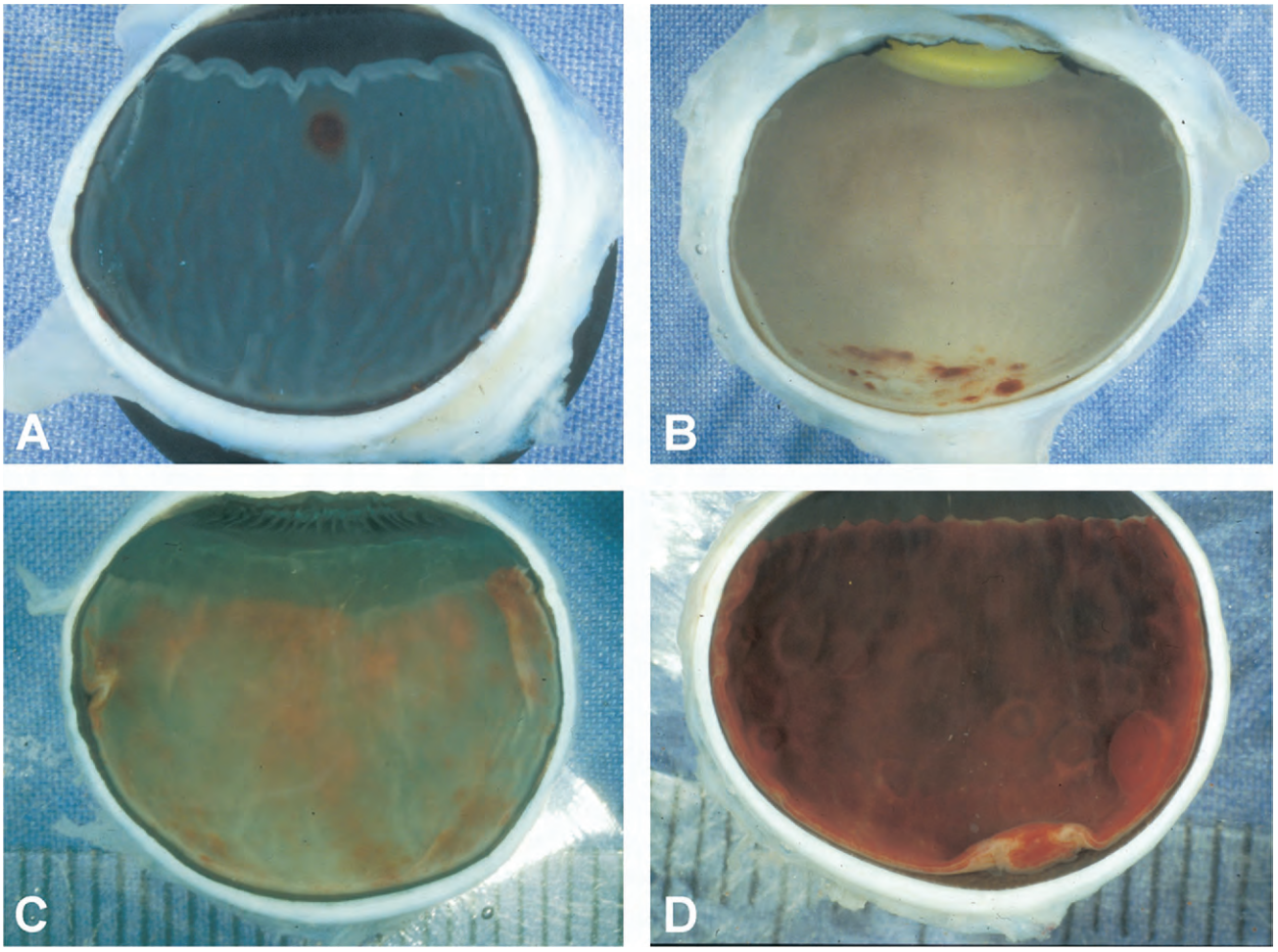

Figure 1. Grading of gross retinal hemorrhages. **A**, Grade 1+ (1–5 hemorrhages). **B**, Grade 2+ (6–10 hemorrhages). **C**, Grade 3+ (11–20 hemorrhages). **D**, Grade 4+ (>20 hemorrhages; confluent in this case).

retinal hemorrhage, retinal venous dilation, circumferential macular fold with schisis cavity, peripheral retinal fold with schisis cavity, subretinal hemorrhage, choroidal hemorrhage, and choroidal vascular congestion. Retinal hemorrhages were further categorized on the basis of retinal depth (nerve fiber layer and deeper), location (macula, midperiphery–periphery, and near the ora serrata), and extent: 0 (no hemorrhage), 1+ (single area of hemorrhage), 2+ (>1 hemorrhage but <25% of the retina involved), 3+ (hemorrhage involving ≥25% but <50% of the retina), 4+ (hemorrhage involving ≥50% but <100% of the retina), and 5+ (hemorrhage involving 100% of the retina). Data collected concerning the optic nerve included optic nerve head swelling, optic atrophy, subarachnoid hemorrhage, subdural hemorrhage, intradural hemorrhage, orbital hemorrhage, and peripapillary scleral hemorrhage. Subdural hemorrhage was graded based on amount of hemorrhage present: 0 (no hemorrhage), 1+ (few erythrocytes in the subdural space and no apparent alteration of dural contour), 2+ (additional erythrocytes with minimal distortion of dural architecture), 3+ (hemorrhage with moderate expansion of the subdural space), and 4+ (marked expansion of the subdural space with hemorrhage) (Fig 2). Peripapillary scleral hemorrhage was graded based on extent of hemorrhage: 0 (no hemorrhage), 1+ (few erythrocytes present in the sclera where the dura attaches), 2+ (erythrocytes present within the sclera, extending >200 μm from the dural attachment), and 3+ (intrascleral hemorrhage tracking from the site of dural attachment to the subchoroidal space) (Fig 3). Location and severity of hemosiderin were graded 0 (no iron), 1+ (mild presence of iron), 2+ (moderate presence of iron), or 3+ (marked presence of iron) (Fig 4). Phosphotungstic acid–hematoxylin–stained sections were examined for presence and location of fibrin. In cases in which special staining (Prussian blue and phosphotungstic acid–hematoxylin) was not done at the time of initial evaluation, the paraffin-embedded specimens were obtained and sections were subsequently performed and stained.

Details from history and autopsy findings were also tabulated in each case.

Results

The eyes of 118 cases of admitted or suspected child abuse were submitted to the Wilmer Eye Pathology Laboratory by Maryland's Office of the Chief Medical Examiner or Johns Hopkins Hospital for evaluation. In all cases, both eyes were available for study. All deaths were due to known or suspected abuse based on initial medical examiner's report or physician's suspicion. Fifty-three (44.9%) were female. Age at time of death ranged from 10 days to 9 years, 75 (63.5%) were younger than 1 year, and 99 (83.9%) were younger than 2. Four children were older than 3 at the time of death. Cardiopulmonary resuscitation was known to have been performed in 10 (8.5%) cases. Intracranial hemorrhage,

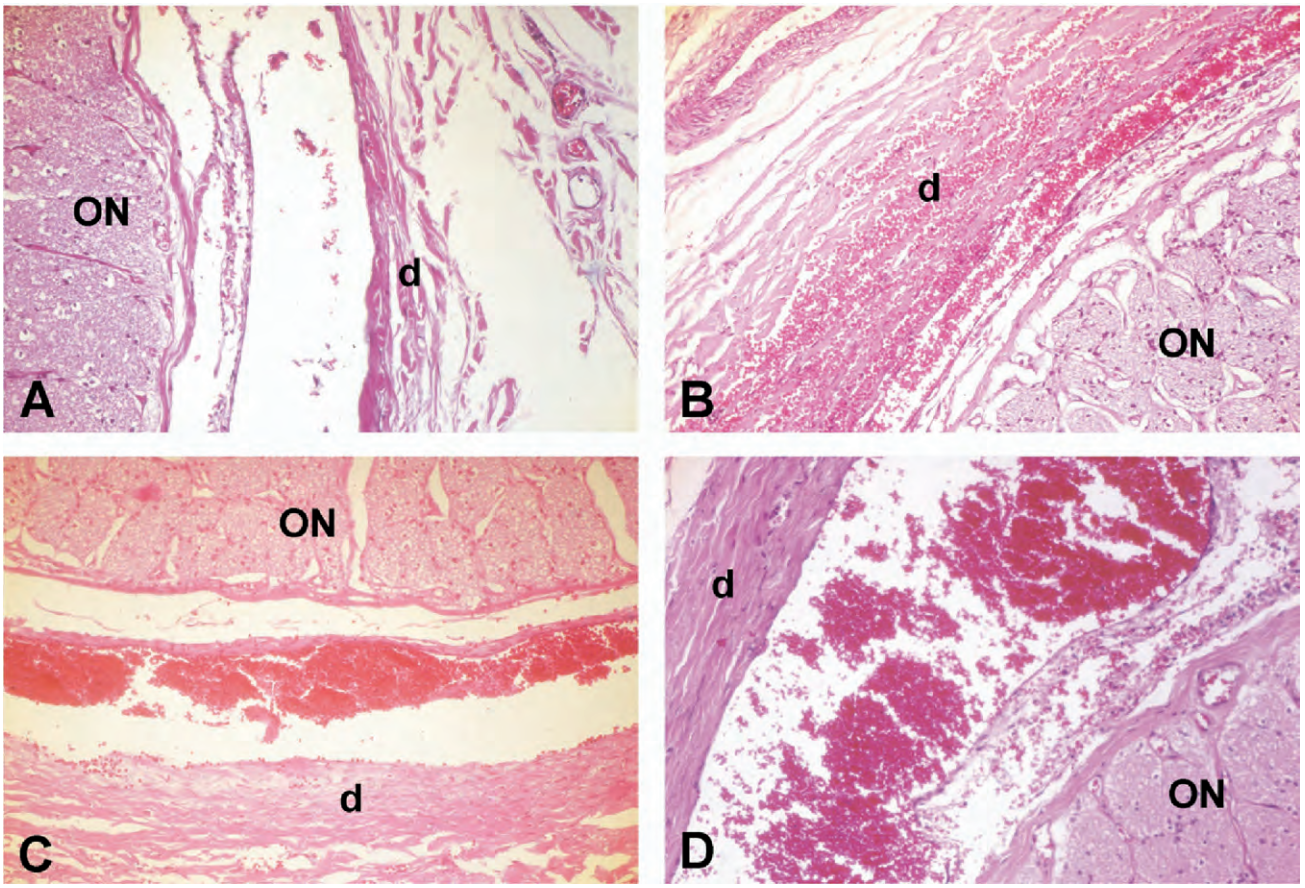

Figure 2. Grading of subdural hemorrhage in cross section of optic nerve (ON) (stain, hematoxylin–eosin; original magnification, ×210). **A**, Grade 1+ subdural hemorrhage. Few erythrocytes are present in the subdural space, between dura mater (d) and arachnoid mater. **B**, Grade 2+ subdural hemorrhage with separation of the dura from the arachnoid by erythrocytes. Hemorrhage is also present in the dura of this case. **C**, Grade 3+ subdural hemorrhage with moderate expansion of the subdural space by erythrocytes. **D**, Grade 4+ subdural hemorrhage and marked distortion of the normal contour of the dura–arachnoid interface.

including hemorrhage in the subdural and subarachnoid spaces, was present in 62 (52.5%) of cases. Autopsy signs of blunt head trauma, including scalp contusions, subgaleal hematomas, and skull fractures, were present in 48 (40.7%) cases. Healing fractures, a sign of prior trauma, were present in 19 (16.1%) cases. The perpetrator specifically admitted to shaking in 7 (5.9%) cases. Demographic, historical, and systemic nonocular autopsy features are based on preliminarily reported data and presented in Table 1.

Conjunctival hemorrhage was grossly present in 13 eyes (5.5%) of 10 (8.5%) cases (Table 2). Retinal hemorrhage was present in 99 (41.9%) eyes of 55 (46.6%) cases. Retinal hemorrhage in the more severely affected eye was graded as 4+ (>20 hemorrhages) in 34 of 54 (68.5%) cases in which any retinal hemorrhage was present. A circumferential macular fold was present in 37 (15.7%) eyes of 23 (19.5%) cases, and optic nerve sheath hemorrhage was present in 88 (37.3%) eyes of 47 (39.8%) cases.

On histologic examination, injury of the anterior segment was present in 15 eyes (6.4%) of 12 patients (8.1%) and consisted of hyphema (n = 11), cataract (n = 5), rubeosis irides (n = 1), corneal scar (n = 1), and ciliary body and iris parenchymal hemorrhage (n = 1). In 4 of the 5 eyes with cataract, hyphema was also present. In the eye with rubeosis irides, cataract and hyphema were also present.

Retinal hemorrhages were present in 97 (41.1%) eyes of 53 (44.9%) cases and were bilateral in 46 (86.8%) cases. Retinal hemorrhages were located in the nerve fiber layer in all except 3 involved eyes. Retinal hemorrhages were located deep to the nerve fiber layer in 79 eyes. Retinal hemorrhages were located more frequently in the posterior pole (94 eyes [39.8%]) than in the midperiphery (86 eyes [36.4%]) or at the vitreous base, near the ora serrata (67 eyes [28.4%]). In the 67 eyes in which retinal hemorrhage was present near the ora serrata, hemorrhage was also present posteriorly. Hemorrhage involved the entire retina of 22 (9.3%) eyes of 14 (11.9%) cases.

Circumferential macular folds with schisis cavities containing proteinaceous material with variable amounts of hemorrhage were present in 41 (17.4%) eyes of 27 (22.9%) cases, 14 (52%) of which were bilateral (Fig 5). In all cases, the vitreous remained attached to the internal limiting lamina and the schisis cavity was formed between the internal limiting lamina and remainder of the retina. Additionally, the vitreous remained attached to the retina peripheral to the macular fold. Material in the schisis cavity stained positively for fibrin in 18 (43.9%) of the 41 involved eyes.

Peripheral circumferential retinal folds were present in 29 (12.3%) eyes of 19 (16.1%) cases. Retinal venous dilation with or without intravascular thrombus was present in 49 (20.8%) eyes of

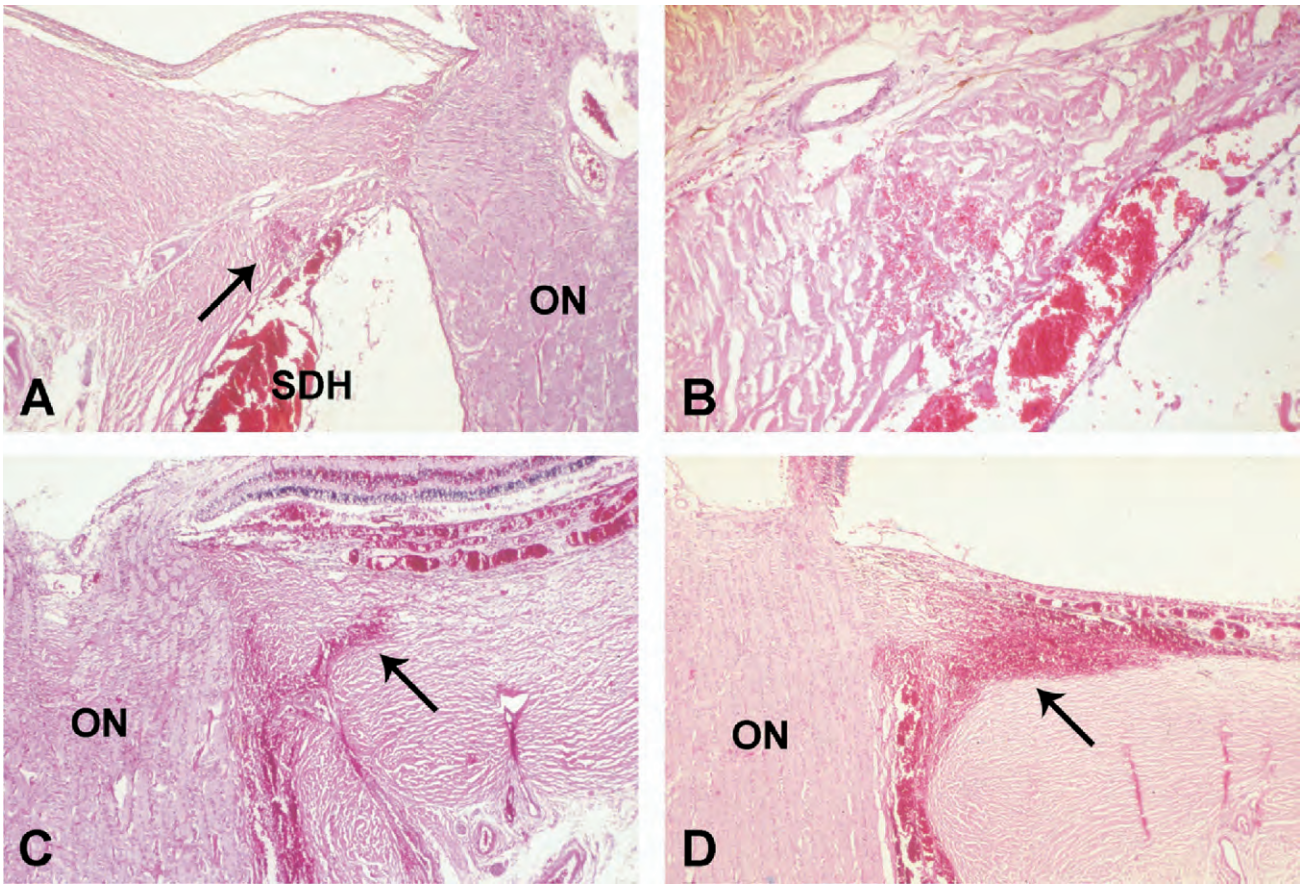

Figure 3. Grading of peripapillary scleral hemorrhage in longitudinal sections of optic nerve (ON) (stain, hematoxylin–eosin). **A**, Grade 1+ peripapillary intrascleral hemorrhage (arrow) and adjacent subdural hemorrhage (SDH) (original magnification, ×85). **B**, Higher power of scleral hemorrhage shown in A. The hemorrhage is located within 200 μm of the subdural space (original magnification, ×210). **C**, Grade 2+ peripapillary scleral hemorrhage (arrow). The hemorrhage extends farther than 200 μm from the attachment of the dura to the sclera, but not to the subchoroidal space (original magnification, ×85). **D**, Grade 3+ peripapillary scleral hemorrhage (arrow). Hemorrhage extends from the subdural space to the subchoroidal space (original magnification, ×85).

32 (27.1%) cases (Fig 6), and choroidal congestion was present in 25 (10.6%) eyes of 15 (12.7%) cases.

Optic nerve swelling was present in 38 (16.1%) eyes of 24 (20.3%) cases and was bilateral in 58% of the cases in which it was present. Optic nerve atrophy was present in 6 (2.5%) eyes of 4 (3.4%) cases. Trauma was known to have taken place 2 months previously in 1 unilateral case and 13 months previously in 1 bilateral case. The other bilateral case had a history of herpetic encephalopathy and cereberal palsy at birth, and death at age 2 years was attributed to abuse. The fourth case of optic atrophy had no history of trauma or medical abnormality and might have been related to unreported nonaccidental trauma.

Peripapillary scleral hemorrhage was present in 86 (36.4%) eyes of 45 (38.1%) cases. In cases in which hemorrhage was present, hemorrhage was graded 1+ (29%) or 2+ (44%) or extended from the attachment of the dura to the subchoroidal space (3+ [27%]) (Fig 3). Peripapillary scleral hemorrhage was associated with a tear in the sclera in 57 (66%) eyes.

Subdural hemorrhage was present in the proximal portion of the optic nerve in 99 (41.9%) eyes of 54 (45.8%) cases and, distally, in 84 (35.6%) eyes of 47 (39.8%) cases. Proximal subdural hemorrhage was bilateral in 87% of cases in which it was present. In 12 eyes of 8 cases, the optic nerve of the submitted specimen was short, precluding evaluation of the distal optic nerve. More cases were graded 4+ proximally (23.7%) than distally (12.7%).

Hemosiderin was present in 64 (27.1%) eyes of 35 (29.7%) cases, including 5 eyes of 4 patients with no concurrent ocular hemorrhagic injuries. Optic atrophy was present in all 5 eyes. A history of abuse was present in each case, and the abuse had occurred 14 months before death in 1 case. Hemosiderin was graded as 1+ in 20 (16.9%), 2+ in 7 (5.9%), and 3+ in 8 (6.8%) cases (Fig 4). Hemosiderin was located in the optic nerve of 46 (19.5%) eyes, the retina of 28 (11.9%), and the choroid of 6 (2.5%).

Discussion

Intraocular hemorrhages have been reported to occur in 81% of children who are known to have been shaken and 94% of children hospitalized with subdural hemorrhage who have no external signs of head trauma and are presumed to have been shaken.[9] Reported rates of clinically apparent retinal hemorrhages in abused children span a wide range (39%,[2] 40%,[3] 67%,[10] 85%,[11] and 89%[12]), which may be largely due to inclusion criteria in each particular study.

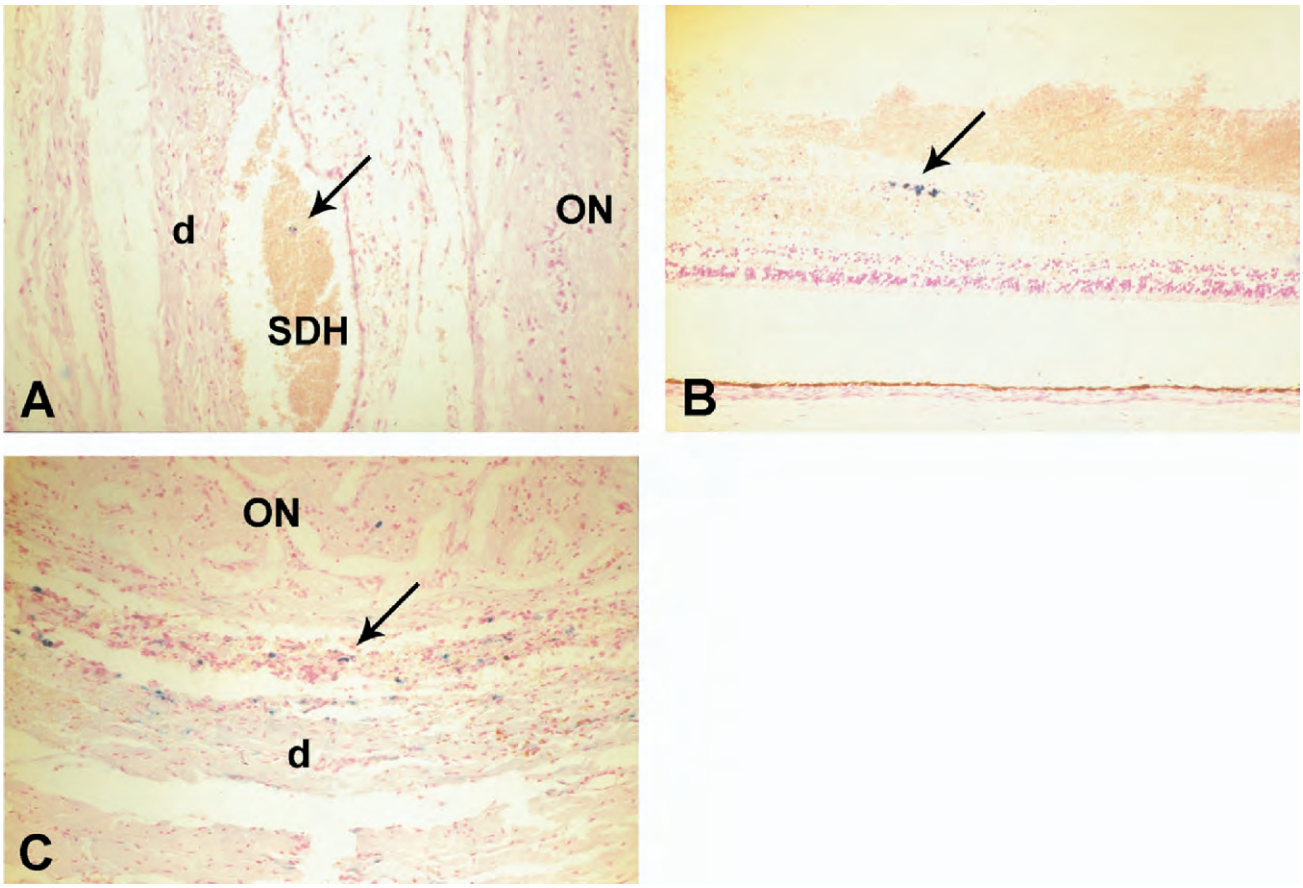

Figure 4. Grading of hemosiderin deposition (stain, Prussian blue). **A**, Longitudinal section of optic nerve (ON) with grade 1+ hemosiderin (arrow) within an area of subdural hemorrhage (SDH) (original magnification, ×85). d = dura mater. **B**, Retina with grade 2+ hemosiderin (arrow) and hemorrhage in the inner aspect of the retina (original magnification, ×210). **C**, Cross section of ON with hemorrhage and grade 3+ hemosiderin (arrow) in the dura mater, subdural space, and arachnoid mater (original magnification, ×210).

Severity of retinal hemorrhage has been correlated with acute neurologic findings.[13] In a prospective study of children younger than 3 years hospitalized for nonaccidental traumatic subdural hemorrhage, retinal hemorrhages were found in 91% of the patients who later died of their injuries as opposed to 60% of those who survived.[12] This difference in rates of ocular findings between survivors and fatalities of child abuse may be a reflection of the increased severity of trauma in children who later die from abuse relative to those whose injuries are not severe enough to result in death. Although clinical studies such as this provide valuable information, only a few studies have reported ocular histopathologic findings in child abuse victims.[4–8,14] The collective data from these case series are presented in Table 3.

Table 1. Demographics, History, and Nonocular Features of the Study Population

No. of cases	118
Female	53 (44.9%)
Median age (mos)	5.0
Mean age (± SD) (mos)	11.7±15.1
Race	
White	37 (31.4%)
Black	74 (62.7%)
Hispanic	4 (3.4%)
Asian	1 (0.8%)
Mixed	1 (0.8%)
Other	1 (0.8%)
Known history of cardiopulmonary resuscitation	10 (8.5%)
Known history of shaking	7 (5.9%)
Intracranial hemorrhage (SDH, SAH)	62 (52.5%)
External head trauma (scalp contusion, subgaleal hemorrhage, skull fracture)	48 (40.7%)
Trauma to torso or abdomen	45 (38.1%)
Healing fracture	19 (16.1%)

SAH = subarachnoid hemorrhage; SD = standard deviation; SDH = subdural hemorrhage.

The mechanism of retinal hemorrhage formation in child abuse has been the subject of great speculation and little agreement. In their series of 6 autopsy cases of child abuse, Marshall et al found cerebral edema, intracranial hemorrhage, and retinal hemorrhages present in all cases.[6] They concluded that retinal hemorrhages are caused by venous obstruction from increased intracranial pressure due to cerebral edema, subdural hemorrhage, and subarachnoid hemorrhage. Of the 53 retinal hemorrhage cases in our series, either intracranial hemorrhage or optic nerve subdural hemorrhage was present in all except 2. These findings lend support to the theory that retinal hemorrhage is linked to submeningeal hemorrhage, but they do not establish causality.

Marshall et al further surmise that intravascular venous obstruction may be exacerbated by compression of the

Table 2. Ophthalmic Features of the Study Population

	No. of Affected Eyes	% of Affected Eyes	No. of Affected Cases	% of Affected Cases
Gross features				
Conjunctival hemorrhage	13	5.5	9	7.6
Vitreous hemorrhage	42	17.8	26	22.6
Retinal hemorrhage	99	41.9	55	46.6
Circumferential macular fold	37	15.7	23	19.5
Optic nerve head edema	17	7.2	10	8.5
Subdural hemorrhage	88	37.3	47	39.8
Orbital hemorrhage	32	13.6	18	15.3
Histologic features				
Conjunctival hemorrhage	11	4.7	8	6.8
Anterior segment abnormality	14	5.9	11	9.3
Vitreous hemorrhage	81	34.3	47	39.8
Sub–internal limiting lamina hemorrhage	64	27.1	38	32.2
Intraretinal hemorrhage	97	41.1	53	44.9
Retinal venous dilation	49	20.8	32	27.1
Circumferential macular fold	41	17.4	27	22.9
Peripheral circumferential retinal fold	29	12.3	19	16.1
Subretinal hemorrhage	61	25.8	37	31.4
Choroidal hemorrhage	12	5.1	8	6.8
Choroidal congestion	25	10.6	15	12.7
Vitreous base avulsion	5	2.1	4	3.4
Optic nerve atrophy	6	2.5	4	3.4
Optic nerve edema	38	16.1	24	20.3
Peripapillary scleral hemorrhage	86	36.4	45	38.1
Subarachnoid hemorrhage	73	30.9	42	35.6
Subdural hemorrhage—proximal	99	41.9	54	45.8
Subdural hemorrhage—distal	84	35.6	47	39.8
Dural hemorrhage	87	36.9	46	39.0
Orbital hemorrhage	76	32.2	45	38.1
Presence of fibrin	35	14.8	23	19.5
Presence of hemosiderin	64	27.1	35	29.7

abdomen and thorax in those cases in which the infant is grasped to be shaken.[6] In our series, there was no apparent difference in the frequency of retinal hemorrhages in cases with and without autopsy evidence of trauma to the torso. This type of grasping compression may not be severe enough to result in thoracic injury detectable by autopsy.

This manner of sustained chest compression likely differs from the rapid succession of chest compressions in cardiopulmonary resuscitation, which has not been found to be causally linked to retinal hemorrhages in an animal model[15] or human infants in clinical studies, in the absence of coagulopathy or preceding trauma related to the cardiac arrest.[16,17] Immature vessels in retinopathy of prematurity may be more fragile than those of full-term infants and may be more susceptible to cardiopulmonary resuscitation forces, resulting in retinal hemorrhages.[18]

The generalized elevated intracranial pressure or venous stasis theories of retinal hemorrhage would explain bilateral and symmetric retinal hemorrhages, which characterize 86.8% of our cases with retinal hemorrhage. However, other factors might play a role in cases of unilateral retinal hemorrhages or markedly asymmetric retinal hemorrhages.

Rao et al proposed that concussion injury occurs when the retina hits the scleral wall as a result of waves of force generated by blunt facial trauma transmitted along the facial bones and orbital soft tissues.[5] They proposed that transmitted force waves cause disruption of blood vessels, resulting in retinal hemorrhage. This mechanism may explain the unilaterality of some cases of retinal hemorrhages; however, no study yet has reported an association between laterality of head injury and pattern of ocular involvement.

One study reports evidence of acceleration–deceleration forces with or without impact in 50 of their 70 cases with retinal hemorrhages.[7] The authors conclude that the preponderance of retinal hemorrhages present near the ora serrata and posteriorly in the macula supports the theory that vitreomacular traction causes shearing forces in these areas where the vitreous is more firmly attached to the retina. In our series, retinal hemorrhage near the ora serrata was present in fewer cases than hemorrhage in the midperiphery, and we therefore cannot support this aspect of the vitreomacular traction theory as the mechanism of retinal hemorrhage formation.

Vitreoretinal traction has also been the proposed mechanism of circumferential macular fold formation whereby tight vitreoretinal adhesions in the area surrounding the macula have been purported to pull the retina into a circumferential fold with violent shaking.[6] This theory is advocated by multiple authors, who report a total of 7 cases of a circumferential macular fold with schisis cavity formation in infants without external signs of blunt head trauma.[19–21] If circumferential macular folds were due to tight vitreomacular adhesions and looser vitreomidperipheral retinal

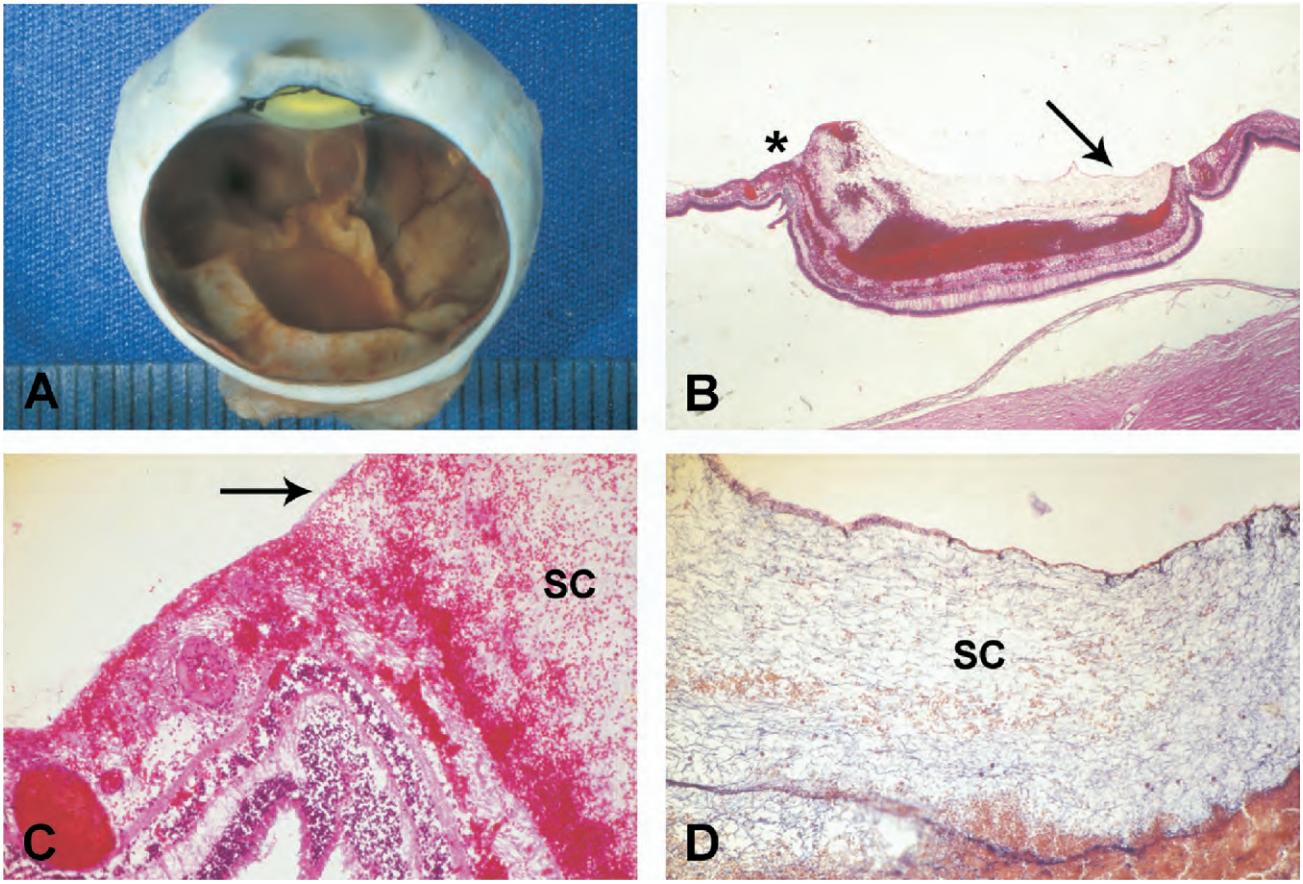

Figure 5

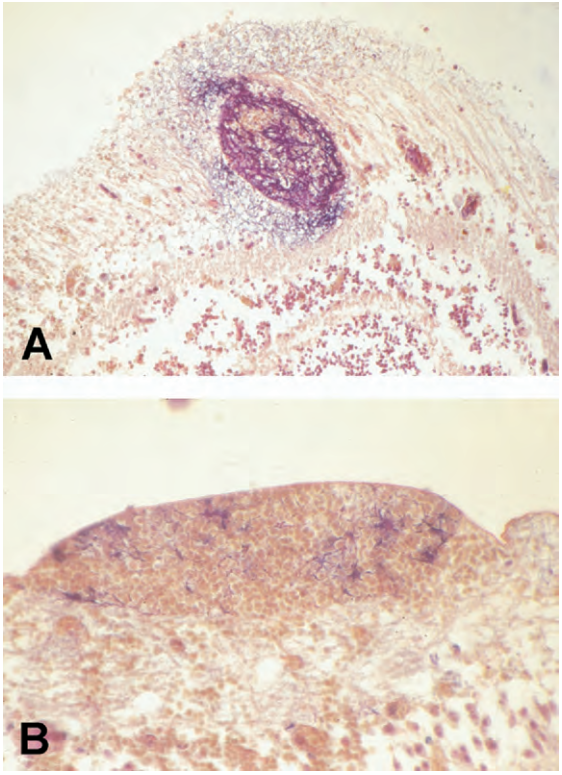

Figure 6

Table 3. Histopathologic Ocular Findings of Reported Child Abuse Autopsy Series

	Betz et al[14] (1996) (n = 7)	Elner et al[4] (1990) (n = 10)	Gilliland et al[7] (1994) (n = 169)*	Marshall et al[6] (2001) (n = 6)	Rao et al[5] (1988) (n = 14)	Riffenburgh and Sathyavagiswaran[8] (1991) (n = 98)	Present Study (n = 118)
Female	—	—	68 (40%)	0 (0.0%)	4 (28.6%)	45 (46%)	53 (44.9%)
Mean age (mos)	2–9 (range)	—	0–119 (range)	12.7	19.7	—	5.0 (median)
Intracranial hemorrhage	—	7 (70%)	—	6 (100%)	9 (64.2%)	61 (62%)	62 (52.5%)
Blunt head injury	5 (71.4%)	10 (100%)	—	5 (83.3%)	11 (78.6%)	18 (18%)	48 (40.7%)
Blunt injury to body	—	—	—	3 (50.0%)	10 (71.4%)	—	45 (38.1%)
Anterior segment findings	—	3 (30%)	—	—	1 (7.1%)	—	11 (9.3%)
Vitreous hemorrhage	4 (57.1%)	7 (70%)	—	—	1 (7.1%)	—	47 (39.8%)
Intraretinal hemorrhage	7 (100%)	7 (70%)	70 (41%)	6 (100%)	8 (57.1%)	51 (52%)	53 (44.9%)
Bilateral intraretinal hemorrhage	6 (85.7%)	—	—	—	5 (35.7%)	48 (49%)	46 (39.0%)
Choroidal hemorrhage		—	—	—	1 (7.1%)	—	8 (6.8%)
Circumferential macular fold/schisis cavity	—	3 (30%)	—	3 (50.0%)	2 (14.3%)	1 (1%)	27 (22.9%)
Optic nerve head edema	—	—	—	—	4 (28.6%)	—	24 (20.3%)
Optic nerve subdural hemorrhage	1 (14.3%)	7 (70%)	62 (37%)	6 (100%)	9 (64.2%)	47 (48%)	54 (45.8%)
Scleral hemorrhage	—	5 (50%)	48 (28%)	0 (0.0%)	—	—	45 (38.1%)
Hemosiderin	—	3 (30%)	—	—	1 (7.1%)	—	35 (29.7%)

*Includes cases of suspected abuse, accidental trauma, and nontraumatic death.

adhesions, vitreous detachment might be present in the midperiphery of some cases, especially in those in which macular folds are present. However, this was not observed in any of our 41 eyes with circumferential macular folds. Our data, therefore, do not support the theory of vitreomacular traction as the etiology of circumferential macular fold formation.

Although circumferential macular folds are most frequently observed in infants, they have been reported in 2 adults with Terson syndrome: a 19-year-old who suffered severe head trauma during a motor vehicle collision and a 26-year-old who sustained a gunshot wound to the forehead.[22] These cases suggest that shaking is not required for circumferential macular fold formation and that some other mechanism accounts for their formation. Furthermore, 2 cases of circumferential macular folds in the setting of crush injury in the absence of shaking have been reported: 1 of bilateral circumferential macular folds in a 14-month-old child attributed to accidental crush injury by a television falling on the child and 1 of a unilateral circumferential macular fold in a 4-month-old infant attributed to a 63-kg child falling on the infant.[23,24] These cases emphasize the possibility that shaking is not required for the formation of circumferential macular folds.

Riffenburgh and Sathyavagiswaran found several peripheral dome-shaped schisis cavities in various stages of development and concluded that they form in the presence of profuse and prolonged retinal hemorrhage.[8] However, they found a schisis cavity in the macula of only one eye of one patient in their cohort of 98 cases and base their conclusion on their evaluation of peripheral retinal folds. Other studies,[4–6] including ours, have found a higher frequency of circumferential macular folds. In our series, 40 of 41 eyes with circumferential macular folds also had optic nerve subdural hemorrhage. Intracranial hemorrhage was also present in all but 1 case. Peripapillary scleral hemorrhage was present in all eyes with a circumferential macular fold. These associations may relate to the mechanism of each individual finding or may indicate that multiple types of trauma occurred in each of these patients.

We submit that the leakage from retinal vessels and presence of fibrin in the proteinaceous material within the

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Figure 5. Circumferential macular fold with schisis cavity (SC). **A**, Gross appearance with a prominent circumferential fold with hemorrhage within the central schisis cavity. **B**, Microscopic cross section of a macular fold with hemorrhage and proteinaceous debris filling the cavity (stain, hematoxylin–eosin; original magnification, ×85). **C**, Higher power of area adjacent to the asterisk in B. The internal limiting lamina (arrow) is separated from the retina by hemorrhage in the SC (stain, hematoxylin–eosin; original magnification, ×210). **D**, Fibrin stain of area around the arrow in B at higher power. Purple-staining fibrin and few erythrocytes diffusely fill the schisis cavity (stain, phosphotungstic acid–hematoxylin; original magnification, ×210).

Figure 6. Retinal fibrin deposition (stain, phosphotungstic acid–hematoxylin). **A**, Branch retinal vein with organizing thrombus within the vein lumen and extravasated fibrin in the surrounding retina (original magnification, ×250). **B**, Microscopic section of a sub–internal limiting lamina schisis cavity with hemorrhage and fibrin (original magnification, ×340).

Table 4. Cases with a History

Subject	Age (mos)	History of Blunt Trauma	Intracranial Injury	External Injury	Vitreous Hemorrhage	Retinal Hemorrhage Grade
1	3.5	"Closed head injury"	SAH	None	−	2
2	16	None	SDH	Blunt injury to back and abdomen	+	3
3	8	"Blunt trauma to head"	SAH, cereberal edema	Skull fracture	+	5
4	2	None	SAH, intraventricular hemorrhage	None	−	0
5	2	"Blows with closed fist"	SAH, cereberal edema, epidural hemorrhage	Subgaleal hemorrhage, new/healing rib fractures, cheek contusions	+	5
6	2	None	SAH, SDH	Skull fracture	+	5
7	15.5	"Dropped in bathtub"	SDH, cereberal edema, cerebellar herniation	None	+	4

CRV = central retinal vein; PTAH = phosphotungstic acid–hematoxylin; SAH = subarachnoid hemorrhage; SDH = subdural hemorrhage.
+, present; −, absent.

schisis cavities of several macular folds in our series support a venous leakage mechanism for macular fold formation; the perpetrator's grasp about the chest increases thoracic pressure, which decreases venous return, causing retinal venous stasis, dilation, and incompetence with subsequent leakage of serous fluid and hemorrhage into the retinal tissue. Prolonged compression might be related to expanding hemorrhage and schisis cavity formation. We propose that the schisis cavity expands, pulling the surrounding retina centripetally into a circumferential fold.

Peripapillary scleral hemorrhages have been mentioned in 2 reports,[4,25] one of which is a case report of hemorrhage within the sclera surrounding the optic nerves of both eyes of a 6-month-old with subdural hematoma and evidence of blunt trauma to the head and body; however, this finding has not been well described. The proposed mechanism for the formation of these hemorrhages is acceleration–deceleration forces caused by head injuries that result in tears of the intrascleral vessels at the circle of Zinn. Our findings in 45 cases of peripapillary scleral hemorrhage included optic nerve subdural hemorrhage in 44 (98%) and evidence of blunt head trauma in 19 (42%, vs. 32% of cases without peripapillary scleral hemorrhage). This may support the theory that the acceleration–deceleration forces of violent shaking may cause enough rotational stress at the optic nerve–globe junction to cause peripapillary scleral hemorrhage and that blunt head trauma need not be involved. We therefore propose that these hemorrhages may be characteristic of shaking abuse.

Optic nerve sheath hemorrhage was first noted in a case of child abuse by Lambert et al.[26] Microscopic examination of the optic nerve disclosed hemorrhage in the subarachnoid and subdural spaces. They attribute these hemorrhages to sudden increases in intracranial pressure with rupture of bridging veins. They attribute the formation of retinal hemorrhage to further increases in intracranial pressure resulting in occlusion of the central retinal vein and its chorioretinal anastamoses. Duhaime et al performed biomechanical studies measuring angular acceleration in shaking and impact scenarios with dolls designed to replicate the features of infants.[27] They concluded that impact against either a soft or hard surface was necessary to produce the forces required to cause the intracranial (subdural or subarachnoid) hemorrhage associated with child abuse. In contrast, the forces associated with isolated shaking are not substantial enough to cause these hemorrhages. However, the authors do not explain how intracranial or retinal hemorrhage occurred in the 18 (37.5%) cases in their study in which no evidence of head trauma was present.

The presence of hemosiderin in autopsy tissues may indicate previous episodes of trauma or that the fatal injury occurred long enough before death for hemoglobin to be converted into hemosiderin.[28] Hemosiderin can be detected as early as 3 days after trauma but may not be apparent by histochemical staining until 5 to 7 days after trauma.[4] In our series, hemosiderin was present in 31 cases of concurrent hemorrhages and 5 eyes of 4 cases without concurrent hemorrhage. Previous trauma had resulted in optic atrophy in each of these 4 cases and had occurred 14 months before death in 1 case, with persistence of grade 3+ hemosiderin in both eyes. Thus, in each of our cases of hemosiderin positivity, some other sign of ocular trauma was also present.

Great focus has been placed on determining the effects of shaking trauma, because shaking is never accidental, whereas blunt or impact trauma can be accidental or nonaccidental. Table 4 summarizes data of the 7 cases in our series with a history of admitted shaking, based on initial reporting. Four of these patients also had a history of blunt trauma, but only 3 had autopsy evidence of trauma to the head (2 with skull fracture and 1 with subgaleal hemor-

of Admitted Shaking

Circumferential Macular Fold	Peripheral Retinal Fold	Peripapillary Scleral Hemorrhage Grade	Optic Nerve Subarachnoid Hemorrhage	Optic Nerve Subdural Hemorrhage Grade	Fibrin Location (PTAH)	Hemosiderin Location
−	−	0	−	0	CRV	−
−	+	2	+	3	−	−
+	+	3	+	4	Schisis cavity	−
−	−	0	−	0	−	−
+	+	2	+	4	Retinal vein, pial vessels, schisis cavity	Subdural space, arachnoid
+	−	3	+	4	CRV, pial vessels, schisis cavity	Subdural space, subarachnoid space
+	−	1	+	4	CRV, retina, schisis cavity	−

rhage). These 3 cases exhibited grade 5+ retinal hemorrhages, circumferential macular folds, peripheral circumferential folds (in 2 of 3 cases), and grade 4+ proximal optic nerve subdural hemorrhage, and 2 of the cases had grade 3+ peripapillary scleral hemorrhage (the third case had grade 2+ peripapillary scleral hemorrhage). One of the cases without external head trauma had a circumferential macular fold. Blunt injury to the abdomen in case 2 may have been related to grasping trauma with compression or to striking trauma and presence of the peripheral retinal folds.

In these cases of known shaking, the ocular involvement was severe. However, shaking need not always result in ocular pathology, as is demonstrated by case 4, which had a history of fatal shaking and no ocular findings. This case demonstrates that in child deaths involving suspicious circumstances, absence of ocular findings at autopsy does not exclude the possibility of nonaccidental trauma.

These 7 cases of known shaking also highlight how little is known about the ocular findings of child abuse. This lack of information stems primarily from incomplete and misleading information concerning the mechanism of injury inherent in the evaluation of any criminal act. Furthermore, no animal or inanimate model has yet been described that adequately mimics injuries during child abuse. Until these difficulties are surmounted, much of what we think we know about the systemic and ocular findings of child abuse will continue to be the result of speculation rather than based on sound evidence.

Ocular hemorrhages are a frequent finding in cases of child abuse. We propose that retinal hemorrhage in shaking abuse is caused by venous stasis and leakage from retinal vessels, which, if prolonged, can lead to circumferential macular and peripheral retinal folds with blood- and protein-filled schisis cavities. This mechanism is supported by our previously unreported findings in fibrin-stained sections. The repetitive rotational torsion about the optic nerve–globe junction causes tears in dural bridging veins and the vessels near the circle of Zinn, resulting in optic nerve subdural hemorrhages and peripapillary scleral hemorrhages, respectively. These peripapillary scleral hemorrhages may be pathognomonic of shaking abuse. Stains for hemosiderin may aid in detecting prior trauma, in the absence of concurrent hemorrhages. The presence of these features should raise the suspicion for nonaccidental trauma.

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Neuropathology of inflicted head injury in children
I. Patterns of brain damage

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Summary
Fifty-three cases of non-accidental head injury in children were subjected to detailed neuropathological study, which included immunocytochemistry for microscopic damage. Clinical details were available for all the cases. There were 37 infants, age at head injury ranging from 20 days to 9 months, and 16 children (range 13 months to 8 years). The most common injuries were skull fractures (36% of cases), acute subdural bleeding (72%) and retinal haemorrhages (71%); the most usual cause of death was raised intracranial pressure secondary to brain swelling (82%). On microscopy, severe hypoxic brain damage was present in 77% of cases. While vascular axonal damage was found in 21 out of 53 cases, diffuse traumatic axonal injury was present in only three. Eleven additional cases, all of them infants, showed evidence of localized axonal injury to the craniocervical junction or the cervical cord. When the data were analysed by median age at head injury, statistically significant patterns of age-related damage emerged. Our study shows that infants of 2–3 months typically present with a history of apnoea or other breathing abnormalities, show axonal damage at the craniocervical junction, and tend also to have a skull fracture, a thin film of subdural haemorrhage, but lack extracranial injury. Children over 1 year are more likely to suffer severe extracranial, particularly abdominal, injuries. They tend to have larger subdural haemorrhages, and where traumatic axonal injury is present, show patterns of hemispheric white matter damage more akin to those reported in adults. Diffuse axonal injury is an uncommon sequel of inflicted head injury in children.

Keywords: non-accidental head injuries; infant head injury; subdural hemorrhage; child abuse

Abbreviations: βAPP = β amyloid precursor protein; NAI = non-accidental injury; DAI = diffuse axonal injury

Introduction
The neuropathology of inflicted head injury, whether adult assault or non-accidental injury (NAI) in children, has not been fully studied. Given the central role that a neuropathologist may be asked to play in a fatal NAI case, it is surprising that since the first paper of Lindenberg and Freytag in 1969, which described 'contusional tears' in the brains of young infants who had suffered inflicted head injury with impact (Lindenberg and Freytag, 1969), there have been only a few published studies of the neuropathology of NAI in the literature (Calder *et al.*, 1984; Vowles *et al.*, 1987; Hadley *et al.*, 1989; Shannon *et al.*, 1998; Gleckman *et al.*, 1999), the largest series being 14 cases; in none of them have detailed clinicopathological correlations been attempted. This dearth of information means that much of the data needed for interpretation of findings in medicolegal situations has to be extrapolated from what is known about adult head injury, despite the fact that the majority of fatal cases fall in the very young age group, and there are many reasons why the immature brain should react differently to trauma. We have examined the brains from 53 well-documented cases of inflicted head injury with the aim of analysing patterns of neuropathology in non-accidental head injury in children of different ages to provide data for use in assessment of such cases.

Methods
Study population
The study population comprised all the cases of paediatric head injury in the files of two neuropathologists (H.L.W. and J.F.G.) that fulfilled one of a series of criteria for the diagnosis of NAI. The diagnostic criteria used were: (i) head injuries in which there had been a confession by the perpetrator

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($n = 7$); (ii) cases in which non-accidental head injury had been established as a result of conviction in a criminal court, and in which there were also unexplained extracranial injuries to support this ($n = 19$); (iii) cases with unexplained injuries elsewhere in the body, in addition to the head injury, but no conviction ($n = 8$); (iv) cases in which the carer was tried and convicted of injuring the child, but in which there were no extracranial injuries ($n = 12$); and (v) cases in which there was a major discrepancy between the explanation of the incident given by the carer and significant injuries such as a skull fracture, or if the history was developmentally incompatible ($n = 7$). Cases were excluded if there was insufficient material in the form of either blocks or residual brain tissue. Full documentation, including witness statements and court papers, was available for 52 out of 53 cases; for one case only a clinical history was available, but there was sufficient detail to merit inclusion into the study.

The research proposal was scrutinized and approved by the Research Ethics Committee of the East London and City Health Authority (T/98/007).

Sampling for microscopy

The brains were systematically sampled for histology, and where initial sampling had been inadequate, further blocks were taken. In 32 cases large hemisphere blocks were used. In 52 out of 53 cases, minimum sampling included several blocks of hemispheric white matter, of corpus callosum, internal capsule, cerebellum, midbrain, pons, medulla and spinal cord. In one case only, there were no blocks of the brainstem, merely cerebrum and several segments of cervical spinal cord.

Staining methods

H & E (haemotoxylin and eosin) staining was performed on all blocks. Immunocytochemistry for β-amyloid precursor protein (βAPP) (Chemicon monoclonal, clone 22C11) was carried out using an avidin–biotin complex, peroxidase-labelling detection system (Vector Universal Elite kit). Anti-βAPP was used at a dilution of 1 : 300, with 1 h incubation at room temperature, after microwave antigen retrieval. For cases with long survival or evidence of a previous head injury, CD68 (Dako, PG-M1) was used in addition, at a dilution of 1 : 100, with 40 min incubation and microwave retrieval. Additional stains, including Perls, HVG (haematoxylin van Gieson) and immunohistochemistry for GFAP (glial fibrillary acidic protein), were performed on selected blocks.

Results

Subjects and details of injury

The data consisted of 53 cases of fatal non-accidental head injury. Details of the presentation to paramedics or doctors were: apnoea, abnormal breathing or collapse, with the child suddenly turning blue and limp (33 cases); found dead (six cases); child dropped (four cases); fell (two cases); found unconscious at the bottom of the stairs (two cases); and thrown, shaken or stabbed (one case of each). There were no details of presentation in three cases. The age at head injury ranged from 20 days (at 37 weeks' gestation) to 8 years. Since one of the aims of our analysis was to focus on the differences between younger and older NAI cases, we divided the series into those aged <1 year at presentation (who we refer to as 'infants', $n = 37$), and those aged ⩾1 year (who we refer to as 'children', $n = 16$). Outline details of the study population and findings are shown in Tables 1 and 2. Figure 1 summarizes the results in Table 2 by displaying the relative risk (and 95% confidence interval) corresponding to each factor, indicating whether the presence of the factor was more common, less common or the same in infants compared with children.

Table 1 *Summary information on 53 NAI cases*

Factor		All cases	Infants (<1 year)	Children (⩾1 year)
Sex (number)	Male	27	18	9
	Female	26	19	7
Age at head injury (days)	Number	53	37	16
	Median	124	73	630
	Mean	347	97	925
	Range	20–2920	20–273	388–2920
Survival (days)	Number	49*	37	12
	Median	1	1	0.7
	Mean	21	27.7	1.2
	Range	0–695	0–695	0–3

*Accurate survival times were not available for four cases.

General autopsy findings

Fifty-one per cent of subjects (27 out of 53) had significant extracranial injury: of these, 10 had recent or old fractures of the ribs or clavicle, four had long bone fractures, six had serious abdominal injuries, principally bleeding from hepatic and/or mesenteric lacerations. Seven had burns or extensive bruising. Evidence of previous trauma to the head was seen in 20% of cases, to the body in 8% and to both head and body in 4%.

Eighty-five per cent of subjects (45 out of 53) had signs of impact to the head at autopsy, in the form of either subscalp bruising or skull fracture. In the remaining eight cases, all infants, neither bruising nor fracture was found at autopsy. Skull fractures, present in 19 out of 53 cases, were found in the parietal and/or the occipital bones in 18 out of 19 cases. In six subjects the fractures were bilateral. The median age in those with a skull fracture was 3 months and 6 months for those without. No extradural haemorrhages were observed in cases with skull fractures.

Macroscopic neuropathology

Eighty-one per cent of the cases (43 out of 53) were found to have subdural haemorrhages, 38 of them acute. The

Table 2 *Principal details of all 53 NAI cases, comparing findings in infants with those in children*

Factor		% All cases (number)	% Infants (<1 year) (number)	% Children (⩾1 year) (number)	Relative risk	*P*-value‡
Clinical						
Apnoea or respiratory systems	No	37 (19)	22 (8)	69 (11)	1.00	0.002
	Yes	63 (33)	78 (28)	31 (5)	2.49	
General autopsy						
Significant extracranial injury	No	49 (26)	59 (22)	25 (4)	1.00	0.03
	Yes	51 (27)	41 (15)*	75 (12)†	0.54	
Earlier injury	No	68 (34)	67 (24)	71 (10)	1.00	1.00
	Any	32 (16)	33 (12)	28 (4)	1.17	
	Head	20 (10)	19 (7)	21 (3)	0.98	
	Body	8 (4)	8 (3)	7 (1)	–	
	Both	4 (2)	6 (2)	0 (0)	–	
Subscalp bruising	No	23 (10)	27 (10)	0 (0)	1.00	0.02
	Yes	77 (43)	73 (27)	100 (16)	0.63	
Macroscopic neuropathology						
Skull fracture(s)	No	64 (34)	57 (21)	81 (13)	1.00	0.12
	Yes	36 (19)	43 (16)	19 (3)	2.31	
Extradural haemorrhage	No	100 (53)	100 (37)	100 (16)	–	–
Intracranial pressure raised	No	18 (9)	19 (7)	15 (2)	1.00	0.71
	Yes	82 (42)	81 (29)	85 (13)	0.93	
Subdural haemorrhage	No	19 (10)	16 (6)	25 (4)	1.00	0.46
	Any	81 (44)	84 (31)	75 (12)	1.12	
	Old	9 (5)	8 (3)	12 (2)	1.00	
	Thin film	64 (34)	70 (26)	50 (8)	1.22	
	Mass lesion	8 (4)	5 (2)	12.5 (2)	–	
Subarachnoid haemorrhage	No	40 (21)	35 (13)	53 (8)	1.00	0.56
	Any	60 (31)	65 (24)	47 (7)	1.39	
	Old	12 (6)	14 (5)	7 (1)	2.50	
	Yes	48 (25)	51 (19)	40 (6)	1.39	
Intracerebral haemorrhage	No	94 (50)	95 (35)	94 (15)	1.00	1.00
	Yes	6 (3)	5 (2)	6 (1)	0.86	
Contusional tears	No	92 (48)	89 (32)	100 (16)	1.00	0.30
	Yes	8 (4)	11 (4)	0 (0)	3.7§	
Microscopy						
Hypoxia-ischaemia	No	19 (10)	13 (5)	31 (5)	1.00	0.17
	Any	81 (43)	87 (32)	69 (11)	1.26	
Focal infarct		4 (2)	3 (1)	6 (1)	–	
	Global hypoxia	77 (41)	84 (31)	63 (10)	–	
Vascular axonal injury¶	No	60 (31)	63 (23)	50 (8)	1.00	0.37
	Yes	40 (21)	37 (13)	50 (8)	0.72	
Focal traumatic axonal injury¶	No	58 (30)	55 (20)	63 (10)	1.00	0.01
	Central wh. m.	21 (11)	14 (5)	37 (6)	0.53	
	Craniocervical	21 (11)	31 (11)	0 (0)	7.45§	
Diffuse axonal injury	No	94 (50)	95 (35)	94 (15)	1.00	1.00
	Yes	6 (3)	5 (2)	6 (1)	0.86	
Retinal bleeding	No	29 (11)	30 (9)	25 (2)	1.00	1.00
	Yes	71 (27)	70 (21)	75 (6)	0.93	

The relative risk is estimated as the percentage of infants with the presence of the specified factor divided by the percentage of children with the same finding. Complete results were not available for all 53 cases. wh. m. = white matter. *Of these 15, nine had an old injury and six had a new injury. †All 12 had a new injury. ‡Using Fisher's exact test. §0.5 used instead of 0 when estimating the relative risk. ¶Impossible to assess in one case.

remaining five showed pigmentation and membrane formation indicative of older subdural bleeding. In 34 cases the subdural was trivial in terms of quantity of blood, almost invariably described in the post-mortem report as a 'thin film'; 28 of these were bilateral. The four haematomas that were large enough to act as space-occupying lesions occurred in four older subjects (ages 8 months, 9 months, 3 years and 4 years). Cases with a significant subdural were on average

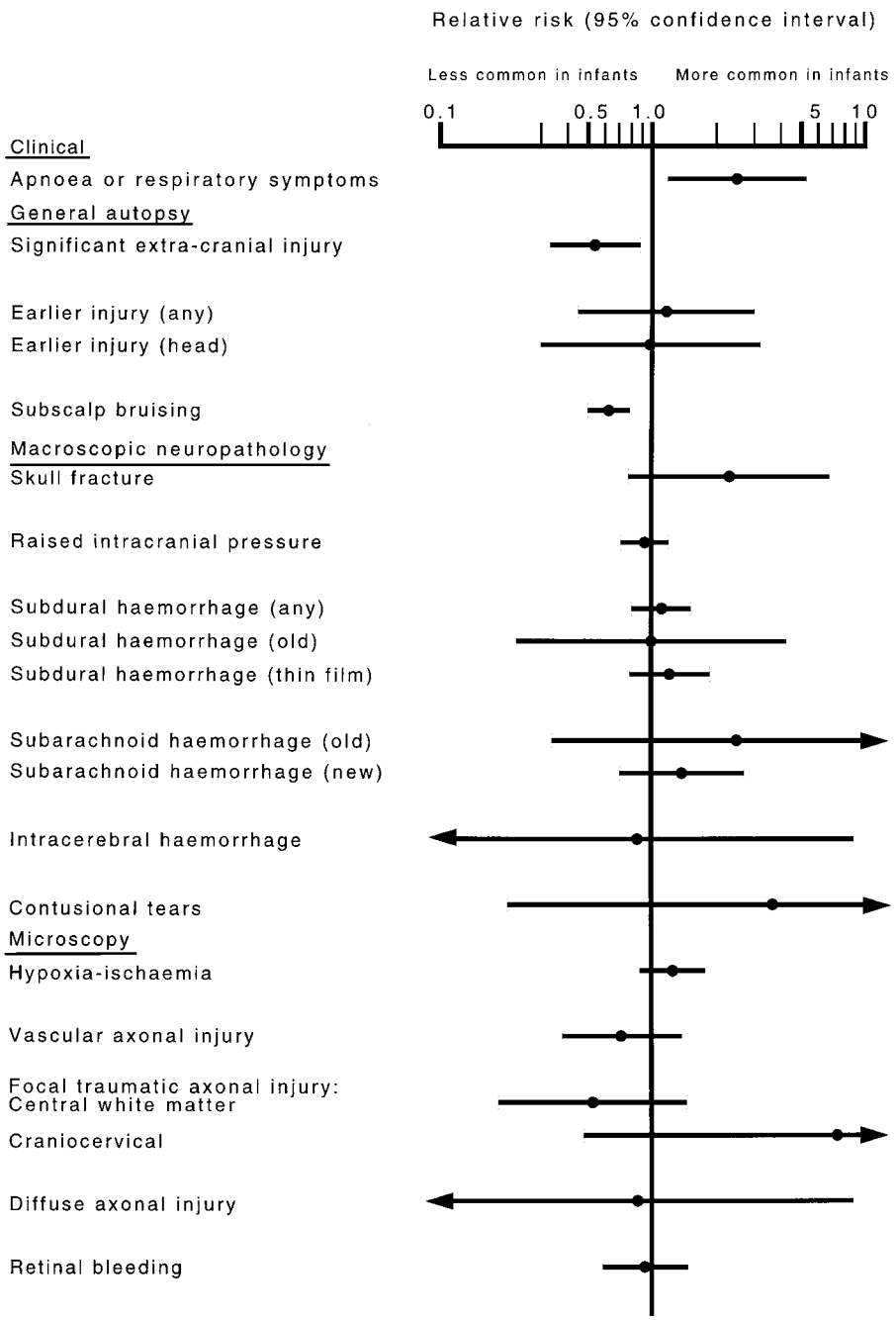

Fig. 1. The relative risk and 95% confidence interval of the presence of specified factors in infants compared with children. The relative risk is the percentage of infants with the specified finding divided by the percentage of children with the same finding. For example, from Table 2, 78% of infants presented with apnoea compared with 31% of children; the relative risk is thus 2.5 (i.e. infants were 2.5 times more likely to present with apnoea than children).

almost 2 years old, ~1.5 years older than those without any subdural haemorrhage or merely trivial bleeding ($P = 0.10$).

The brain weight was increased in 82% of the cases in which it had been recorded at post-mortem, and raised intracranial pressure was the most common cause of death.

Of other features, subarachnoid haemorrhage was present in about half the cases (25 out of 52), in association with subdural bleeding or with a fracture, or both. It did not occur on its own. Cortical contusions were seen in five cases only. Contusional tears were found in only four cases and all were <4.5 months old. Three had fractures and the fourth showed neither scalp bruising nor fracture. Intracerebral haemorrhage was seen in only three cases, of which it was trivial in one, but acted as a mass lesion in two. A layer of epidural

bleeding was seen round the upper cervical cord in three cases.

Microscopic changes

Of the 38 cases in which the eyes were examined by a pathologist, 71% (27 out of 38) had retinal haemorrhages, of which 26 were bilateral. The original sampling of the eyes had not been uniform, however, and we were unable to analyse the distribution of the bleeding further. There was a significant association between subdural bleeding and the presence of retinal haemorrhages; in all cases with intraocular bleeding there were subdural haemorrhages ($P < 0.001$). Of the 10 cases in which there were no subdural haemorrhages, five had the eyes examined and none of these showed retinal bleeding.

According to the histories given, 21 of the 49 children for whom there were accurate survival data were either found dead or died in <2 h; survival data was insufficient for any histological changes to be detectable. However, 13 of these subjects, including 11 infants, had clearly lived longer after the cerebral insult than the history suggested, since they showed widespread hypoxic changes on haematoxylin and eosin staining (generally believed to take ~4–6 h to develop in adults; Adams and Graham, 1994). Ten of them also showed foci of recent traumatic axonal damage on βAPP immunocytochemistry (not detectable in adults with <2 h survival; Geddes *et al.*, 1997), which were restricted to the corticospinal tracts in the brainstem in five cases.

The most frequent microscopic finding in the brains was global neuronal hypoxia–ischaemia, identified by routine neuropathological criteria (widespread neuronal cytoplasmic eosinophilia and shrinkage), and present in 84% of infants and 63% of older children. Damage to axons, both vascular and traumatic, was detected with βAPP immunohistochemistry in a number of cases. Vascular axonal damage was diagnosed when a 'geographic' pattern of white matter immunoreactivity was present, which was usually widespread and related to vessels. Focal geographic βAPP expression, commonly seen in the diencephalon and brainstem, was taken to be outlining areas of incipient ischaemia resulting from brain swelling (Geddes *et al.*, 2000). [Typical patterns interpreted as vascular in origin, are illustrated in Fig. 1 in the companion paper (Geddes *et al.*, 2001)]. Such appearances were seen in 37% of infants and 50% of the older children, and were assumed to be the sequelae of ischaemic white matter damage secondary to brain swelling and raised intracranial pressure—indeed, raised intracranial pressure was associated with the presence of vascular βAPP expression ($P = 0.06$).

Traumatic damage to axons was identified by finding βAPP-immunoreactive axons or bulbs, scattered or in groups in hemispheric white matter, corpus callosum and internal capsule. Where the brainstem was also involved, the case was—by definition—one of diffuse axonal injury (DAI) (see below). In cases with survival of >24–48 h, ischaemic axonal damage could be excluded by routine histopathological criteria, and with survival of 1 week or over, microglial clusters were present around foci of axonal pathology (Geddes *et al.*, 2000). The traumatic damage seen in the series varied from scattered foci in the hemispheres only, to damage severe enough to be called DAI. This is defined as widespread traumatic axonal damage occurring throughout the centrum semiovale, particularly parasagittal white matter, corpus callosum, internal capsule and cerebellar peduncles in the rostral brainstem (Adams *et al.*, 1989). In our series it was found in only three out of 53 cases: two of these were infants, who had multiple skull fractures; the other was an 8-year-old child. Lesser degrees of hemispheric axonal damage were detected in only 11 cases, five infants and six older children (see Table 2, which distinguishes between focal 'traumatic axonal injury' and the more severe 'diffuse axonal injury'; Geddes *et al.*, 2000). In one case the state of preservation of the brain made the immunohistochemistry impossible to assess.

While DAI was rare, localized axonal damage was detected in 11 subjects in the infant group, in the lower brainstem (eight cases) and in cervical cord roots (three cases). The damage in the brainstem was found to be anatomically confined to the corticospinal tracts in the lower pons and medulla, and the number of axonal swellings varied from very few in the long tracts to a large number, but were only present in these tracts, bilaterally (the worst case is illustrated in Fig. 2). In one other case survival had been sufficient for a microglial reaction to be established round the bulbs.

Patterns of injury in different age groups

Abdominal injuries were less common in infants (two out of 37 infants compared with four out of 16 children; relative risk 0.22 of infants to children; $P = 0.07$). Infants were less likely to have a significant extracranial injury (relative risk 0.54; $P = 0.03$), and they tended also to have evidence of previous traumatic damage (nine out of 15 infants had an old injury compared with none out of 12 children, $P = 0.001$).

Specific neuropathological features were also analysed according to median age at head injury (Figs 3 and 4). At 2–3 months of age, cases tended to have presented with apnoea ($P = 0.002$) and damage at the craniocervical junction, particularly in the corticospinal tracts ($P = 0.02$). There was a suggestion that they also tended to have a skull fracture and trivial subdural bleeding ($P = 0.10$). The average age differences were: cases with apnoea were 1 year younger than those without apnoea; those with corticospinal tract damage were 3 months younger than those without; those with a skull fracture were 3 months younger than those without the corresponding damage. The youngest cases also tended not to have significant extracranial injury ($P = 0.10$) and those without significant extracranial injury were ~1 month younger than those with. Finally, cases with traumatic axonal damage in the cerebrum tended to be ~15 months old on average, and ~10 months older than those without any

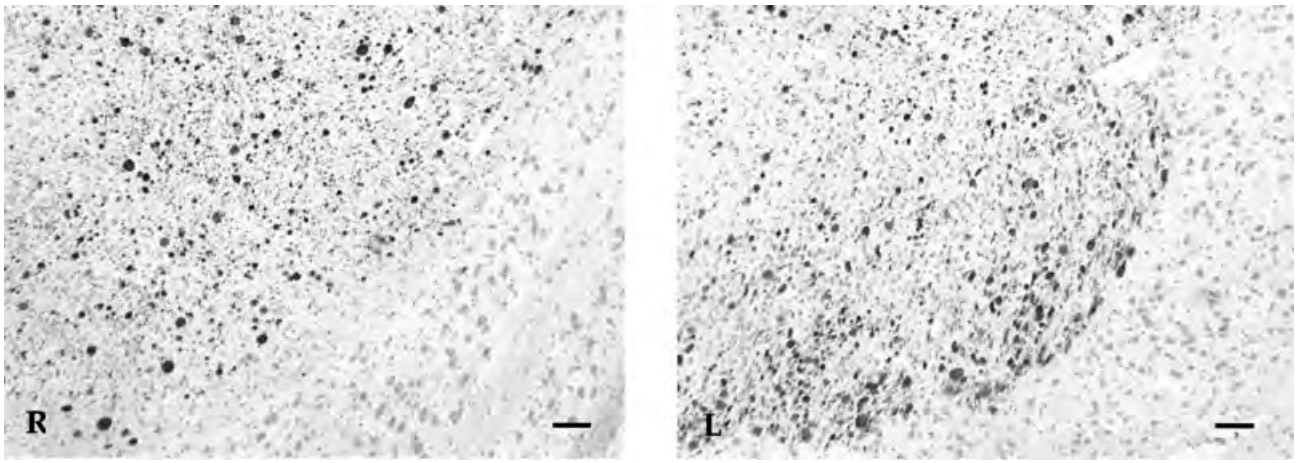

Fig. 2. A low power view of both corticospinal bundles in the caudal pons of a 20-day-old girl. Very many βAPP-immunoreactive axonal bulbs and swellings are seen. Note that there is no βAPP expression in the transverse fibres. R = right; L = left. Bar = 75 μm.

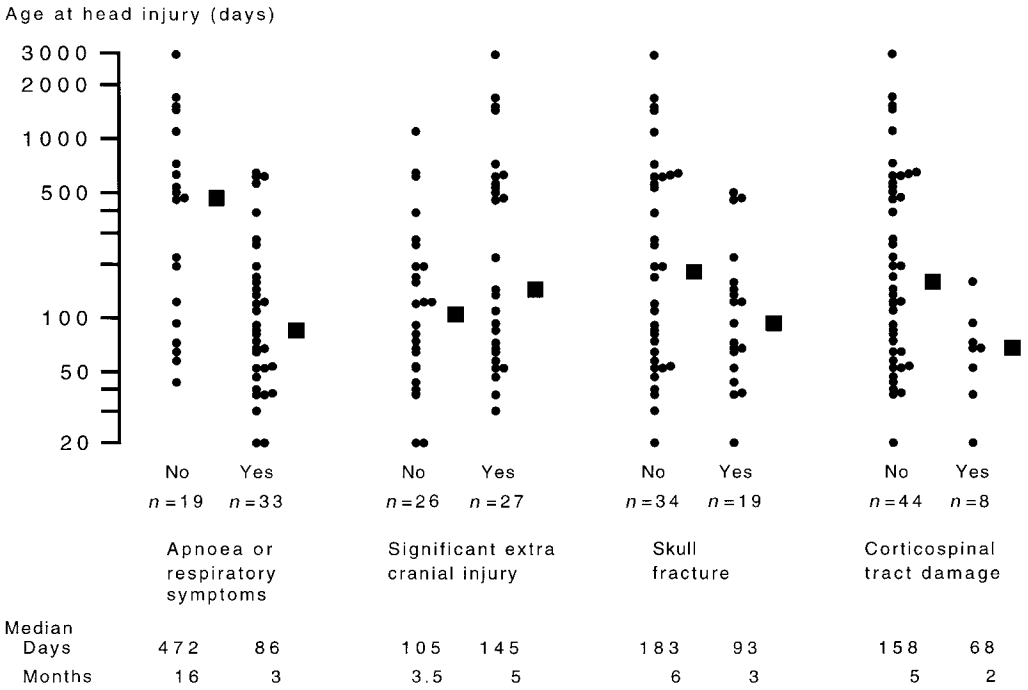

Fig. 3. The age (or approximate age) at head injury in the 53 cases of NAI according to the presence or absence of specified factors. The median in days and months (square symbols) are shown. The *P*-values for the difference between the medians (using a non-parametric Wilcoxon rank test) were $P = 0.002$ (apnoea or respiratory problems); $P = 0.10$ (significant extracranial injury); $P = 0.10$ (skull fracture); $P = 0.02$ (corticospinal tract damage). The presence or absence of apnoea at presentation was not known for one case and for another case the lower brainstem was not available.

traumatic axonal damage ($P = 0.008$). The tendency for large subdural haemorrhages to be found in older children has been recorded above.

'Shaken-only' infants versus others

The eight infants who showed no signs of impact were assumed to have been shaken, and in one case a carer had confessed to having done so. The clinical presentation of all but one of these eight 'shaken-only' cases was of collapse or respiratory arrest. In seven the brain was swollen sufficiently to cause death; the eighth child survived 5 months in hospital after his head injury with severe hypoxic–ischaemic brain damage, before dying of bronchopneumonia. One of the eight had a rib fracture, but no other extracranial injuries were seen. Seven had subdural haemorrhages, described in each case as a 'thin film' or 'small', and five of the six cases in which the eyes were examined had bilateral retinal

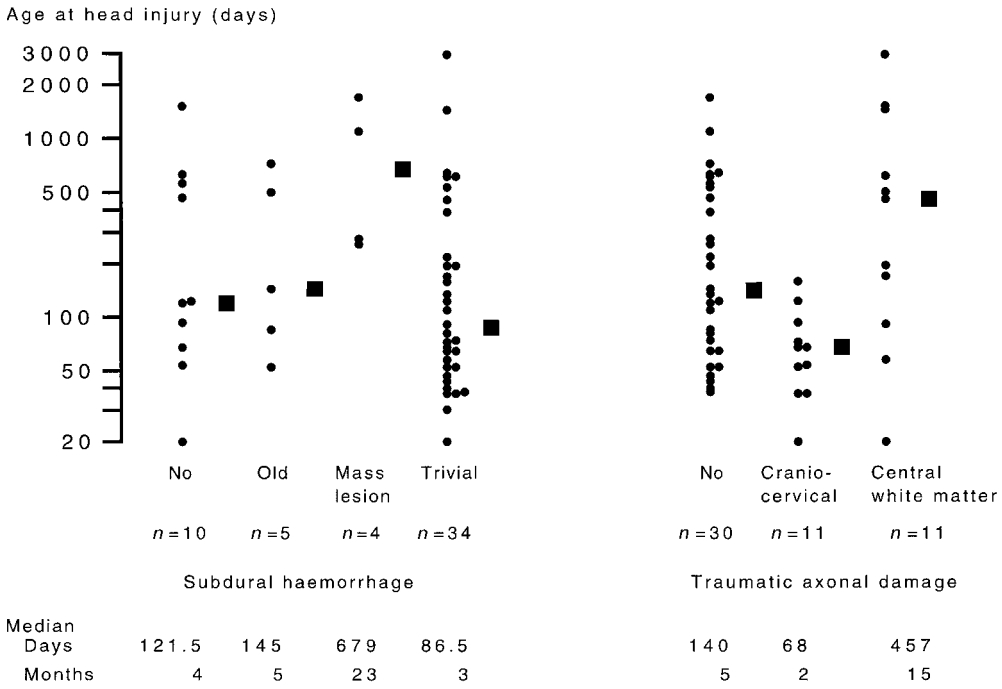

Fig. 4. The age (or approximate age) at head injury in the 53 cases of NAI according to the absence or type of subdural bleeding and traumatic white matter damage. The median in days and months (square symbols) is shown. The *P*-values for the difference between the medians (using a non-parametric Kruskal–Wallis analysis of variance) were $P = 0.10$ (subdural haemorrhage); $P = 0.008$ (traumatic axonal damage). Information on traumatic axonal damage was not known for one case.

haemorrhages. One case had corticospinal tract damage, and one had axonal damage in the nerve roots in cervical spinal cord segments. There was no evidence of differences between the pathology in this 'shaken-only' group and the 29 infants who had evidence of impact.

Discussion

While the literature contains many studies of neuropathological findings in adult head injury, there is virtually no comprehensive information from neuropathologists on brain damage in fatal paediatric head injury. The few studies that have been published have concentrated on specific features only, rather than patterns of injury, and the numbers of cases have been small. Lindenberg and Freytag reported a series of 16 head-injured infants, collected because they all demonstrated unusual impact lesions in white matter which the authors termed 'contusional tears' (Lindenberg and Freytag, 1969). These lesions were also documented by Calder and colleagues in seven out of nine infants, all <5 months (Calder *et al.*, 1984), almost all in association with skull fractures. Hadley and colleagues described damage to the craniocervical junction in a small series (Hadley *et al.*, 1989), while Hart reported on correlations between post-mortem imaging of the brain and autopsy findings (Hart *et al.*, 1996). Three further papers, which looked at microscopic brain changes in NAI, have suggested that diffuse axonal injury is a feature of non-accidental head injury (Vowles *et al.*, 1987; Shannon *et al.*, 1998; Gleckman *et al.*, 1999); their findings are discussed in more detail in a companion paper (Geddes *et al.*, 2001).

A common drawback of most of these series, particularly those relating to the question of microscopic damage, is that they contain limited additional clinical detail, and very little attention has been paid to potentially confounding factors such as artificial ventilation, brain swelling, the presence of global hypoxia and so on (Geddes *et al.*, 2000). In some, the paucity of neuropathological detail supplied undermines the validity of the authors' conclusions: this is discussed in more detail elsewhere (Geddes *et al.*, 2001). The lack of firm published data on the neuropathology is compounded by the fact that review articles or texts which mention the pathology of NAI, particularly that caused by shaking, tend merely to reiterate the neuropathological features of adult trauma (Leestma, 1988; Brown and Minns, 1993; David, 1999). Our study in fact confirms that there are significant differences not only between the pathology of non-accidental head injury in children and adults, but also between children of different ages.

The diagnosis of NAI

One of the major problems encountered in assembling cases for NAI series is that the presenting history may not be accurate, and very few confessions are obtained (seven out of 53 in our series). Often child abuse is first suspected when subdural bleeding or retinal haemorrhages are detected—i.e. on the basis of the pathology alone. However, if one aims to

produce reliable data this approach is untenable, since it may result in cases of accidental injury being included.

In an attempt to be as certain as possible that we were indeed dealing with cases of inflicted head injury, we drew up diagnostic criteria for this study (see Methods). Even so, we are aware that the 12 cases in one category (where a conviction was obtained, in the absence of extracranial injuries), might conceivably include cases that were not in fact NAI, even though they had the pathology widely taken to be pathognomonic or at least 'highly suggestive' of child abuse. Lack of firm objective grounds for concluding that cases were NAI is another drawback of many series in the literature, including clinical and forensic series.

Distinctive features of inflicted head injury in children

Despite the frequency of skull fractures, extradural haematomas are rarely reported in NAI. There were none in our series. This may be because the common sites of skull fracture [almost exclusively parietal or occipital in our series, as in others (Duhaime *et al.*, 1998)], are ones that would not be expected to compromise a major artery, but the fact that the dura is very densely adherent to the undersurface of the infant skull, and not easily stripped from the bone, probably also contributes. Extradural haemorrhage around the cervicomedullary junction, however, while not a common feature in adult head injury, has been reported in NAI (Hadley *et al.*, 1989), and was noted in three of our cases.

In a general review of the pathology of what he first described as the 'whiplash shaken infant syndrome', Caffey talked of 'massive traumatic intracranial bleeding' resulting from shaking (Caffey, 1974). In point of fact, subdural haemorrhages in NAI in young children are materially different from those seen in adults, and are rarely 'massive' (Carter and McCormick, 1983; Duhaime *et al.*, 1998; David, 1999). They are almost invariably bilateral thin films of blood over the cerebral hemispheres, which do not require neurosurgical intervention—indeed, they may be missed on scans, even by magnetic resonance imaging, and only found at post-mortem (Hart *et al.*, 1996; Feldman *et al.*, 1997). Of themselves they do not usually cause mass effect, and may be survivable—as the occasional finding of organizing haematomas at autopsy shows. In our series, the few cases in which the subdural acted as a significant mass lesion were seen in the older infants and children. The question that needs answering is: given the differences between 'adult'-type subdurals and those seen in infants in NAI, are the conditions or forces that produce the two necessarily the same?

Subarachnoid bleeding is also rarely clinically significant in NAI. Most occurs over the hemisphere, associated with fracture sites or underlying subdurals, where it presumably results from rupture of veins crossing the subarachnoid space. Contusions, superficial foci of haemorrhagic necrosis which characteristically affect the base of the brain and areas of cortex underlying skull fractures, are seldom seen in infants, although common in older children. This difference is probably explained in part by the fact that the floor of the infant skull is smooth, and in part by the soft consistency of the incompletely myelinated brain.

Macroscopically, the majority of brains showed merely swelling, manifest as increased brain weight. Histological study revealed that a significant proportion of the children who had been reported to have been found dead or died rapidly had survived an insult sufficiently long for neuronal cytoplasmic or axonal pathology to be detectable. In all brains in which there were microscopic changes, global hypoxia was the most usual finding. While prolonged ventilation might be thought to account for this in a proportion of cases, the clinical notes for our cases generally indicate that brain swelling and hypoxic changes were evident when the child was scanned on arrival at hospital. In terms of microscopic brain damage, we took great care to sample the brain adequately, using large brain blocks where possible, and to attempt to distinguish between axonal damage caused by trauma and axonal damage secondary to hypoxia–ischaemia, raised intracranial pressure and/or brain shift. Our experience has always been that DAI is a rarity in NAI. In the present series it was only found in three out of 53 cases: in an 8-year-old child (in whom one might reasonably expect neuropathology akin to that seen in a young adult) and in two infants, both of whom had very severe head injuries with bilateral skull fractures. These findings would tend to confirm the study of Duhaime and her colleagues (Duhaime *et al.*, 1987) which suggested that shaking does not reproduce the forces necessary for DAI to occur. The localized axonal damage demonstrated in corticospinal tracts in the lower brainstem, similar to that reported in adults with non-disruptive cervical cord damage due to a hyperextension neck injury (Lindenberg and Freytag, 1970; Geddes *et al.*, 2000), may in fact be more significant. Injury at this point, presumably caused by stretch to the neuraxis produced by cervical hyperextension, might provide an explanation for the frequent occurrence of apnoea at presentation. We have discussed this finding, and the general question of what our study has revealed of the nature of diffuse brain damage and mechanisms of injury, elsewhere (Geddes *et al.*, 2001).

Contusional tears, the traumatic lesions peculiar to the brains of young infants (Lindenberg and Freytag, 1969; Calder *et al.*, 1984), which are occasionally detected by neuro-imaging (Hausdorf and Helmke, 1984; Jaspan *et al.*, 1992), were found in four of our infant cases at post-mortem. Because these tissue tears are believed to be the result of shearing of the interface between grey and white matter, the conditions have been assumed by authors in the NAI literature to be the same as those that cause DAI and the tissue tears reported in adult head injury (Graham and Gennarelli, 1996). However, this may not necessarily be true: the severe angular or rotational acceleration, with or without deceleration, necessary to produce widespread 'shearing' forces to axons

throughout the brain may well be quite different from the forces necessary to produce localized 'shearing' between grey and white matter (two regions of very different consistency in the infant), after impact injury.

Retinal haemorrhages, the detection of which in a moribund child so frequently first raises suspicions of NAI—for which they are thought to be an important marker (OCA Working Party, 1999)—were found in 71% of our cases, a figure comparable with findings of clinical series (Duhaime *et al.*, 1998). They were seen both in cases in which impact had occurred, and in cases in which there was no macroscopic evidence of impact. There was, however, a statistically significant association between subdural and retinal bleeding, mentioned above, and we found no cases of retinal haemorrhages without subdural bleeding. A discussion of the aetiology of retinal haemorrhages, characteristic of the young population who are biomechanically particularly vulnerable to shaking-type injuries, is beyond the scope of this paper.

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Neuropathology of inflicted head injury in children
II. Microscopic brain injury in infants

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Summary
There are very few reports in the literature dealing with the neuropathology of infant head injury, and the question of whether diffuse traumatic brain damage [diffuse axonal injury (DAI)] occurs in such children has not yet been reliably established by detailed neuropathological studies. We report the findings in the brains of a series of 37 infants aged 9 months or less, all of whom died from inflicted head injuries, and 14 control infants who died of other causes. Axonal damage was identified using immunohistochemistry for β-amyloid precursor protein. Full clinical details were available for each case, the most constant of which in the study cohort was an episode of significant apnoea at presentation, found to have been recorded in 75% of cases. Global hypoxic damage was the most common histological finding. Widespread axonal damage, interpreted as vascular, was present in 13 cases, but widespread traumatic axonal injury was found in only two children, both of whom had severe head injuries with multiple skull fractures. Epidural cervical haemorrhage and focal axonal damage to the brainstem and the spinal nerve roots, found in 11 cases but not in controls, indicate that the craniocervical junction is vulnerable in infant head injury, the neuropathology being that of stretch injury from cervical hyperextension/flexion. Damage to this region could account for the observed apnoea, which could in turn lead to hypoxic damage and brain swelling. The observation that the predominant histological abnormality in cases of inflicted head injury in the very young is diffuse hypoxic brain damage, not DAI, can be explained in one of two ways: either the unmyelinated axon of the immature cerebral hemispheres is relatively resistant to traumatic damage, or in shaking-type injuries the brain is not exposed to the forces necessary to produce DAI.

Keywords: cervical hyperextension; diffuse axonal injury; infant head injury; non-accidental head injury

Abbreviations: βAPP = β-amyloid precursor protein; DAI = diffuse axonal injury; NAI = non-accidental injury

Introduction
Clinical reviews of fatal non-accidental infant head injury, especially of the 'shaken baby syndrome', tend to emphasize a constellation of findings: little or no evidence of impact to the head, acute subdural haemorrhage, intraocular haemorrhages of various types, brain swelling and 'diffuse brain damage', often specified as diffuse axonal injury (DAI) (Caffey, 1974; Brown and Minns, 1993; Munger *et al*., 1993; Duhaime *et al*., 1998; Lancon *et al*., 1998; David, 1999). The fact that such children are unconscious on arrival at hospital, with swollen brains and markedly hypoxic parenchymal changes on CT scan, suggests that they have indeed suffered severe diffuse brain damage. However, review of the literature suggests that the scientific evidence for this being traumatic damage is scanty. We have undertaken a detailed neuropathological study of 37 infants known to have suffered inflicted head injury to see whether DAI, an entity originally described in severe acceleration–deceleration injuries in adults, does in fact occur in non-accidentally injured infants. We have then critically reviewed papers dealing with the neuropathology of microscopic brain injury in non-accidental injury (NAI), comparing our findings with those reported previously.

Methods
Study population
The study population comprised 37 cases of head injury, established to be non-accidental according to criteria described previously (Geddes *et al*., 2001). The subjects were all infants, defined as children <1 year of age. Their age

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range at the time of the head injury was 20 days to 9 months (median 2.4 months, mean 3.2 months). In 28 out of 37 cases (76%), the presenting history given by the carer to the ambulance and/or hospital staff was of the child having stopped breathing, having had respiratory difficulties, or having turned blue and floppy. Four infants were said to have been found dead, three others were reportedly dropped, one was said to have fallen and one was thrown across the room. Survival ranged from 0 to 23 months (median 2.4 months, mean 3.2 months).

A control series of 14 infants who had died of other causes was also used, to determine whether any changes found in the NAI cases were present in other infant deaths. These comprised 12 males and two females, of ages ranging from 2 days to 11 months (median 3 months). The causes of death were SIDS (sudden infant death syndrome) (seven), respiratory tract infection with breathing difficulties (five), perinatal asphyxia (one) and gastroenteritis (one). All of the SIDS cases were found dead, and one of those with respiratory symptoms survived $<$30 min. Survival of the remaining six cases was $>$4 h.

The research proposal was approved by the local research ethics committee. Full documentation, including clinical histories and witness statements, was available for all cases.

Sampling for microscopy

The brains were systematically sampled for histology, and where initial sampling had been inadequate, further blocks were taken. In 21 study cases and 13 out of 14 controls, large blocks were used. In 50 out of the 51 brains (subjects + controls), minimum sampling included several blocks of hemispheric white matter, of corpus callosum, internal capsule, cerebellum, midbrain, pons, medulla and spinal cord. In the remaining brain, an NAI case, there were no blocks of the brainstem, merely cerebrum and several segments of cervical spinal cord.

Staining methods

Immunocytochemistry for β-amyloid precursor protein (βAPP) was performed on all cases, with CD68 additionally used on long surviving cases or cases in which there appeared to have been an earlier head injury. Additional routine stains such as Perls were used as required. Technical methods and the criteria used in this study for the diagnosis of microscopic brain injury (hypoxic neuronal damage and the interpretation of axonal damage) have been described in a companion paper (Geddes *et al*., 2001) (see also Fig. 1).

Literature seach

A literature search was undertaken, looking for neuropathological series describing histological findings in NAI children.

Results

Clinical details and neuropathological findings have already been reported for the study population as part of a larger series of non-accidental head injury in children (Geddes *et al*., 2001). The principal details are summarized in Tables 1 and 2; further relevant information is given below.

Microscopic axonal damage

Study group

In total, βAPP positivity in axons was detected in 25 out of 37 cases, including 11 of the 14 cases who were said to have been found dead. In 13 out of 25, the axonal pathology appeared to be largely vascular in nature, associated with brain swelling and raised intracranial pressure (Fig. 1). Five brains showed minimal traumatic axonal damage affecting only the corpus callosum or central white matter, while severe traumatic damage, widespread enough to be described as DAI (see Discussion), was present in two. Distinguishing between terminal hypoxic–ischaemic and traumatic damage to axons (Geddes *et al*., 2001) was not always easy, but in a few cases both pathologies appeared to be present. Positivity in one case could not be assessed because of the state of preservation of the brain.

In eight other cases, βAPP immunohistochemistry revealed axonal bulbs in the brainstem, anatomically localized to the corticospinal bundles on both sides of the caudal pons and medulla (Fig. 2); in seven out of eight cases this was the only axonal βAPP expression detected in the brain. In some of the cases the bulbs in the long tracts were readily detectable on haematoxylin and eosin staining, and in one a microglial reaction was present round them (Fig. 3) suggesting that they were of several days' duration (Geddes *et al*., 1997). In three further cases axonal damage was detected in the cervical spinal cord and/or dorsal nerve roots.

Control group

Two cases displayed vascular axonal damage in central white and, to a lesser extent, in deep grey matter. In one of these, a child with severe gastroenteritis, there was raised intracranial pressure caused by brain swelling. The other was a child born at 36 weeks who had severe perinatal hypoxia–ischaemia and who lived for only 2 days. There was no βAPP expression in either corticospinal tract tracts or axons in the cervical cord in any of the controls; indeed, there was no axonal damage, either recent or old, that might be interpreted as being traumatic.

Other microscopic findings

In the NAI group, severe widespread neuronal hypoxia was seen in 29 out of 37 cases (78%), of which eight had no documented survival, having been reportedly found dead. In the control group, one case, the premature child who suffered

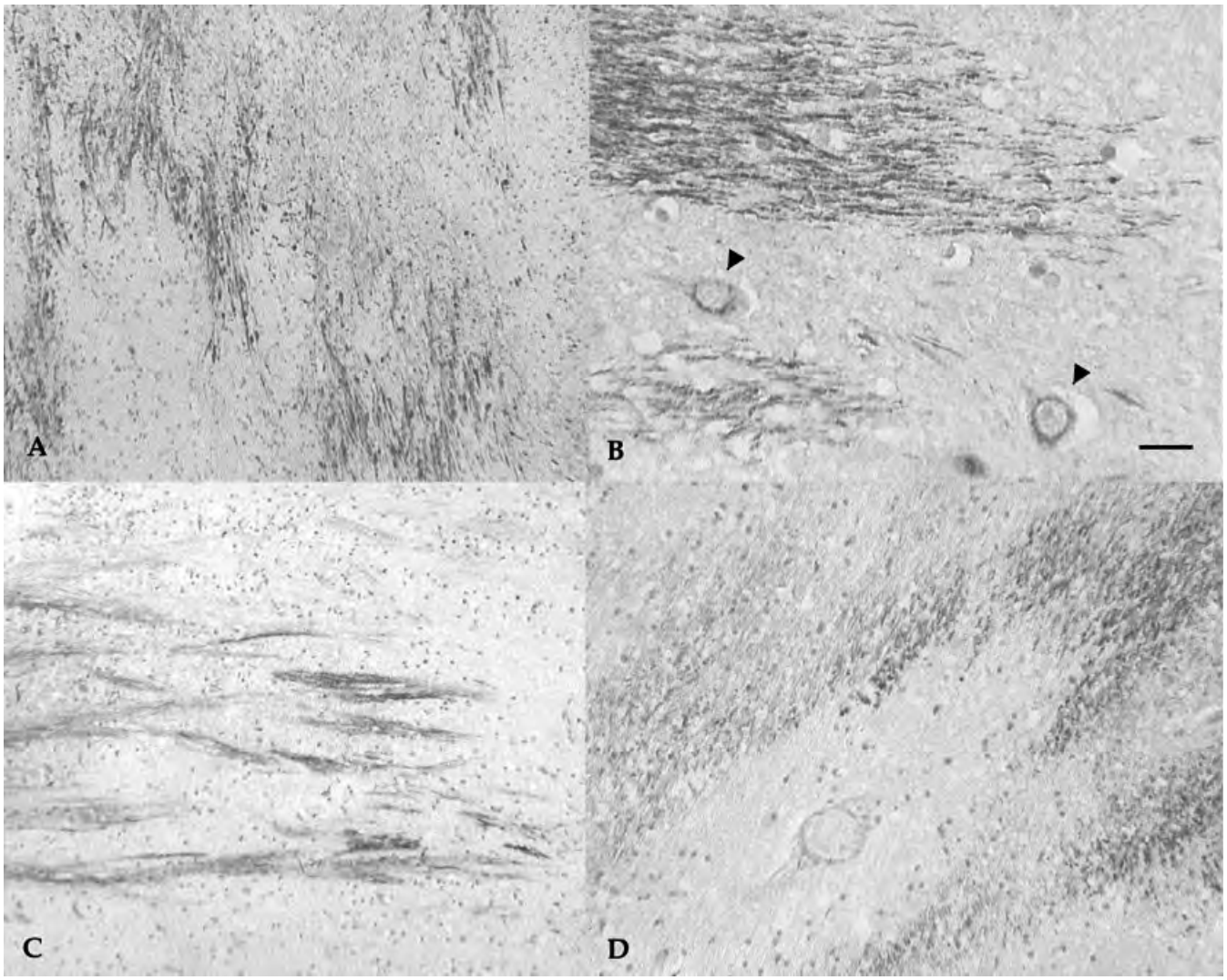

Fig. 1 Patterns of βAPP immunoreactivity interpreted as vascular, seen in three low power views (**A**, **C** and **D**) and at higher magnification in the deep grey matter (**B**). (**A**) Geographic staining, outlining areas of ischaemia in the centrum semiovale; (**B**) bundles of diffusely reactive axons running through the putamen [the two neurones in the lower half of the field (arrowheads) also show cytoplasmic expression of βAPP]; (**C**) olivocerebellar fibres just to one side of the midline in the medulla, in a grossly swollen brain; (**D**) perivascular staining. Bar = 50 μm. Reproduced with permission from Geddes and Whitwell (2001).

Table 1 *Outline details of 37 infants (<1 year) with inflicted head injury*

Factor	No. of days
Age at head injury (n = 37)	
Median	73
Mean	97
Range	20–273
Survival (n = 37)	
Median	1
Mean	27.7
Range	0–695

perinatal asphyxia, showed severe hypoxic changes. Two further NAI cases had changes throughout the brain, although of a milder degree.

Literature review

Five studies of NAI were found in which there had been a microscopic examination of the brain (Lindenberg and Freytag, 1970; Calder et al., 1984; Vowles *et al*., 1987; Shannon *et al*., 1998; Gleckman *et al*., 1999). Outline details of these papers, including the authors' principal conclusions, are given in Table 3.

Axonal damage described in the studies of Calder and Vowles (most of whose cases are the same) did not fulfil criteria for a diagnosis of DAI—by definition, damage to cerebral white matter and the upper brainstem (Adams *et al*., 1989; Adams *et al*., 1991). Shannon and co-workers found widespread hypoxia–ischaemia in their NAI cases, and concluded that altered cerebral perfusion, not trauma, was responsible for widespread axonal pathology. Localized axonal damage in the high cervical cord in their head-injured

Table 2 *Principal details of the 37 infants in this series*

No.	Sex	Age at injury (years)	History of presentation	Survival time	Significant extracranial injury	Skull fracture	Raised ICP	Acute SDH	Other significant neuropathology	Diffuse axonal injury	Vascular axonal injury	Global hypoxia–ischaemia	Evidence of neck injury*	Retinal haems.
1	F	16 weeks	Apnoea	5 days	Old rib fractures	No	Yes	Yes	Contusions; tears	No	Yes	Yes	No	Bilat.
2	M	2 months	Apnoea	None §	Old rib fractures	Yes	No	No		No	No	No	Yes	No
3	M	7 weeks	Thrown	None §	Recent fracture femur	Bilat.	Yes	Yes		No	Yes	Yes	No	No
4	M	4 months	Dropped	None	No	Bilat.	Yes	No	Contusions; tear	No	No	Yes	No	N/E
5	M	2 months	Found dead	None	No	Yes	Yes	Yes		No	No	No	Yes	N/E
6	M	20 days	Apnoea	None §	No	No	Yes	Yes		No	No	Yes	Yes	No
7	F	4 months	Apnoea	24 hours	Mesenteric bruising	Bilat.	Yes	Yes		No	Yes	Yes	No	Bilat.
8	M	2 months	Apnoea	5 months	Recent rib fracture	No	*Not at death*	No		No	No	Yes	No	N/E
9	F	2 months	Found dead	None §	Extensive bruising	No	No	Yes		No	No	Yes	Yes	Bilat.
10	F	2 months	Apnoea	2 days	No	Yes	Yes	Yes		No	Yes	Yes	Yes	Bilat.
11	M	6 weeks	Apnoea	6 days	No	No	Yes	Yes		No	Yes	Yes	No	Bilat.
12	M	5 weeks	Apnoea	2 days	Recent rib fracture	Yes	Yes	Yes		No	Yes	Yes	Yes	Bilat.
13	F	7 weeks	Apnoea	3 days	Old rib fractures	Yes	Yes	Yes		No	Yes	Yes	Yes	Bilat.
14	M	2 months	Apnoea	None §	No	No	Yes	Yes		No	Yes	Yes	No	Bilat.
15	F	5 weeks	Apnoea	23 months	No	Yes	*Not at death*	Yes		No	No	No	No	Bilat.
16	F	10 weeks	Apnoea	6 days	No	No	Yes	Yes		No	No	Yes	*No brainstem*	Bilat.
17	M	4 months	Apnoea	3 days	No	No	Yes			No	No	Yes	No	N/E
18	M	3 months	Dropped	None §	Old fractures tibia and femur	Bilat.	Yes	No	Tears	Yes	Yes	No	Yes	No
19	F	9 weeks	Found dead	None §	Ruptured liver; old rib fractures	Yes	Yes	Yes		No	No	Yes	Yes	Bilat.
20	F	3 months	Apnoea	None §	No	No	Yes	Yes		No	No	Yes	No	Bilat.
21	F	9 months	Apnoea	2 days	No	No	Yes	Yes, SOL		No	No	Yes	No	Bilat.
22	F	12 weeks	Apnoea	9 hours	No	No	Yes	Yes		No	Yes	Yes	No	Bilat.
23	F	5 months	Apnoea	2 days	No	Bilat.	Yes	Yes		No	No	Yes	Yes	Bilat.
24	M	7 weeks	Apnoea	3 days	No	No	Yes	Yes		No	Yes	Yes	No	Bilat.
25	F	5 weeks	Apnoea	6 days	Old rib fractures	No	Yes	Yes	ICH	?	?	Yes	?	Bilat.
26	F	3 months	Apnoea	2.5 months	Old fracture clavicle	No	*Not at death*	Yes	ICH	No	No	Yes	No	No
27	F	4.5 months	Apnoea	11 days	Old rib fractures	Yes	No	Yes	Tear	No	No	Yes	No	N/E
28	M	8 months	Apnoea	2 days	No	No	Yes	Yes, SOL		No	No	Yes	No	Bilat.
29	M	6 months	Fall	12 hours	No	No	Yes	Yes		No	Yes	Yes	No	Bilat.
30	M	6 months	Apnoea	24 hours	No	No	Yes	Yes		No	No	Yes	No	Bilat.
31	M	3 weeks	Apnoea	None §	No	Yes	Yes	No		No	No	Yes	No	No
32	M	6 weeks	Apnoea	None	Recent rib fractures	No	Yes	Yes		No	No	No	No	No
33	F	5 months	Apnoea	24 hours	No	Bilat.	Yes	Yes		Yes	No	Yes	Yes	No
34	F	7 months	Apnoea	None §	No	No	Yes	Yes		No	Yes	Yes	Yes	No
35	F	8 weeks	Apnoea	4 days	No	No	Yes	No		No	No	Yes	No	N/E
36	F	7 months	Dropped	3 days	Old rib fracture	Yes	No	Yes		No	No	Yes	No	Bilat.
37	M	5 weeks	Found dead	None §	No	No	Yes	Yes		No	No	No	Yes	N/E

The figures for ages at injury and survival times have been rounded up: exact ages and survival times were used for Table 1. Terms used: apnoea = any case in which the carer described the child turning blue, having breathing difficulties, gasping or stopping breathing; old = injuries antedating the terminal head injury; evidence of neck injury = axonal injury to craniocervical junction, and/or bleeding around the cord at that point. *Note that the spinal cord was not fully examined in five cases. Bilat. = bilateral; SOL = mass lesion; N/E = not examined by a pathologist; SDH = subdural haemorrhage; EDH = extradural haemorrhage; 'None §' = cases in which, on histological grounds, there had clearly been survival after an insult (see text); ICP = intracranial pressure; ICH = intracranial haemorrhage. The state of preservation of the brain in Case 25 made it impossible to assess the immunohistochemistry.

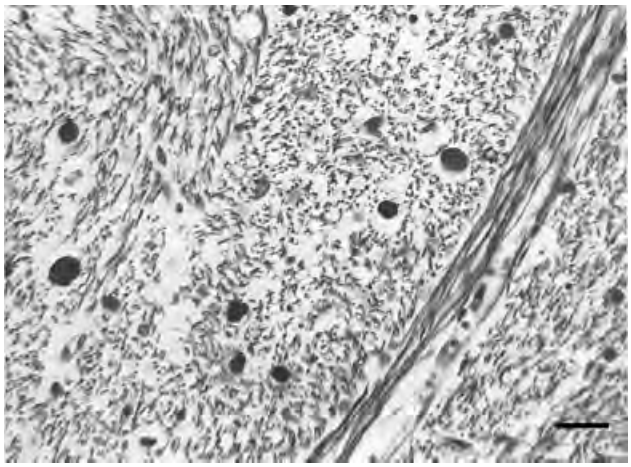

Fig. 2 A number of axonal bulbs seen in the corticospinal tract in the mid-pons in a 2-month-old boy (silver preparation). Bar = 30 μm.

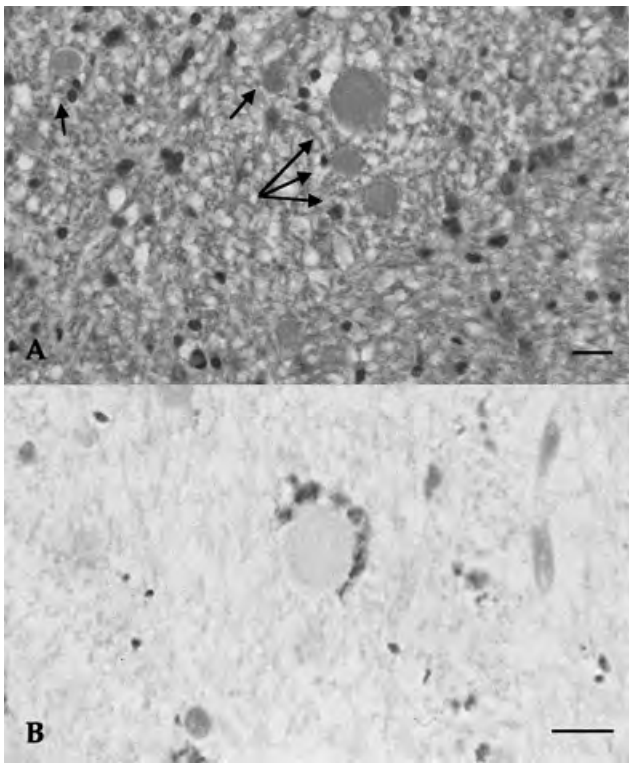

Fig. 3 (**A**) Several bulbs (arrows) are visible on a haematoxylin and eosin stain in one of the pyramids in the medulla of the same case as shown in Fig. 2. (**B**) With immunocytochemistry, a microglial reaction was seen around some of the bulbs (anti-CD68/PG-M1), suggesting that they had been present for a few days. Bars = 25 μm.

cases, also present, was attributed to trauma. Gleckman's series detected axonal damage, described as DAI, in the majority of their NAI cases. The role of brain swelling and hypoxia–ischaemia was not, however, fully addressed by this study (see Discussion).

Discussion

Head injury is a particular feature of non-accidentally injured infants, in whom it is commonly the cause of death. From a clinical point of view, the picture after inflicted head injury is quite stereotyped in severe cases: the infant arrives at hospital collapsed or moribund, having suffered an episode of apnoea or cardiorespiratory arrest, requiring resuscitation and ventilation (Johnson *et al*., 1995; Duhaime *et al*., 1998). Fundoscopic examination frequently shows bilateral retinal haemorrhages of variable severity. The principal abnormality detected by CT scan is that of a diffusely swollen hypoxic brain: the acute subdural bleeding that is almost invariably present in such cases is frequently trivial in terms of the quantity of blood in the subdural space, and rarely necessitates neurosurgical intervention (Brown and Minns, 1993; Duhaime *et al*., 1998); indeed, it may not be detected by neuroimaging (Hart *et al*., 1996; Feldman *et al*., 1997). Such a child is assumed to have diffuse brain damage which, together with brain swelling, accounts for the loss of consciousness and prolonged coma. The immediate mode of death is raised intracranial pressure.

The first reports in the literature that attempted to investigate the nature of the diffuse brain damage in NAI were two papers looking at the same cases (Calder *et al*., 1984; Vowles *et al*., 1987), the second of which by Vowles, who is one of the authors of this paper, has been widely quoted. Investigating the optimal silver method to demonstrate injury to thinly myelinated infant axons, Vowles and colleagues found axonal bulbs in six out of 10 cases and concluded that DAI occurred in inflicted infant head injury. However, the paper was published before the routine use of βAPP immunocytochemistry had made clear that there is a wide spectrum of traumatic axonal damage, from very mild to very severe, and that not all axonal injury is 'DAI' (Geddes *et al*., 2000): in their cases, lack of involvement of the brainstem would imply far less severe axonal damage (Geddes *et al*., 1997, 2000). It is difficult to make any further comment about the findings of this study, partly because the extent of sampling of the cases is not given, and partly because there is no clinical information about what are now recognized to be relevant factors—mechanical ventilation, brain swelling, raised intracranial pressure and so forth—all potential causes of axonal damage (Geddes *et al*., 2000). The papers of Calder and Vowles were the only ones on the subject for 10 years, and the idea that DAI was a feature of shaking injury became entrenched in the literature.

The question of whether DAI or lesser degrees of traumatic axonal damage occur in infant head injury is not just a matter of semantics. A diagnosis of 'DAI', which by definition is traumatic damage (Geddes *et al*., 1997, 2000), means very severe brain injury, caused by angular or rotational acceleration of high magnitude; in the context of shaking a young baby, it would imply extremely violent forces to the brain. The forces necessary to cause DAI are of such magnitude that some authors have questioned whether shaking

Table 3 *Published series dealing with microscopic findings in the brains of NAI cases*

Study	No. of cases and ages	Criteria for diagnosis of NAI?	Method for axonal damage	Widespread sampling?	Controls	Authors' principal conclusions
Lindenberg and Freytag, 1969	16 ⩽5 mo	No	Silver	Not stated	No	Contusional tears, caused by impact, characteristic lesion in infants <5 months of age
Calder *et al.*, 1984	9 ⩽3 mo[†] 3 <2 years	No	Silver	10/12	10 (no details)	1. Contusional tears in seven out of nine cases. No axonal injury in infants 2. 'White matter damage similar to that seen in adults' in the three older children
Vowles *et al.*, 1987	9 ⩽3 mo[†] 1 >4 mo[†]	No	Silver	Not stated	No	DAI in hemispheres and corpus callosum, but not in brainstem in 6/10, in association with skull fractures and contusional tears
Shannon *et al.*, 1998	11 <1 year 3 >18 mo	Yes	βAPP	'Where available'	7 HIE 6 normal	1. Hypoxic–ischaemic changes in 14/14 NAI cases 2. βAPPpositive axons in 14/14 NAI and in 6/7 HIE controls 3. βAPP-positive axons in cervical cord roots in 7/11 NAI cases
Gleckman *et al.*, 1999	10 ⩽10 mo	No	βAPP	'Usually'	7*	DAI in 5/7 shaken cases and 2/3 cases with impact; none in controls

Terms and abbreviations: widespread sampling = blocks from cerebrum and brainstem; mo = months; HIE = hypoxic–ischaemic encephalopathy. *Two controls known to have died instantly, survival time not available for two others. [†]Same cases.

alone can cause it, without some form of impact to the head as well (Duhaime *et al*., 1987). Despite this, papers and review articles on the 'shaken-baby syndrome' have tended to cite DAI as one of the inevitable and devastating sequelae of shaken-baby or 'shaken-impact' syndrome (Brown and Minns, 1993; Munger *et al*., 1993; David, 1999).

Two recent studies have used immunocytochemistry for βAPP, a sensitive marker of axonal damage (Gentleman *et al*., 1993; McKenzie *et al*., 1996), to re-address the question of axonal pathology in NAI (see Table 3). Shannon's study demonstrated widespread axonal damage secondary to hypoxia–ischaemia, as well as localized axonal damage suggestive of stretch injury to the cord, while Gleckman described the widespread βAPP expression in his cases as DAI. The principal drawback of the latter paper is that it does not properly address the question of brainstem damage due to raised intracranial pressure, because in the only control cases in which the brain was swollen, tissue sampling was inadequate: blocks of midbrain and pons, which in a swollen brain commonly express βAPP, were not available. In addition, the paper illustrates patterns of staining that we would interpret as typical of axonal damage due to brain swelling and raised intracranial pressure. This is an important criticism, for the lesson from neuropathological studies of adult head injury is that interpretation of axonal damage in cases of short survival is not easy, and requires extensive sampling using large blocks, with full clinical information (Geddes *et al*., 2000).

Our series appears to be the largest neuropathological study of non-accidental infant head injury, and we can confirm that axonal damage occurs in the brains of both head-injured subjects and in controls in much the same distribution, and with similar appearances to those described and published by Shannon and Gleckman. This is not 'DAI', but diffuse vascular or hypoxic–ischaemic injury, attributable to brain swelling and raised intracranial pressure. Despite the fact that our series of 37 infants probably includes three cases that did not survive long enough for any axonal injury to be detectable (see Results), our findings strongly suggest that severe traumatic axonal damage is a rarity in infant NAI unless there is considerable impact, and that the diffuse brain damage responsible for loss of consciousness in the majority of cases is hypoxic rather than traumatic. And while one might tend to dismiss the statements of carers in child abuse cases, the story of respiratory abnormalities or apnoea recurs with great regularity in the clinical notes of the infants. Apnoea may well be, as has been suggested by others (Johnson *et al*., 1995), an integral part of many severe cases of non-accidental infant head injury or shaken-baby syndrome. The hypoxic damage resulting from apnoea would lead to severe brain swelling, which is the usual cause of death. Such a sequence of events fits the observed clinical and neuropathological features in most NAI cases more closely than the alternative explanation that brain swelling is the result of reactive hyperaemia or deranged cerebral autoregulation. It is noteworthy that none of our control cases who were reported to have presented with respiratory abnormalities showed changes as severe as the NAI group.

So what causes apnoea and collapse in young babies who have had a head injury? One candidate would be damage to

the lower brainstem or upper cervical spine (Johnson *et al*., 1995). Infants and young children have been shown to be susceptible to high cervical cord injury without radiological evidence of bony injury, as a result of which they may suffer apnoea and cardiorespiratory arrest, or severe hypotension (Bohn *et al*., 1990). In a rather unusual study, 25% of 199 infants undergoing manipulation of the craniocervical region by a chiropractor reacted with apnoeic attacks to the stimulus (Koch *et al*., 1998). One attempt to find brainstem or high cervical cord damage in cases of NAI with MRI failed to detect abnormalities (Feldman *et al*., 1997). However, autopsy studies of NAI cases have described bleeding into paraspinal muscles, epi- or subdural haematomas at the cervicomedullary junction, and occasionally macroscopic lesions in the upper cervical cord segments (Hadley *et al*., 1989; Johnson *et al*., 1995; Hart *et al*., 1996; Leetsma, 1997; Saternus *et al*., 2000). Such reports, together with the microscopic cord findings described by Shannon (Shannon *et al*., 1998), suggest that in a significant proportion of cases the craniocervical junction can be shown to be damaged at autopsy, if carefully examined.

In their study, Shannon and his colleagues demonstrated for the first time axonal damage in the high cervical cord. They also recorded βAPP expression in a number of dorsally situated brainstem tracts, in both shaken infants and hypoxic–ischaemic controls. In our cases, we too saw foci of ischaemic-type staining in the dorsal brainstem, particularly the lower medulla, in both NAI cases and controls. However, the staining in the corticospinal tracts was quite distinct, affecting variable numbers of axons in these fibre bundles bilaterally, and appeared to represent localized traumatic axonal injury at the craniocervical junction. We believe that this pattern results from non-disruptive stretch injury to the neuraxis. Corticospinal tract damage has been suspected in cases of adult cervical hyperextension injury (Riggs and Schochet, 1995), and demonstrated in a few cases which have been neuropathologically examined, where the corticospinal fibres in the pons and medulla were preferentially involved (Lindenberg and Freytag, 1970; Hardman, 1979; Geddes *et al*., 2000). The mechanism has been discussed in some detail by Lindenberg, who drew attention to clinical signs resulting from stretching of corticospinal tracts in hyperextension injuries, particularly at the level of the pyramids (Lindenberg and Freytag, 1970).

The anatomical features that make head injury in infants biomechanically unique are well known. The immature skull is pliable, not rigid, and the conditions necessary to fracture it will be different from those in older subjects (Lancon *et al*., 1998). High head : body ratio means that in a very young baby a large mass is pivoted by the neck. Underdeveloped neck muscle tone and an inherently elastic spinal column, in which the juvenile arrangement of facet joints and upper cervical vertebral bodies renders the cord particularly vulnerable to both flexion and extension injuries, are also important factors (Kriss and Kriss, 1996). Although mechanisms of shaking must vary (Jones, 2000), and nobody really knows how babies are injured, it may not be necessary to shake an infant very violently to produce stretch injury to its neuraxis. It is true that the more vigorous the shaking, the greater the stretch that would take place at the extremities of movement, and the worse the damage produced. In all of our cases in which there was axonal pathology at the craniocervical junction, the damage was survivable; what was life-threatening was the hypoxic injury and brain swelling that resulted. Seen from this point of view, the debate over shaken versus shaken–impact becomes irrelevant, and because there is no DAI it is possible that the severe acceleration–deceleration injury that is so often cited does not in fact occur in shaken-baby syndrome. We have discussed elsewhere (Geddes *et al*., 2001) the fact that subdurals in infants are strikingly different from those in adults, who do not commonly get retinal haemorrhages with a head injury. This discrepancy, together with the lack of widespread microscopic damage due to trauma, suggests that beliefs about the conditions that produce these haemorrhages in infants, inferred as they often are from adult head injury, require fresh examination.

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BRIEF COMMUNICATION

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Interval Duration Between Injury and Severe Symptoms in Nonaccidental Head Trauma in Infants and Young Children*

REFERENCE: Gilliland MGF. Interval duration between injury and severe symptoms in nonaccidental head trauma in infants and young children. J Forensic Sci 1998;43(3):723–725.

ABSTRACT: Forensic pathologists are frequently asked to describe the interval between injury and the onset of symptoms in child abuse head injury deaths. A prospective, postmortem study examined the interval between injury and onset of symptoms in 76 head injury deaths in which this information was available. The head injury deaths were divided by mechanism of injury. The mechanisms were shake (no impact), combined shake and blunt impact, and blunt impact (no history of shaking). The interval was less than 24 hours in 80% of shakes, 71.9% of combined, and 69.2% of blunt injuries. The interval was greater than 24 hours in more than 25% of each of these latter groups and was more than 72 hours in four children. The variable intervals between injury and severe symptoms warrant circumspection in describing the interval for investigators or triers of fact. It should be noted that in all of the cases where information was supplied by someone other than the perpetrator, the child was not normal during the interval.

KEYWORDS: forensic science, child abuse, head injury, interval to symptoms

As more head injury child deaths are recognized as abusive and therefore investigated, forensic pathologists are more frequently asked to determine the time of injury. This information is used to identify or exclude possible perpetrators. Many forensic pathologists have had the experience of investigating several such deaths and finding that the interval between injury and presentation is brief. In 1995 Nashelsky and Dix found minimal data to substantiate or contradict the concept that the interval is very short (1). Howard, Bell and Uttley reported the intervals from injury to neurosurgical evaluation for 28 children with subdural hemorrhage in 1993 (2). They found two of the three children with documented shaking injury had intervals within 24 hours but the third was 72 hours. For the other 25 infants with subdural hemorrhage 13 presented in 24 hours, three in 24–72 hours, and nine after more than 72 hours. The present study was undertaken to examine the interval from injury to symptom onset.

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Methods

A prospective, postmortem study investigated 169 child deaths and examined this interval in the 76 head injury deaths with such information available. These children with head injuries are a subset of a larger group of children reported previously (3).

Sample Selection

One hundred seventy-five of nearly 400 deaths of young children investigated at the Dallas County Medical Examiner's Office from 1982 to 1989 were studied prospectively. Case selection depended on random assignment of cases and on the prosector's willingness to participate in the study. Nineteen pathologists contributed one or more cases each by the end of case collection. All child deaths were equally likely to be included in the study. The deaths included diagnoses of child abuse, suspected child abuse, apparent accidental trauma, and apparent natural death. History, autopsy findings, and ocular findings were gathered and reviewed for the more general study. Children whose immediate cause of death was head injury were selected to examine the interval from injury to severe symptoms.

Symptom Onset Definition

The onset of severe symptoms was identified as the time when an external event occurred or the caretaker called for medical assistance. In these young children the symptoms were extreme: unresponsiveness, difficulty breathing, cardiorespiratory collapse. The persons identifying the symptoms were usually the caregiver calling or presenting for emergent medical attention. In some cases the identifiers were persons witnessing an external event such as a motor vehicle collision. The need for emergent medical attention was confirmed by the health care workers who evaluated the children and found them unresponsive, commonly without vital signs or with failing vital signs.

Mechanism of Injury

The deaths caused by head trauma were divided by mechanism of injury as described previously (4). The factors used in the definition included: finger marks or rib fractures; history of shaking; subdural and/or subarachnoid hemorrhage; and evidence of impact (contusions, subscalpular hemorrhage, skull fractures). The mechanisms so defined were shake (no impact with two of the following—finger marks or rib fractures, subdural or subarachnoid hemorrhage, history of shaking), combined shake and blunt impact

(impact with finger marks or rib fractures, history of shaking), and blunt impact (no finger marks or rib fractures, no history of shaking).

Results

Forty-six percent were less than one year old, 22% were between one and two years of age, and 32% were over two years of age. Forty-two were white; 24 were black; 7 were of Hispanic origin; and 3 were of other ancestry. Forty-one of the 76 children were male.

Five of the infants had exclusive shaking mechanism of injury. Both shaking and blunt mechanisms were identified in 32 infants and children. Exclusively blunt mechanisms of injury were identified in 39 of the infants and children.

The interval was less than 24 hours in all but one of the five shaken infants. It was less than 24 hours in 71.9% of 32 infants with combined, and 69.2% of 39 with blunt injuries (Table 1). The interval was greater than 24 hours in more than 25% of the groups with a blunt force component and extended more than 72 hours in four children with blunt trauma as a part of the mechanism—one with combined shake and blunt mechanisms, and three with exclusively blunt mechanism.

The 22 cases with intervals longer than 24 hours were reviewed to determine if any symptoms had been described prior to the catastrophic collapse leading to death or brief hospitalization prior to death. Ten of these children were described as lethargic or otherwise abnormal during the interval. The other twelve were in the care of the presumed perpetrator and had no credible description of their condition.

These findings are depicted graphically in Fig. 1. The columns with no volume are the graphical representation of zero.

Discussion

The interval from injury to catastrophic or near-catastrophic collapse requiring medical attention, or death is observed to be short, less than 24 hours, in almost all the babies with shaking as the exclusive mechanism of injury. This correlates with our understanding of the effect of violent shaking causing global disruption of the nervous system. Diffuse axonal injury can be demonstrated if life support is maintained. The expression ''violent'' is appropriate, although some find it objectionable (5,6).

In this study some of the infants with blunt force as part or the exclusive mechanism of injury presented more than 24 hours after injury. Blunt injuries are not necessarily as immediately disruptive of the nervous system and brain functioning as violent shaking. Secondary phenomena including brain swelling and edema produce symptoms. Although brain swelling and edema can develop very rapidly, in less than 24 hours, delayed onset of symptoms is not uncommon.

TABLE 1—*Interval from injury to severe symptoms.*

Interval in Hours	Mechanism of Injury			
	Shake	Combined	Blunt	Total
Less than 24	4	23	27	54
24 to 48	1	8	6	15
48 to 72	0	0	3	3
More than 72	0	1	3	4
Total	5	32	39	76

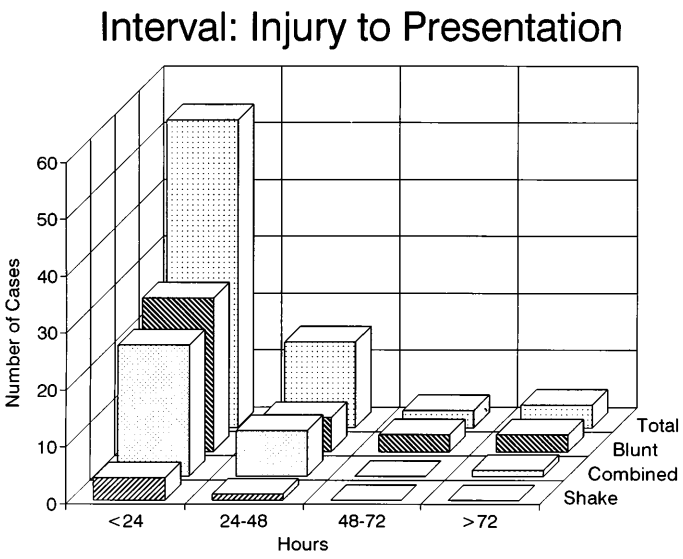

FIG. 1—*The graphic display confirms the impression that most of the children will present with severe symptoms in an interval of less than 24 hours after injury.*

The proportion of children presenting beyond 24 hours was not as great as found by Howard et al. (2) in their retrospective clinical study of 28 infants and young children identified as having subdural hematoma after presenting for neurosurgical evaluation. Six of the children in their study died within a week of hospitalization, and two others 8 and 9 years later. No autopsy information was provided. Nine of the children survived neurologically intact (2). Thus, the cases of Howard et al. were not as severely injured and do not serve as a comparable group for fatally injured children.

Conclusion

Enough variability in the interval between injury and the time of severe symptoms or presentation for medical care in fatally injured children exists to warrant circumspection in describing such an interval for investigators or triers of fact. Our data indicate that the interval is brief (less than 24 hours), in almost $^{3}/_{4}$ of cases of head injury death, especially in shaking injuries. However, in more than $^{1}/_{4}$ of the cases, the interval from injury to the onset of severe symptoms is longer. In all cases in which the children were seen by an independent observer after injury, they were described as not normal.

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EXPERIENCE AND REASON—Briefly Recorded

"In Medicine one must pay attention not to plausible theorizing but to experience and reason together. . . . I agree that theorizing is to be approved, provided that it is based on facts, and systematically makes its deductions from what is observed. . . . But conclusions drawn from unaided reason can hardly be serviceable; only those drawn from observed fact." Hippocrates: *Precepts. (Short communications of factual material are published here. Comments and criticisms appear as Letters to the Editor.)*

Retinal Hemorrhage After Cardiopulmonary Resuscitation in Children: An Etiologic Reevaluation

Retinal hemorrhage detected after cardiopulmonary resuscitation has important medical, social, and legal implications. When a child is brought to the hospital in circulatory arrest, these hemorrhages raise the question of preceding trauma, frequently child abuse.[1] Several authors have suggested that retinal hemorrhage may be virtually pathognomonic of child abuse.[2,3] We have questioned this belief because our experience, as well as that of others,[4,5] suggests that retinal hemorrhage may result from resuscitative efforts. We therefore undertook a prospective study to evaluate whether cardiopulmonary resuscitation can cause retinal hemorrhage.

MATERIALS AND METHODS

During a 4-month period, all children admitted to the Pediatric Critical Care Medicine Service who underwent cardiopulmonary resuscitation were considered for inclusion in the study. Patients who met the following criteria were included: (1) chest compression during resuscitation; (2) no historical or physical evidence of preceding trauma; and (3) no history of conditions known to be associated with retinal hemorrhage, including seizure disorders, hypertension, or bleeding diatheses. Age, weight, cause of arrest, and duration of chest compressions were recorded. Fundoscopic examination was performed by an experienced pediatric neurologist shortly after resuscitative efforts were completed. Positive findings were confirmed by two additional examiners. Pharmacologic dilation of pupils was used when necessary.

RESULTS

A total of 362 children were admitted to the Pediatric Critical Care Medicine Service during the 4-month study period. Of these, 20 children met the criteria for inclusion in the study.

The study sample had a median age of 2 years (range, 2 weeks to 17 years) and a median weight of 13 kg (range, 3 to 60 kg). The median duration of chest compressions was 26.5 minutes (range, 3 to 120 minutes). The causes of circulatory arrest included sepsis in five children, sudden infant death syndrome in five, near-drowning in three, asthma in three, poisoning in two, asphyxia in one, and aspiration in one (Table).

Of the 20 patients, 2 (10%) had retinal hemorrhages. They did not differ from the study sample in age, weight, duration of cardiopulmonary resuscitation, or cause of arrest. Their cases are summarized below.

Patient 1

A 2-year-old developmentally delayed girl was brought to the hospital by ambulance in cardiorespiratory arrest due to water immersion. The patient had been playing in a full bathtub of water while in the care of her 14-year-old sibling. From another room the sibling heard irregular (nonrhythmic) splashing for about 15 seconds followed by silence. She found the patient submerged face down 1 to 1½ minutes later. The child was limp, cyanotic, and apneic. The sibling did not attempt any resuscitative measures, but she removed the child from the tub. Paramedics arrived within 15 minutes. Cardiopulmonary resuscitation was initiated and continued for 40 minutes, the last 15 of which the patient was in the emergency

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TABLE. Clinical Characteristics of Study Patients

Age	Weight (kg)	Cause of Arrest*	Cardiopulmonary Resuscitation Duration (min)	Retinal Hemorrhages
2 wk	3.6	Sepsis	20	No
2 wk	3.7	Sepsis	17	No
1 mo	3.0	SIDS	120	No
1 mo	3.0	SIDS	75	Yes
1 mo	3.7	SIDS	15	No
2 mo	5.5	SIDS	105	No
7 mo	8.2	SIDS	45	No
1 y	12.0	Sepsis	20	No
1 y	8.8	Near-drowning	20	No
2 y	14.2	Near-drowning	40	Yes
2 y	14.5	Near drowning	3	No
6 y	20	Poisoning	60	No
7 y	18	Sepsis	25	No
11 y	32	Sepsis	25	No
12 y	30	Aspiration	30	No
14 y	48	Asthma	43	No
15 y	60	Asphyxia	28	No
15 y	51	Poisoning	25	No
15 y	51	Asthma	20	No
17 y	50	Asthma	40	No

* SIDS, sudden infant death syndrome.

department. Spontaneous circulation resumed. The patient was comatose, although brainstem reflexes returned. Severe bilateral aspiration pneumonia was evident upon arrival. Fundoscopic examination performed approximately 2 hours after immersion showed multiple large retinal hemorrhages bilaterally. Her coagulation profile was normal, with a platelet count of 250 000/mm^3, prothrombin time of 11.5 seconds, and partial thromboplastin time of 31.0 seconds. There was no history of hypertension, bleeding tendency, or seizure disorder. On day 4, the patient was declared brain dead and life support was discontinued. Skeletal survey, a complete forensic autopsy, and Children's Protective Services and police investigations all concluded that there was no preceding traumatic event.

Patient 2

A 6-week-old twin girl of an uncomplicated 36-week gestation was admitted to the hospital with fever, upper respiratory tract infection, and possible sepsis. During her hospital stay she ate well and behaved normally. However, 38 hours after admission, she was found pulseless and apneic in her crib. Attempts to resuscitate the infant continued for 75 minutes without success. Immediately after the termination of cardiopulmonary resuscitation, fundoscopic examination revealed a single fresh retinal hemorrhage 0.3 disc diameters in size at 1.2 disc diameters temporal to the right disc. At the time of admission, cerebrospinal fluid had been clear, with six red blood cells/mm^3, and blood, cerebrospinal fluid, and urine cultures had been negative. The platelet count was 212 000/mm^3 several hours before her death. With the exception of her nurse, no one had been seen in the infant's room for at least 4 hours preceding the arrest. Skeletal survey and complete autopsy showed no evidence of trauma. The final diagnosis was sudden infant death syndrome.

DISCUSSION

Chest compression from any cause raises intrathoracic pressure. Elevated intrathoracic pressure can directly cause increased retinal venous pressure and subsequent retinal hemorrhage. This occurs by two mechanisms: elevated jugular venous pressure and elevated intracranial pressure.

During the compression phase of standard cardiopulmonary resuscitation, the intrathoracic pressure is markedly increased. This pressure is transmitted to all vascular structures within the thorax. In previous work, we have shown that the average peak right atrial pressure in adults is 55 mm Hg.[6] Peak right atrial pressures of approximately 100 mm Hg have been recorded in an infant animal model.[7] In our experience with pediatric patients, mean right atrial pressures are commonly 35 to 40 mm Hg during cardiopulmonary resuscitation. In addition, simultaneous chest inflation with compression, which frequently occurs in clinical cardiopulmonary resuscitation, further increases the right atrial pressure.[8] Although functional venous valves at the thoracic inlet do exist, they do not completely protect the head and neck veins from transmission of high intrathoracic pressure.[9,10] During cardiopulmonary resuscitation in adults, the jugular venous pressures average 14 and 29 mm Hg in the relaxation and compression phases, respectively.[6] Pressure in some patients has exceeded 50 mm Hg with compression. Presumably, jugular

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venous pressures would be even greater during simultaneous compression and ventilation. Therefore, cardiopulmonary resuscitation can produce a marked pulsatile venous hypertension of the head and neck.

Intrathoracic pressure is transmitted partially to the intracranial vault during cardiopulmonary resuscitation by way of thoracic cerebrospinal fluid and the paravertebral venous plexus.[11] In animal cardiopulmonary resuscitation models, the mean intracranial pressure ranges between 22 and 30 mm Hg, with significantly higher pressures occurring during the compression phase.[7,12–14] In two children, the intracranial pressure rose to 30 and 50 mm Hg with compression.[15] Increased intracranial pressure has been shown to cause retinal venous hypertension and hemorrhage.[16] Retinal venous blood drains posteriorly through the central retinal vein (Fig. 1). The central retinal vein parallels the optic nerve and in the cranial vault is surrounded by cerebrospinal fluid within the subarachnoid space. The retinochoroidal anastomosis, which originates from the central retinal vein, allows retinal venous drainage extracranially into the facial veins. During intracranial hypertension, venous drainage through the central retinal vein is impeded. In addition, increased cerebrospinal fluid pressure in the subarachnoid space, which extends anteriorly along the optic nerve, will occlude the retinochoroidal anastomoses, thereby blocking all retinal venous drainage.

Increased intrathoracic pressure from various causes has been shown to induce retinal hemorrhage. Purtscher retinopathy is a hemorrhagic retinoangiopathy that results from a sudden increase in intrathoracic pressure. It has been documented after automobile accidents with compression of the chest by shoulder safety belts[17] and after prolonged Valsalva maneuvers such as childbirth.[18] Crush injuries to the chest, as in trampling during riots and in children whose chests have been rolled over by motor vehicles, have also been associated with retinal hemorrhage.[19]

Retinal hemorrhages are present in 20% to 50% of vaginally delivered newborns and in only 0.8% born by cesarean section.[20] Of infants born vaginally, retinal hemorrhages are almost exclusively seen in those delivered cephalad and not breech.[21] These observations can be explained as follows: The rise in intrathoracic pressure and accompanying rise in retinal venous pressure, which occur during vaginal delivery as the thoracic cage passes through the birth canal, do not occur during cesarean delivery. In breech deliveries, the infants do experience a rise in intrathoracic pressure and retinal venous pressure but the opposing intraocular pressure of the yet undelivered head prevents a significant venous transmural pressure gradient from developing and, therefore, prevents retinal hemorrhage.

Other authors have supported the theory that retinal hemorrhage could result from thoracic compression during resuscitative efforts. In 1978, Bacon et al.[5] described a 2-month-old boy found limp, pale, and apneic by his parent, who attempted to revive him with repeated back blows. The infant had extensive retinal hemorrhages, which were attributed to his parent's resuscitative efforts. Similarly, Kirschner and Stein[4] reported retinal hemorrhages discovered in a 3-month-old infant whose father had unsuccessfully attempted vigorous resuscitation by chest compression. The authors concluded that retinal hemorrhages are not invariably indicative of preceding trauma and should not be misinterpreted as child abuse.

In summary, a thorough evaluation that includes historical, clinical, and radiologic data, as well as an autopsy when appropriate, is warranted to exclude occult trauma as the cause of retinal hemorrhage after a circulatory arrest. Once trauma is excluded, we propose that increased intrathoracic pressure during cardiopulmonary resuscitation causes sufficient retinal venous hypertension to result in retinal hemorrhage. Therefore, caution should be taken when using retinal hemorrhage as exclusive evidence of preceding trauma or child abuse in children who have received chest compressions for circulatory arrest.

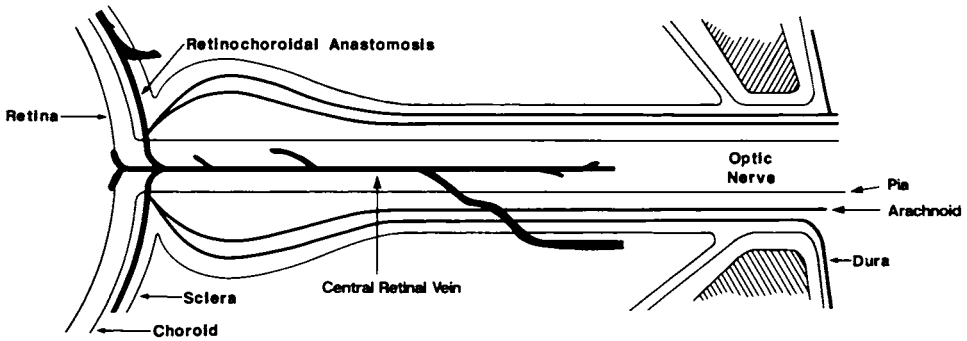

Fig 1. Relationship between retinal venous drainage and subarachnoid space. Modified from Muller and Deck,[16] with permission.

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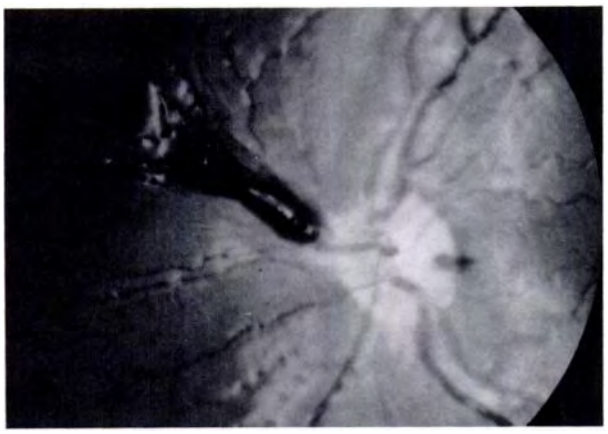

Fig 2. Retinal hemorrhages in the 3-o'clock and 10-o'clock positions at the disc margins.

ADDENDUM

After our series was completed, we saw a 9-year-old boy who had two episodes of cardiac arrest due to asthma on the day of admission. Minutes before his third arrest he had no retinal hemorrhages, and his right atrial pressure did not exceed 18 mm Hg. He then became hypotensive and bradycardic due to postresuscitation cardiomyopathy. Cardiac compressions lasted 12 minutes, producing mean right atrial pressures of 37 to 39 mm Hg. After return of spontaneous circulation, two retinal hemorrhages were seen in the left fundus (Fig. 2). Clotting studies and a platelet count were repeated and found to be normal.

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Infantile Subdural Haematoma and its Relationship to Whiplash Injuries

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British Medical Journal, 1971, 2, 430–431

Summary

Subdural haematoma is one of the commonest features of the battered child syndrome; yet by no means all the patients so affected have external marks of injury on the head. This suggests that in some cases repeated acceleration/deceleration rather than direct violence is the cause of the haemorrhage, the infant having been shaken rather than struck by its parent. Such an hypothesis might also explain the remarkable frequency of the finding of subdural haemorrhage in battered children as compared with its incidence in head injuries of other origin, and the fact that it is so often bilateral.

Introduction

Subdural haematoma is a commoner complication of head injury in infancy than at any other age. Lewin (1966) reported 47 cases of subdural haemorrhage in an unselected series of 1,750 head injuries occurring in patients of all ages—an incidence of about 2·7%—but Hendrick *et al.* (1964) found 5·2% in a large series of head-injured children ranging in age from birth to 15 years, the excess over Lewin's series being entirely accounted for by a greatly increased frequency in infants aged under 2 years, and particularly in those under 6 months, in which latter group it was as high as 20%. But there is no report of an incidence of subdural haematoma complicating road traffic accidents, etc., in very young children which approaches the 42% quoted by Weston (1968) in his series of necropsies of fatal cases of child assault, which is a particularly remarkable figure when one considers that the children in Weston's series ranged in age from 2 months to 5 years, with an average of 24 months—that is, considerably older than the high-risk group of Hendrick *et al.*—and that in some of them a head injury did not seem to have been noticed before death.

The commonest cause of infantile subdural haemorrhage is rupture of one or more of the delicate bridging veins which run from the cerebral cortex to the venous sinuses, the mode of injury being either a single acceleration or deceleration due to a heavy moving object striking the head or the rapidly moving head being brought up against a stationary mass; multiple applications of force would increase the total strain on the bridging veins and might result in an increased incidence of rupture. But direct violence is not an essential part of the picture. It has been known for some time that chronic subdural haematomas commonly occur in adults after disproportionately slight head injuries, sometimes when there is no reason to suppose that the head was ever injured at all. Meredith (1951) reported one such case in an adult after a fall on to the buttocks, and a senior American neurosurgeon (German *et al.*, 1964) developed a subdural haematoma after his head had been jerked by the violent motion of the "bobsled" which he was riding at a fun fair. More recently Ommaya and Yarnell (1969) published two well-documented cases of subdural haematoma, in both of which the subject sustained a whiplash injury to the neck as a result of an automobile accident, the head itself not being injured at all. There was no immediate loss of consciousness and it was not until several days later that cerebral signs developed.

It is now submitted that the conditions which are known to exist in many cases of the battered child syndrome are particularly favourable to the production of subdural haematoma in infants by an essentially similar mechanism. Kempe *et al.* (1962) noted that in their experience the extremities of the child are often used as handles during an episode of rough treatment; "sometimes an arm is pulled to jerk a reluctant child to his feet, sometimes the legs are held while the tiny body is swung in a punitive way." In one of Caffey's (1946) cases of subdural haematoma the history is given in the following words: "Baby rolled off a table and his mother grabbed him by the forearm and jerked him in the air to prevent his fall." It seems clear that the relatively large head and puny neck muscles of the infant must render it particularly vulnerable to whiplash injury in this sort of situation. Moreover, since one would expect that the child is often grasped more or less symmetrically by chest or limbs the rotation-acceleration strains on the brain would tend to occur fairly symmetrically also, in an anteoposterior direction. This may be the reason why infantile subdural haematoma is even more often bilateral—for example, in 14 out of 18 cases (78%) in my earlier series (Guthkelch, 1953)—than subdural haematoma in adults, for which the proportion of bilateral cases does not exceed 50%.

One cannot say how commonly assault in the form of violent shaking rather than of direct blows on the head is the cause of subdural haematoma in infants who are maltreated by their parents. Possibly it will be found that the frequency of this mechanism varies between different nations according to their ideas of what is permissible, or at least excusable, in the treatment of children. Among the children mentioned in Weston's (1968) detailed records there were three—a 4-month-old girl, a 5-month-old boy, and a 2-year-old girl—in whom the cause of death was subdural haemorrhage, it being admitted by the assailant that the child had been violently shaken. In only one of these was there any mark of injury on the head, and this is described as a superficial bruise of the scalp, the baby's head having apparently struck the side of her cot, as it were, incidentally. Court (1969) quoted a mother's confession that she was in the habit of shaking her babies "in an insane rage."

Patients

Twenty-three cases of proved or strongly suspected parental assault on children all under the age of 3 years, and all except one under 18 months, were admitted to the Hull Royal Infirmary between June 1967 and May 1970. Subdural bleeding had occurred in 13 (57%) of these and was bilateral in 10, the only sort of injury which was recorded more often being bruising of the skin. One or more fractures of long bones were found in association with subdural haematoma in six cases.

There were eight children in whom a fracture of the skull was discovered, and six of these had a subdural haematoma. Of the seven children suffering from subdural haemorrhage who had no skull fracture five had no external marks of injury

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on the head either, though in one of these necropsy showed surface haemorrhages on the cerebral cortex, and another was found at operation to have extensive cortical bruising. Though 8 out of 16 battered babies with definite evidence of head trauma had a subdural haematoma, in 5 out of 13 cases of subdural haemorrhage no evidence of the application of direct violence to the head was forthcoming.

In two additional personal cases there was very strong reason indeed to suppose that the mechanism of production of the subdural haemorrhage had been by shaking rather than battering.

CASE 1

A 6-month-old boy was found by his father lying unconscious in his cot and having repeated convulsions. His mother was sitting beside him too shocked to speak or move. On admission to hospital he was found to be well nourished and well cared for, without any external marks of injury. He was stuporous and breathing heavily, with a temperature of 38°C. There was pronounced hyperreflexia of all limbs and a tense fontanelle. Immediate subdural taps showed almost solid haematomas on both sides, but despite the evacuation of these he died three days later.

At necropsy several cortical bridging veins were found to have been torn from their dural attachments near the falx cerebri and the surface of the underlying brain was contused, with some small lacerations. There were no other internal injuries of any sort though some mucopus was present in the upper respiratory passages. The mother eventually admitted that the baby had had several fits of coughing and said that she feared that he was going to choke. She therefore held him up and shook him several times in order to try to clear his throat, whereupon he went into convulsions.

In this case the possibility of compression of the thorax with a consequent rise in jugular venous pressure and rupture of the cortical veins is not excluded, but there were no signs of bruising of the chest wall or of the lungs, nor any rib fractures. It was felt that the mother's account was probably truthful and that the tragedy occurred because of her lack of realization of the damage that can result from rough handling of a small baby. It has indeed been repeatedly observed that the parents of battered children may handle them exceedingly clumsily, even when they are under observation during their visits to hospital.

CASE 2

A 6-month-old boy was admitted to hospital on account of vomiting and convulsions, the suspected diagnosis being one of meningitis. Again there were no external signs of injury and no fractures of the skull or long bones, but the combination of a tense bulging fontanelle and bilateral retinal haemorrhages gave rise to a suspicion of subdural haematoma, which was confirmed and duly treated. At this time no suspicion of parental violence was entertained, but soon after his discharge from hospital the patient's twin brother was admitted suffering from a fracture of the femur for which his parents could give no explanation, and shortly afterwards the patient himself was readmitted with recurrent subdural haematomas on both sides. This time there were oval bruises on each of his forearms which fitted the pads of the fingers and thumb of the examiner's hand when the limbs were gripped. The parent denied having struck or beaten him but eventually his mother admitted that she and her husband "might have" shaken him when he cried at night.

Comment

The mere absence of visible injury on the head does not exclude direct violence, for Weston (1968) pointed out that in fatal cases "examination of the galea frequently revealed numerous . . . haemorrhages . . . even in the absence of conspicuous external bruising, abrasion or laceration." In a non-fatal case there is, of course, no opportunity to expose and examine the deep layers of the scalp, particularly now that craniotomy has been almost completely abandoned in the treatment of infantile subdural haematoma. None the less, one has the impression that "a good shaking," is felt, at least by British parents, to be socially more acceptable and physically less dangerous than a blow on the head or elsewhere.

As applied to the complicated mechanics of the brain and its coverings, subjected to the many different forces which constitute the causes of head injury, a limited series of experiments with a simple model proves nothing. Nevertheless, the following device, for the idea of which I am indebted to Dr. A. K. Ommaya, who has used a similar one to demonstrate the value of head restraint in preventing cerebral commotion in automobile accidents, may be found instructive.

An ordinary round-bottomed litre glass flask is filled with liquid paraffin in which have been suspended a few spoonsful of desicated coconut. When this mixture has been agitated the flakes will remain stationary in a state of even dispersion for periods of several minutes at a time. The flask is completely filled, closed with a rubber bung, and held firmly by the neck. It will be found that the flakes can be more readily set in motion, and will continue to swirl about for longer, after shaking for a few seconds than after the hardest blow that can be delivered without breaking the flask.

The phenomena observed are essentially similar to the movements of the brain which have been shown in anaesthetized monkeys in which the scalp and the top of the calvarium had been replaced by a Perspex window and which were then subjected to sudden accelerations (Ommaya *et al.*, 1968).

Conclusion

It has been shown that there is a discrepancy between the frequency of subdural haematoma occurring in battered children and of the same condition complicating head injuries of other origin, the incidence in the former being unexpectedly high, though in most of those in whom there was no actual skull fracture there was not even clear evidence of the application of direct violence to the head. This suggests that when the head is not the main target of attack the likely mechanism of production of the haematoma is one in which repeated sheering strains of one sort or another are applied to the cranial contents.

It follows that since all cases of infantile subdural haematoma are best assumed to be traumatic unless proved otherwise it would be unwise to disregard the possibility that one of these has been caused by serious violence, repetition of which may prove fatal, simply on the basis that there are no gross fractures or other radiological bone changes in the limbs, nor any fractures of the skull. One must keep in mind the possibility of assault in considering any case of infantile subdural haematoma, even when there are only trivial bruises or indeed no marks of injury at all, and inquire, however guardedly or tactfully, whether perhaps the baby's head could have been shaken.

I wish to acknowledge the co-operation of Dr. M. G. Philpott and Dr. R. J. Pugh, who permitted me to quote the series of battered children admitted under their care to Hull Royal Infirmary, and also of Mr. W. Tuffnell and Mr. L. Hodge, of Hull University, who prepared the model.

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The Mortality of Childhood Falls

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Falls accounted for 5.9% of the childhood deaths due to trauma in a review of the medical examiner's files in a large urban county. Falls represented the seventh leading cause of traumatic death in all children 15 years of age or younger, but the third leading cause of death in children 1 to 4 years old. The mean age of those with accidental falls was 2.3 years, which is markedly younger than that seen in hospital admission series, suggesting that infants are much more likely to die from a fall than older children. Forty-one per cent of the deaths occurred from "minor" falls such as falls from furniture or while playing; 50% were falls from a height of one story or greater; the remainder were falls down stairs. Of children falling from less than five stories, death was due to a lethal head injury in 86%. Additionally, 61.3% of the children with head injuries had mass lesions which would have required acute neurosurgical intervention.

The need for an organized pediatric trauma system is demonstrated as more than one third of the children were transferred to another hospital, with more than half of these deteriorating during the delay. Of the patients with "minor" falls, 38% had parental delay in seeking medical attention, with deterioration of all. The trauma system must also incorporate the education of parents and medical personnel to the potential lethality of "minor" falls in infants and must legislate injury prevention programs.

Trauma is the leading cause of death in children. Falls, as a sub-component, most frequently result in emergency room visits (5) and are the fourth leading cause of trauma deaths (1). In order to suggest means of preventing this unnecessary loss of life in children, it is important to understand the specific mechanism of deaths due to falls. With this knowledge, improvement in treatment and the prevention of morbidity and mortality should be possible. We reviewed the autopsy records of children who died from falls in the county of Cook, Illinois, which encompasses the city of Chicago and many of its suburbs, to better clarify these issues.

METHODS

The records of pediatric deaths due to falls of the Cook County Medical Examiner's office from January 1983 through December 1986 were examined. The majority of patients had a full internal autopsy. Gross examination and fluoroscopy were used to determine the presence of fractures. The prehospital and hospital records of these patients are not kept with the autopsy reports due to legal reasons; however, a brief summary was available in most of the charts. During this 4-year time period, 30,843 persons of all ages were autopsied for unexplained deaths, of whom 2,066 (6.7%) were children 15 years of age or less. Of the pediatric deaths, 746 (36.1%) were due to trauma, with falls accounting for 44 (5.9%) of these deaths.

RESULTS

Falls were the seventh most frequent cause of trau matic death for all children (burns, 29%; pedestrians, 16%; abuse, 9%; gunshot wounds, 8%; drowning, 8%; motor vehicle passengers, 7%; falls, 5.9%) but the third leading cause in the group aged 1 to 4 years (burns, 42.5%; abuse, 12.1%; falls, 11.8%). Of the 44 children suffering falls, 13 (30%) were girls and 31 (70%) were boys; 31 (70%) were black, 12 (28%) were white, and 1 (2%) was oriental. Sixty-four per cent were 2 years of age or younger; 11% were older than 5 years of age. The majority (59%) of the falls occurred between May and August and 15 were from a window or balcony with a height of one story or greater.

Based on the distance of the fall, victims could be divided into three groups (Table I). The first group is composed of eight children who fell from heights of five stories or higher. All of these had multiple lethal injuries and 75% were dead at the scene (DOA). Five of these children (mean age, 2.8 years) accidently fell from open windows or gratings; the remaining three were older (mean age, 10 years) who either jumped or were thrown. None of these deaths were deemed preventable.

The second group consisted of 18 children who fell from a height four stories or less, but higher than 3 feet.

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Seventeen of these children had a lethal head injury; one of these had an associated lethal injury and two had associated nonlethal injuries. One child who fell from a tree died from a liver laceration. Seven had delays of greater than 4 hours before definitive treatment with documented deterioration of five children during this time. Two children died during operative intervention at community hospitals (no pediatric or trauma surgeon) of injuries usually salvageable. The mean age of this group of children was 2.4 years. None of these children were DOA.

The final group of 18 children suffered "minor" falls sustained while running or from falls from furniture (less than 3 feet). These children all died from head injuries without any associated injury. Nine children had a delay in definitive treatment of greater than 4 hours with deterioration of eight. Six of these involved delays on the part of the parents and three of the six also had a delay due to hospital transfer. The one DOA in this group was an 8-month-old girl who fell off a couch onto a hard wood floor and was dead on arrival at the hospital. Her autopsy revealed a large acute subdural hematoma.

Of the 36 children in the latter two groups, one third were transferred to another hospital (one child was then transferred to yet another hospital) for care; 56% of these patients were at the primary receiving hospital more than 3 hours. The primary reason for delay was failure to recognize the injury, or performance of a CT scan. Of the patients transferred, 67% showed signs of deterioration between data recorded at the scene and at the final hospital. As not all data were listed, however, this figure could actually be higher. Additionally, nine children were initially normal after their falls but did not seek medical care until there was neurologic deterioration (range, 1 hour to 3 days).

Of the 31 children who died primarily of head injury, 19 (61.3%) had mass lesions (subdural or epidural hematomas, some associated with edema and/or subarachnoid hemorrhage), seven (22.6%) had edema and/or subarachnoid hemorrhage, and five (16.1%) had extensive brain lacerations; skull fractures were present in 42, 71, and 100%, respectively. Seventy-six per cent of those in the "minor" falls group had mass lesions.

TABLE I
Summary of injuries per distance of fall

Distance of Fall	High (≥5th floor)	Mid	Minor (≤3 feet)
Number	8	18	18
Mean age (yrs)	5.2	2.4	2.4
% DOA	75%	0%	5.5%
% Multiple lethal injuries	100%	5.5%	0%
% Pure lethal head injury	0%	72%	100%
% Lethal head plus nonlethal associated injuries	0%	17%	0%
% Lethal injury without head injury	0%	5.5%	0%

DISCUSSION

In comparing our study to other reviews of falls, several striking points are seen. In a review of children with injuries due to falls admitted to a Chicago medical center, Meller et al. found that the peak age of children falling was 6 years (7). This compares to that found in other studies (8) where 50% of falls occurred in children less than 5 years old. In our study, however, 89% of the children were less than 5 years of age and 64% were 2 years old or less. The first difference is that ours is an autopsy study, while the others are from hospital admissions. The second difference is that we have included all falls including those from running or falls from furniture as compared to the other studies which only included falls from heights. Nevertheless, if we look only at the children who fell from heights (eliminating as well the suicide, murder and fire-induced falls), the mean age of the remaining 23 children was still 2.3 years. This age difference could suggest that younger children are much more likely to die from a fall than are older children. The fact that these deaths were due to head injuries is consistent with the anatomic fact that younger children have softer cranial bones and thus less cerebral protection.

The second major distinction is the percentage of subdural and epidural hematomas in our study. Mayer et al. have shown that 30% of pediatric head trauma patients have significant mass lesions and thus operative neurosurgical intervention is often unneeded (6). This fact, along with interest in the nonoperative approach to abdominal injuries in children, has led some (4) to suggest that pediatricians trained in emergency medicine are better qualified to deliver care to pediatric trauma patients than either general or pediatric surgeons. From this study, however, it is clear that there is a need for trained pediatric neurosurgeons, as 61.3% of the children had mass lesions requiring neurosurgical intervention. In fact, as stated by Velcek (10), the delayed deaths of the children with head trauma illustrate the requirement of the "skill and continued availability of a neurosurgical team." If a neurosurgeon is not available, then someone with surgical experience must be immediately available for intervention.

While some of the "minor" falls may have been secondary to abuse despite negative investigations (all of these had intense police investigation to rule out abuse), it is important that two of these falls did occur under medical observation. "Minor falls" can be lethal, especially in a toddler, and must be evaluated. It is rather disconcerting, however, that of the final two groups, 42% suffered injuries that are usually associated with survival had the injuries been immediately recognized and aggressive resuscitation and definitive treatment initiated. Additionally, we have found, as have other authors (2, 8), that it is extremely rare to have visceral, thoracic, or non-skull fracture injuries in children who fall from less

than 3 floors. It is, in fact, possible to suggest that if these injuries are found in a child with a fall from less than 3 stories, one should suspect abuse as the etiology of the injury.

The purpose of this study was to seek means to improve the treatment and the prevention of childhood injuries. A 61% incidence of potential survivors is unacceptable. With the advent of pediatric trauma centers (such as those designated by legislation in the city of Chicago in November 1986), one would expect that the delays in treatment would be avoided. This is, however, only the first step. A means of educating parents regarding falls must be implemented. If the parents of 20% of these children had sought medical care sooner, their deaths could possibly have been prevented. Additionally, a fall prevention program like that instituted in New York City (9) with provision of barriers over windows, would have prevented the deaths of the 19 (43%) children who fell from windows. One 2-old child was left on a table next to an open window on the ninth floor of an apartment building while her mother answered the door. A child playing on a bed or table next to an open window is in reality playing Russian roulette, yet we allow it to happen. Finally, again as shown by other authors (3), a fair number of our records indicated that the child was being watched by an "older" sibling or by a friend who "fell asleep on the couch." In essence, these children were unattended. Society must provide a means of child care for parents so that their children are not unsupervised while the parent works or must be away from home. Moreover, parents must be educated to understand the necessity of seeking appropriate medical care if their children do fall, *regardless* of the height. We cannot expect young children to understand the dangers of their environment, so we must protect them. As physicians and child advocates, we must push for legislation to provide this protection.

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Intracranial Hemorrhage and Rebleeding in Suspected Victims of Abusive Head Trauma: Addressing the Forensic Controversies

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Does an expanded subarachnoid space predispose to subdural bleeding? What does heterogeneity in the appearance of a subdural collection on CT or MRI imaging indicate? Spontaneous rebleeding? Minor re-injury? Major re-injury? In some specific cases, answers to these questions have important forensic implications. To conclude objectively that an infant's intracranial hemorrhage or rebleeding resulted from inflicted injury or re-injury requires an in-depth understanding of the pathogenesis of posttraumatic subdural and subarachnoid collections. The authors present two cases of indoor, accidental, pediatric, closed-head trauma that resulted in intracranial rebleeding. Both accidental cranial impacts occurred in medical settings and were independently witnessed by medical personnel. In addition, the authors summarize the relevant medical literature regarding pediatric intracranial bleeding and rebleeding.

Abusive head trauma is the leading cause of traumatic death during infancy. Subdural hematoma is a frequent finding in these young victims. There are many nontraumatic etiologies for subdural hemorrhage. On the other hand, in the absence of an adequate alternate explanation for subdural bleeding during infancy or childhood, child abuse must be considered.

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How can we *objectively* interpret the forensic significance of pediatric intracranial hemorrhage or rebleeding? We will explore this question in depth by addressing the following questions in order:

1. What are the potential explanations for enlargement of the subarachnoid space in an infant or young child?
2. Is an infant or young child with an enlarged subarachnoid space predisposed to subdural bleeding?
3. What is the differential diagnosis for subdural hematoma?
4. What is the pathophysiology of traumatic subdural hemorrhage?
5. What does serial cranial imaging reveal in young victims of head trauma?
6. What are the potential explanations for heterogeneity in the appearance of a subdural collection on CT or MR imaging?
7. Under what circumstances do subdural hemorrhages rebleed?
8. What are the expected clinical consequences of traumatic subdural bleeding and rebleeding in an infant or young child?

What Are the Potential Explanations for Enlargement of the Subarachnoid Space in an Infant or Young Child?

In the first 2 years of life, the subarachnoid spaces are relatively larger than in older children or adults (see Table 1) (Alper et al., 1999; Fessell, Frankel, &

TABLE 1: Differential Diagnosis of an Expanded Subarachnoid Space

Benign congenital enlargement (secondary to immaturity of the arachnoid villi)
Acute subarachnoid hemorrhage
Posttraumatic communicating hydrocephalus (secondary to adhesive arachnoiditis induced by acute subarachnoid hemorrhage)
Expansion of the subarachnoid space secondary to partial obstruction of regional CSF flow by a contiguous or overlying acute subdural hematoma
Cerebral atrophy
Subdural hygroma (misinterpreted as an expanded subarachnoid space)
Chronic subdural hematoma (misinterpreted as an expanded subarachnoid space)

Wolfson, 1997; Kleinman, Zito, Davidson, & Raptopoulos, 1983; Libicher & Troger, 1992; Prassopoulos & Cavouras, 1994). This benign condition is more common in premature infants and in young infants with macrocephaly. Alvarez, Maytal, and Shinnar (1986) found a positive family history of macrocephaly in 88% of cases. Congenital expansion of the subarachnoid spaces during infancy is characterized by reversible enlargement of the subarachnoid spaces (most evident in the frontal regions), prominence of the basilar cisterns and the anterior interhemispheric fissure, and mild ventriculomegaly. Early CT studies incorrectly suggested that these extracerebral collections resided in the subdural space (Mori, Handa, Ito, & Okuno, 1980; Robertson, Chun, Orrison, & Sackett, 1979). Benign enlargement of the subarachnoid spaces probably represents a physiologic and transient form of communicating or external hydrocephalus resulting from immaturity of the arachnoid villi (Wolpert & Barnes, 1992).

Enlargement of the subarachnoid space may result from subarachnoid hemorrhage—a common finding in young victims of inflicted cranial trauma (Cohen, Kaufman, Myers, & Towbin, 1986; Dolinskas, Zimmerman, & Bilaniuk, 1978; Duhaime et al., 1992; Ewing-Cobbs et al., 2000; Reece & Sege, 2000; Wolpert & Barnes, 1992). Blood products in the subarachnoid space may cause an adhesive arachnoiditis, which impedes absorption of cerebrospinal fluid (CSF) by the arachnoid villi (Barkovich, 1995; Fitz & Harwood-Nash, 1978). Through this mechanism, isolated subarachnoid hemorrhage may predispose to posttraumatic, communicating hydrocephalus manifesting as expansion of the subarachnoid space.

More commonly in cases of child abuse, enlargement of the subarachnoid space is seen in association with contiguous or overlying acute subdural hemorrhage (Kapila, Trice, Spies, Siegel, & Gado, 1982; Mori et al., 1980; Orrison, Robertson, & Sackett, 1978; Robertson et al., 1979; Rothenberger & Brandl, 1980). The presence of extensive subdural hemorrhage overlying a large surface of the cerebral convexity may impede the flow of cerebrospinal fluid into the region underlying the hematoma (Elvidge & Jackson, 1949; Mori et al., 1980), thereby expanding the surrounding subarachnoid spaces. In some cases of inflicted pediatric head trauma with subdural hematoma, a definitive expansion of the subarachnoid spaces and/or the ventricles is noted on follow-up cranial imaging a few days after injury (Kleinman & Barnes, 1998).

Finally, an expanded subarachnoid space may also represent cerebral atrophy—a frequent and oftentimes devastating complication of inflicted pediatric head trauma. Using cranial CT imaging alone, it may be difficult or impossible to differentiate between cerebral atrophy, posttraumatic communicating hydrocephalus, and/or a chronic subdural collection. An enlarged or enlarging head circumference suggests communicating hydrocephalus or a more chronic subdural collection. Static or decreasing head circumference suggests cerebral atrophy or subdural hygroma—a space-filling lesion (Barkovich, 1995). In most cases of abusive head trauma, the acute subdural hemorrhage, with associated diffuse brain injury, precedes the development of cerebral atrophy, communicating hydrocephalus with expansion of the subarachnoid space, and/or a chronic subdural collection (Kleinman & Barnes, 1998).

Is an Infant or Young Child With an Enlarged Subarachnoid Space Predisposed to Subdural Bleeding?

The frequent coexistance of an enlarged subarachnoid space with overlying subdural collections has led some authors to suggest that an enlarged CSF space predisposes to the development of acute subdural hemorrhage. Authors have postulated that the amount of trauma needed to produce subdural hemorrhage in these infants is less than that required to produce acute subdural bleeding in infants with "normal" subarachnoid spaces (Aoki, 1994; Aoki & Masuzawa, 1984; Howard, Bell, & Uttley, 1993; Ikeda et al., 1987; Kapila et al., 1982; Papasian & Frim, 2000; Veyrac, Couture, & Baud, 1990). In addition, authors have proposed that susceptibility to subdural hemorrhage is related to racial variations in the size of the subarachnoid space and head circumference (Aoki, 1994; Howard et al., 1993; Rekate, 1985).

Does an expanded subarachnoid space predispose a child to traumatic subdural hemorrhage (e.g., as a

complication of a minor fall)? The medical literature does not give us a definitive answer to this question. More likely, however, the opposite is true. Acute subdural hemorrhage may facilitate secondary expansion of the subarachnoid space (Kleinman & Barnes, 1998). Consider the following:

1. Benign prominence of the subarachnoid space is common in normal infants.
2. Though limited in number, long-term observations of infants with benign, congenital expansion of their subarachnoid spaces revealed no increased frequency of subdural hematomas over time (Briner & Bodensteiner, 1981; Ment, Duncan, & Geehr, 1981; Robertson et al., 1979).
3. Very likely, some severely head-injured infants with traumatic subdural hemorrhage had pre-existing and benign enlargement of their subarachnoid spaces unrelated to the development of their subdural hematoma (Kleinman & Barnes, 1998).
4. Finally, as described previously, displacement of the subarachnoid compartment by an overlying subdural collection may cause secondary expansion of the surrounding subarachnoid spaces (Elvidge & Jackson, 1949; Mori et al., 1980).

Until a prospective, comparative study concludes that normal infants with prominent subarachnoid spaces are at a statistically greater risk for subdural hemorrhage, this concept should be viewed with caution (Kleinman & Barnes, 1998). On the other hand, our first case report of subdural bleeding secondary to witnessed, minor, closed-head trauma suggests that expansion of the subarachnoid space may indeed predispose to subdural bleeding under certain abnormal clinical circumstances.

A 20-month-old boy suffered a linear skull fracture, an epidural hemorrhage, and transtentorial herniation after he fell down several concrete stairs. Despite neurosurgical intervention, CT imaging one month later revealed right hemispheric cerebral atrophy, ventriculomegaly, and an expanded subarachnoid space (see Figure 1). Three months later, he struck his forehead against a low windowsill during physical therapy. He cried immediately and was not easily consoled. He remained persistently irritable with decreased appetite over the following week. Cranial CT imaging several days later revealed an acute, right occipital, subdural hematoma in the region of his maximal cerebral atrophy (see Figure 2). He recovered gradually thereafter with resolution of his new subdural hematoma without additional neurosurgical intervention.

His first injury did not cause subdural or subarachnoid hemorrhage. Therefore, the later expansion of his subarachnoid space reflects cerebral atrophy—not posttraumatic communicating hydrocephalus or a chronic subdural collection. At the time of his second, minor, closed-head injury during physical therapy, acute subdural bleeding occurred in the region of his pre-existing cerebral atrophy and expanded subarachnoid space. His clinical signs were limited to irritability and loss of appetite. Because the second impact was relatively minor, the child did not suffer severe and acute clinical deterioration suggestive of traumatic axonal injury. Instead, his clinical signs were caused by the limited mass effect of focal subdural bleeding.

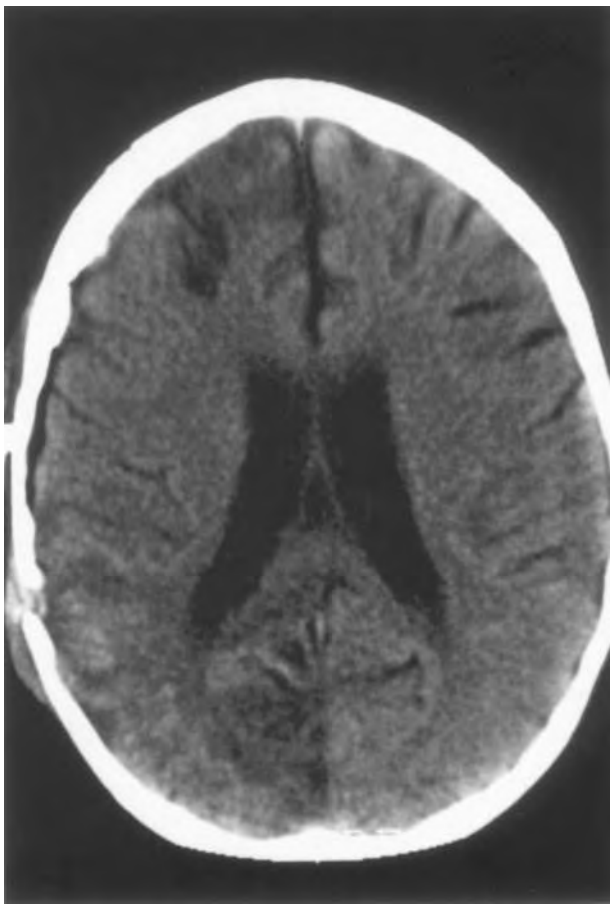

FIGURE 1: This 20-Month-Old Male Suffered a Linear Skull Fracture, an Epidural Hemorrhage, and Transtentorial Herniation After He Fell Down Several Concrete Steps. Despite Neurosurgical Intervention, CT Imaging One Month Later Revealed Right Hemispheric Cerebral Atrophy, Ventriculomegaly, and an Expanded Subarachnoid Space.

Because this child's expanded subarachnoid space was an acquired, posttraumatic condition, this case example demonstrates that an expanded subarachnoid space resulting from cerebral atrophy can pre-

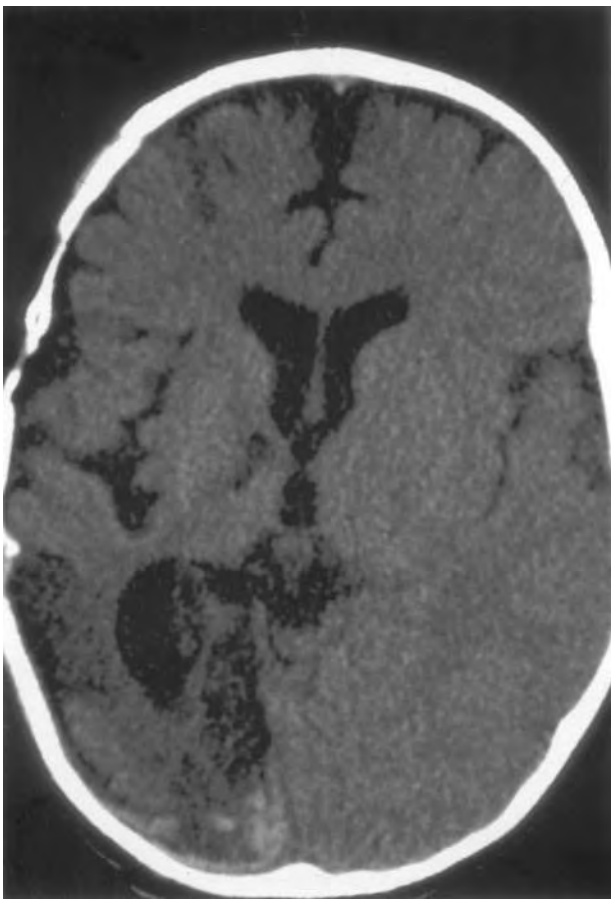

FIGURE 2: Three Months After His Initial Cranial Injury, This 20-Month-Old Male Struck His Forehead Against a Low Windowsill During Physical Therapy. He Cried Immediately and Was Not Easily Consoled. Over the Following Week, He Remained Persistently Irritable With Decreased Appetite. Cranial CT Imaging Several Days After This Witnessed, Minor Fall Revealed an Acute, Right Occipital, Subdural Hematoma in the Region of His Maximal Cerebral Atrophy.

dispose to regional subdural hemorrhage as a consequence of a minor, indoor fall. This witnessed case example should not be extrapolated to predict that benign expansion of the subarachnoid spaces during infancy predisposes to subdural bleeding.

What Is the Differential Diagnosis for Subdural Hemorrhage?

Subdural hemorrhages have been linked etiologically to accidental trauma; inflicted trauma; medical or surgical interventions; prenatal, perinatal, and pregnancy-related conditions; birth trauma; metabolic diseases; congenital malformations; genetic diseases; oncologic diseases; autoimmune disorders; clotting disorders; infectious diseases; the effects of poisons, toxins, or drugs; and other miscellaneous conditions. The vast majority of these entities can be diagnostically excluded (or confirmed) by careful history, physical examination, radiological studies, and/or laboratory testing (see Table 2).

What Is the Pathophysiology of Traumatic Subdural Hemorrhage?

The dura mater is composed of fibroblasts and large amounts of extracellular collagen. The dural border cell layer forms its innermost region. The arachnoid consists of an outer barrier cell layer and the inner arachnoid trabeculae, which bridge the subarachnoid space. An actual or potential subdural space does not exist in humans. In most instances, when a tissue space is created traumatically in this general area of the meninges, it represents a cleaving of the innermost dural cell border layer (Haines, 1991). Traumatic, acute, subdural hemorrhage is a blood clot without a membrane within the cleaved dural border cell layer (also known as an intradural hematoma). Given its common usage, in this article, we use the term *subdural* to refer to these posttraumatic, intradural collections.

Acute, traumatic subdural hemorrhage may result from either a contact or noncontact mechanism of injury. When subdural hemorrhage is limited to the impact site or the region underlying a skull fracture, it likely represents an isolated contact injury (i.e., resulting primarily from skull deformation and not from cranial acceleration). Because linear skull fractures actually begin in a region of cranial outbending and not necessarily at the point of impact and inbending (Arnholz, Hymel, Hay, & Jenny, 1998; Gurdjian, Webster, & Lissner, 1950), a contact subdural hemorrhage can occur at a location other than the impact site as a complication of a linear skull fracture.

Occasionally, intracranial hemorrhage resulting from contact forces alone may cause coma or death if sufficiently large to cause brain shifts, herniation, or brainstem compression (Gennarelli, 1984). If cranial motion is restricted but impact occurs with sufficient force and energy over a very large cranial surface area (e.g., crush injury), large and/or bilateral underlying subdural hematomas can occur as a massive contact injury unrelated to cranial acceleration (Duhaime, Eppley, Margulies, Heher, & Bartlett, 1995; Hymel, Bandak, Partington, & Winston, 1998). On the other hand, a small, focal subdural collection can disappear rapidly on neuroimaging (Duhaime, Christian, Armonda, Hunter, & Hertle, 1996).

(Text continues on page 338)

TABLE 2: Differential Diagnosis for Subdural Hemorrhage

Condition	*Differentiate by*				*Comments*
	History	*Physical Exam*	*Radiological Studies*	*Laboratory Tests*	
Trauma					
Nonaccidental trauma (Deb et al., 2000; Jayawant et al., 1998)	Yes/No	Yes/No	Yes/No	No	History may be unreliable. Physical exam and radiologic studies often yield information leading to diagnosis. Blunt and penetrating trauma can cause SDH.
Accidental trauma (Wilkins, 1997)	Yes	Yes/No	Yes/No	No	Validate history. When history matches injury, accidental trauma is more likely.
Traumatic aneurysm of the middle meningeal artery (Aoki, Sakai, & Kaneko, 1992)	Yes	No	Yes/No	No	Seven reported cases in adults. Can cause delayed SDH after severe injury.
"Headbanging" to rock music (Mackenzie, 1991)	Yes	No	No	No	Two adults who violently shook their heads to the beat of rock music developed SDH. One also had a traumatic dissection of the carotid artery.
"Breakdancing" (McNeil, Spruill, Langley, Shuping, & Leonard, 1987)	Yes	Yes/No	No	No	Breakdancing can involve repetitive trauma to the head. One case is reported in the literature of SDH caused by breakdancing.
Roller-coaster rides (Kettaneh, Biousse, & Bousser, 2000)	Yes	No	No	No	Three cases of SDH are reported in the French literature after roller-coaster rides.
Medical or surgical interventions					
Spinal tap (Hart, Bone, & Hadley, 1988)	Yes	No	No	No	SDH has been reported as a rare complication of a spinal tap in an adult. SDH symptoms developed after the tap for an unrelated condition.
Spinal anesthesia (Ortiz et al., 1991)	Yes	No	No	No	Intracranial SDH have rarely been reported as complications of spinal anesthesia. The mechanism is debated.
Epidural anesthesia (Diemunsch et al., 1998)	Yes	No	No	No	Few reports are noted.
Lumbar myelography (Suess, Stendel, Baur, Schilling, & Brock, 2000)	Yes	No	No	No	In adults, 16 cases of intracerebral SDH have been reported after lumbar myelograms.
Ventriculoperitoneal or ventriculoarterial shunt (Badiane et al., 1992; Sharma, Mahapatra, Pawar, Sousa, & Athale, 1999)	Yes	Yes	Yes	No	SDH can be a long-term complication of shunts for hydrocephalus.
Craniotomy (Wyllie, Comair, Kotagal, Raja, & Ruggieri, 1996)	Yes	Yes	Yes	No	Disruption of vessels during surgery can cause subdural bleeding.
Prenatal, perinatal, and pregnancy-related conditions					
Intrauterine trauma (Gunn & Becroft, 1984; Stephens, Richardson, & Lewis, 1997)	Yes/No	Yes/No	No	No	Usually occurs with skull fracture. History may not be reliable, particularly if trauma is the result of domestic violence.
Idiopathic intrauterine SDH (Green, Wilson, Romaniuk, May, & Welch, 1999)	Yes	No	Yes	No	In some cases, the cause of intrauterine SDH cannot be identified.
Intrauterine isoimmune thrombocytopenic purpura (Zalneraitis, Young, & Krishnamoorthy, 1979)	Yes	No	No	Yes	Rare condition where maternal antibodies destroy fetal platelets.
Pre-eclampsia (Giannina, Smith, Belfort, & Moise, 1997)	Yes	Yes	No	Yes	SDH is rarely reported in pre-eclamptic, pregnant women.

(continued)

TABLE 2 (continued)

Condition	*Differentiate by*				*Comments*
	History	*Physical Exam*	*Radiological Studies*	*Laboratory Tests*	
Postnatal cerebral infarction (Steinbok, Haw, Cochrane, & Kestle, 1995)	No	No	Yes	No	Cerebral infarction associated with SDH has been reported shortly after birth. The etiology of this condition is unknown.
Birth trauma					
Forceps delivery (Hankins, Leicht, Van Hook, & Uckan, 1999)	Yes	Yes/No	No	No	Occurs in less that 0.01% of deliveries. Scalp trauma is obvious initially.
Vacuum extraction (Hanigan, Morgan, Stahlberg, & Hiller, 1990)	Yes	Yes/No	No	No	SDH around the tentorium has been reported as a complication.
Other birth trauma (Hayashi, Hashimoto, Fukuda, Ohshima, & Moritaka, 1987)	Yes	No	No	No	Births other than breech births and suction deliveries can cause SDH. It is more likely to occur in infants of primiparous mothers. Symptoms develop shortly after birth.
Metabolic diseases					
Glutaric aciduria type I (Baric et al., 1998)	Yes/No	Yes	Yes	Yes	Can present with SDH. Often have large heads and dystonia. Persistent acidosis occurs. Urine organic acids abnormal. Degeneration of putamen and caudate seen on CT.
Galactosemia (Sychlowy and Pyda 1971)	Yes	No	No	Yes	One case is reported in the Polish literature.
Pyruvate carboxylase deficiency (Ahmad et al., 1999)	No	No	No	Yes	An inborn error of carbohydrate metabolism presenting with lactic acidemia and acidosis.
Congenital malformations					
Intracranial arteriovenous malformations (Oikawa, Aoki, & Sakai, 1993)	No	No	Yes	No	Not common in children under 6 years.
Cerebral aneurysm (O'Leary & Sweeny, 1986)	No	No	Yes	No	Ruptured aneurysm usually causes subarachnoid hemorrhage.
Arachnoid cyst (Romero, Rovira, Ibarra, Piqueras, & Rovira, 1989)	No	No	Yes	No	Rupture of vessels surrounding arachnoid cyst can cause SDH.
Spontaneous rupture of a cerebral artery (Koc, Pasaoglu, Kurtsoy, Oktem, & Kavuncu, 1997; Tokoro, Nakajima, & Yamataki, 1988)	No	No	No	No	Rare condition reported only in adults. Deformed artery is often found at autopsy or at surgery.
Schizencephaly or porencephaly (Osaka, Shirataki, Matsumoto, Yokoyama, & Ogino, 1977)	No	No	Yes	No	Subdural effusions of CSF can be caused by direct connection of the dura to the lateral ventricle through a brain malformation.
Genetic diseases					
Osteogenesis imperfecta (Cole & Lam, 1996)	Yes/No	Yes	Yes	Yes	Familial, but can occur as a spontaneous mutation. Physical characteristics may include poor growth, poor teeth, blue sclera, and joint laxity. Bones usually appear demineralized on X-ray. Testing of collagen production in fibroblasts is positive in 85% of cases.
Sickle-cell anemia (Falter, Sutton, & Robinson, 1973)	Yes	No	No	Yes	The mechanism of SDH in patients with sickle-cell anemia is unknown. The SDH is frequently fatal.
Alagille syndrome (Woolfenden et al., 1999)	Yes	Yes	Yes	Yes	Children with Alagille syndrome can develop vasculitis similar to Moyamoya disease. They are also more likely to develop other types of serious intracranial hemorrhages such as epidural hematomas, presumably secondary to the thinness of their skulls.

Ehlers-Danlos syndrome (Ortiz Remacha, Candia, & Conde, 2000)	Yes	Yes	No	Yes	SDH can be the presenting manifestation of Ehlers-Danlos syndrome.
Autosomal dominant polycystic kidney disease (Wijdicks, Torres, & Schievink, 2000)	Yes/No	No	Yes	Yes	Chronic SDH was noted in 5 patients. ADPKD is known to be associated with vascular anomalies of the CNS.
Menkes' kinky hair syndrome (Seay et al., 1979)	Yes	Yes	Yes	Yes	SDH is associated with brain atrophy.
Oncologic diseases					
Meningeal carcinomatosis (McKenzie, Rengachary, McGregor, Dixon, & Suskind, 1990)	Yes	Yes/No	Yes	Yes/No	Metastases of neoplasms to the leptomeninges can cause SDH. This has been reported with many types of cancer, including sarcomas, lung cancers, prostate cancers, breast cancers, and others.
Leukemia (Mashiyama et al., 2000)	Yes/No	Yes/No	No	Yes	SDH has been reported as the presenting symptom of both acute monocytic leukemia in a child and chronic lymphocytic leukemia in adults.
Solid CNS tumors (Ozhan, Tali, Isik, Saygili, & Baykaner, 1999; Timothy, Lafuente, Chakrabarty, Saxena, & Marks, 1999)	Yes/No	Yes/No	Yes	Yes/No	Solid tumors can present as SDH or be complicated by SDH, including fibrous histiocytoma, meningiomas, and others.
Primary mucosa-associated lymphoma in the dura (Kambham, Chang, & Matsushima, 1998)	No	No	No	Yes	Diagnosis is made by histology of tumor cells.
Mass lesions in the subdural space (Cinalli et al., 1997)	No	No	Yes	Yes	Two cases of mass lesions in the subdural space (liposarcoma and fibrohistiocytic sarcoma) are reported, where a subdural effusion around the tumor was misdiagnosed as a chronic SDH.
Autoimmune disorders					
Lupus erythematosus (Bovim, Jorstad, & Schrader, 1990)	Yes	Yes	No	Yes	Spontaneous SDH has been described in adults with lupus, not necessarily related to antiplatelet antibodies. Other lupus patients with lupus anticoagulant factors are also reported to have SDH.
Clotting disorders					
Anticoagulant therapies (Massicotte, Marzinotto, Vegh, Adams, & Andrew, 1995; Snyder, Sridharan, & Pagnanelli, 1997)	Yes	Yes/No	No	Yes	Warfarin and other anticoagulants can lead to abnormal bleeding from minor injuries. SDH has been reported in anticoagulated patients after roller-coaster rides (adults).
Ginko biloba ingestions (Rowin & Lewis, 1996)	Yes	Yes/No	No	Yes/No	One case of an adult developing a spontaneous SDH after chronic ingestion. The herb has platelet-inhibiting properties.
Coagulopathy related to cirrhosis of the liver (Furui, Yamada, & Iwata, 1989)	Yes	Yes	No	Yes	Liver disease can impair clotting.
Hemophilia A and B (de Tezanos Pinto, Fernandez, & Perez Bianco, 1992; Lutschg & Vassella, 1981; Ohga et al., 1988)	Yes/No	Yes/No	No	Yes	In the absence of surgical procedures, most hemophilia patients do not develop bleeding problems during the neonatal period. SDH is usually related to minor trauma, rather than spontaneous. SDH is rare in these patients.
Factor V deficiency (Salooja, Martin, Khair, Liesner, & Hann, 2000)	Yes/No	Yes/No	No	Yes	SDH has presented at birth in an infant with severe Factor V deficiency (<1%).
Factor XII deficiency (Nicholls, Chan, Koo, Kwong, & Tsoi, 1993)	Yes/No	Yes/No	No	Yes	Was reported in an infant.
Idiopathic or drug-induced thrombocytopenic purpura (Kolluri, Reddy, Reddy, Naidu, & Kumari, 1986)	Yes/No	No	No	Yes	Platelet deficiency creates coagulopathy.
Hemorrhagic disease of the newborn (vitamin K deficiency) (Behrmann, Chan, & Finer, 1985)	Yes	No	No	Yes	Occurs in infants not receiving vitamin K injections at birth.

(continued)

TABLE 2 (continued)

Condition	*Differentiate by*				*Comments*
	History	*Physical Exam*	*Radiological Studies*	*Laboratory Tests*	
Diffuse intravascular coagulation (Furui et al., 1983; Hymel et al., 1997; Stein, Young, Taluchi, Greenbaum, & Ross, 1992)	Yes	Yes	No	Yes	DIC from cancer, infection, or trauma can be associated with SDH.
Acquired inhibitors of plasma clotting factors (Ryan, Arkel, Walters, Frimmer, & Desposito, 1986)	Yes/No	No	No	Yes	Thought to be a postviral phenomenon. Can cause a severe coagulopathy.
Hermansky-Pudlak syndrome (Russell-Eggitt, Thompson, Khair, Liesner, & Hann, 2000)	No	Yes	No	Yes	Associated with a bleeding diathesis caused by impaired platelet function. Affected children usually have albinism and nystagmus.
Alpha 1-antitrypsin deficiency (Israels & Gilfix, 1999)	No	No	No	Yes	Presents as "late hemorrhagic disease of the newborn" with vitamin K deficiency.
Infectious diseases					
Hemophilus influenzae meningitis (Ogilvy, Chapman, & McGrail, 1992)	Yes	Yes	Yes	Yes	Meningitis can cause subdural effusions and empyemas that can be confused with old hematomas.
Streptococcus pneumoniae meningitis (Ogilvy et al., 1992)	Yes	Yes	Yes	Yes	Meningitis can cause subdural effusions and empyemas that can be confused with old hematomas.
Other bacterial meningitis (Syrogiannopoulos, Nelson, & McCracken, 1986)	Yes	Yes	Yes	Yes	Subdural effusions are caused less commonly by other organisms such as *Neisseria*, Group B *Streptococcus*, *Escherichia coli, Listeria monocytogenes*, and others.
Kawasaki disease (Aoki, 1988)	Yes	Yes	No	Yes/No	Subdural *effusions* (rather than hematomas) can occur with Kawasaki disease secondary to leptomeningeal vasculitis.
Endocarditis leading to septic emboli of a cranial artery causing aneurismal rupture of the vessel (Bandoh, Sugimura, Hosaka, & Takagi, 1987)	Yes	Yes	No	Yes	Rare, not reported in children.
Chronic otitis media (Gower & McGuirt, 1983)	Yes	Yes	No	No	Subdural effusions and empyemas (not hematomas) have been reported with chronic middle ear disease.
Intracranial extension of sinus infections (Dolan & Chowdhury, 1995)	Yes/No	No	Yes	No	Subdural empyemas can form from intracranial extension of sinusitis. (More common in the pre-antibiotic era.)
Malaria (Omanga, Shako, Ntihinyurwa, Mbuyu, & Beltchika, 1979)	Yes	No	No	Yes	Malaria can cause subdural effusions (not hematomas).
Herpes simplex encephalitis (Saito et al., 1983)	Yes	Yes/No	No	Yes	One case of chronic SDH is reported after HSV encephalitis in the Japanese literature.
Congenital toxoplasmosis	Yes	Yes	Yes/No	Yes	Reported in the Polish literature. Possibly related to cerebral atrophy.
Poisoning, toxins, or drug effects					
Lead poisoning (Sensirivatana, Supachadhiwong, Phancharoen, & Mitrakul, 1983)	Yes	No	Yes	Yes	One case of subdural effusions with high protein content after lead poisoning has been reported. The etiology of the effusions was unknown.
Cocaine (Keller & Chappell, 1997)	Yes	No	No	Yes/No	Cocaine use has been associated with spontaneous SDH.
Tamoxifen (Missori et al., 1998)	Yes	Yes/No	No	No	Chronic SDH has been reported in patients taking tamoxifen. It is unknown if this is a primary effect or an occurrence related to other aspects of the patients' disease.

Other					
Hemodialysis of patients with kidney disease (Bechar, Lakke, van der Hem, Beks, & Penning, 1972)	Yes	Yes	No	No	Spontaneous SDH in dialysis patients can be related to liver disease or anticoagulant therapy, but sometimes the cause is not apparent.
Open heart surgery (Yokote et al., 1985)	Yes	Yes	No	No	Rarely reported complication; etiology is unclear, but may be related to anticoagulation.
Moyamoya disease (Takeuchi, Ichikawa, Koike, Tanaka, & Arai, 1992)	No	No	Yes	No	Moyamoya is a rare vascular disease of the central nervous system affecting the distal arteries. Moyamoya means "hazy puff of smoke" in Japanese, referring to the angiographic appearance of the abnormal vessels developing at the base of the brain.
Bone marrow transplant (Colosimo et al., 2000)	Yes	Yes	No	Yes	In one series, 2.6% of patients receiving allogeneic or autologous bone marrow transplants developed SDH. The therapeutic maneuver involves many factors leading to central nervous system bleeding, including intrathecal methotrexate therapy, low platelet counts, and coagulopathies.
Hyperostosis frontalis interna (Ishiguro, Nakagaw, Yamamura, & Kurokawa, 1997)	Yes	Yes	Yes	No	A rare condition causing frontal bossing.
Wegener's granulomatosis (Yokote, Terada, Nakai, & Itakura, 1997)	Yes	No	Yes	Yes	Subdural effusions containing blood and pus reported in a few cases.
Hemorrhagic shock and encephalopathy	Yes/No	Yes	No	Yes	A rare condition of unknown etiology, usually fatal, causing severe hyperthermia, coagulaopathy, and shock. Possibly is a postviral condition. Although subdural hematomas have not been reported in the literature resulting from this condition, the author (CJ) has seen one case.
Complication of parenteral nutrition (Rushforth, Green, Levene, & Puntis, 1991)	Yes	No	No	Yes	One case is reported in the literature of a preterm infant developing subdural "fat effusions" as a result of parenteral nutrition through a central venous catheter.
Spontaneous intracranial hypotension (Nakajima, Sakai, Aoki, & Takakura, 1996)	Yes	No	Yes	No	Bilateral chronic subdural hematomas have been linked to cerebral hypotension caused by a CSF leak; sometimes associated with collagen abnormalities; has not been reported in children.

NOTE: Some of the conditions listed cause subdural effusions or empyemas, which can be confused with subdural hemorrhage.

Traumatic, acute, subdural hemorrhage can also occur as a noncontact injury (i.e., resulting primarily from cranial acceleration and not from skull deformation). Whole head, rotational, cranial acceleration induced by impact or impulsively (i.e., without impact) can rupture parasagittal bridging veins. Noncontact, subdural hematomas in infants and young children tend to be large, interhemispheric, and/or bilateral. Noncontact subdural hematoma is a frequent finding in young victims of inflicted head trauma (Alexander, Schor, & Smith, 1986; Aoki & Masuzawa, 1986; Bruce & Zimmerman, 1989; Cohen et al., 1986; Duhaime et al., 1992; Ewing-Cobbs et al., 1998, 2000; Guthkelch, 1971; Hymel, Rumack, Hay, Strain, & Jenny, 1997; Jayawant et al., 1998; Ludwig & Warman, 1984; Merten & Carpenter, 1990; Merten, Osborne, Radkowski, & Leonidas, 1984; Reece & Sege, 2000; Sato et al., 1989; Tsai, Zee, Apthorp, & Dixon, 1980).

It is widely presumed that chronic subdural hematoma develops directly from acute subdural hematoma. Radiologically, subdurals evolve from high density in the acute phase, to isodensity with brain parenchyma in the subacute phase, and finally to low density similar to cerebrospinal fluid in the chronic phase. For this reason, a subdural collection that was observed to have evolved from an acute subdural hematoma is appropriately referred to as a *chronic subdural hematoma* (Kleinman & Barnes, 1998). However, at least in adults, only a few cases of chronic subdural hematoma evolve directly from acute subdural hematoma (Croce et al., 1994; Dolinskas, Zimmerman, Bilaniuk, & Gennarelli, 1979; Lee et al., 1996; Mathew et al., 1993). Experimental models largely fail to produce an enlarging, chronic, subdural hematoma from an acute solid clot (Goodell & Mealey, 1963; Watanabe, Shimada, & Ishii, 1963). Instead, the vast majority of chronic, posttraumatic, subdural collections evolve from subdural hygroma (Lee, Bae, Doh, Bae, & Yun, 1998).

A posttraumatic, subdural hygroma represents an accumulation of cerebrospinal fluid in the subdural space without a membrane, frequently with modified composition (Lee, 1998; Lee, Bae, Park, & Yun, 1994). An arachnoid tear has been widely proposed as a potential pathogenic mechanism (Borzone et al., 1983; Fobben et al., 1989; Gade et al., 1990; Miller, 1987; Murata, 1993). More likely, subdural hygroma develops by effusion upon resolution of acute subdural hemorrhage (Gutierrez, McLone, & Raimondi, 1979; Wetterling et al., 1988).

Acute subdural hematoma often resolves rapidly. Such rapid resolution likely reflects the high levels of tissue thromboplastin in brain tissue and cerebrospinal fluid (Astrup, 1965). As it resolves, a posttraumatic space may persist within the cleaved intradural membrane, particularly in the presence of decreased intracranial pressure. In the presence of sufficient, persistent, intradural space, subdural hygroma develops by effusion. Persistence of the traumatic, intradural defect may be facilitated by prolonged spinal fluid drainage, excessive therapy with osmotic agents, dehydration, and/or evolving cerebral atrophy. Increased arachnoid and vascular permeability resulting from trauma may also facilitate the effusion (Gutierrez et al., 1979; Wetterling et al., 1988). Thus, subdural hygroma begins not as a mass lesion but as a delayed, space-filling lesion. For this reason, a majority of subdural hygromas are aymptomatic (Lee, 1998; Lee et al., 1994, 1998).

Typically, subdural hygroma appears as an extra-axial collection of similar density to cerebrospinal fluid and must be differentiated radiologically from chronic subdural hematoma and/or cerebral atrophy with a widened subarachnoid space (Kleinman & Barnes, 1998) (see Table 1). The early fate of a posttraumatic subdural hygroma is either resolution or continued expansion. Early expansion is related to continued effusion and brain shrinkage. Resolution of a subdural hygroma is related to fluid absorption and brain re-expansion. When the brain shrinks and/or effusion exceeds absorption, a subdural hygroma will enlarge (Lee, 1998; Lee et al., 1996, 1998).

If the hygroma persists, a neomembrane will form eventually from proliferating dural border cells. Once a neomembrane has formed, neovascularization occurs. Spontaneous microhemorrhages from this neomembrane occur frequently into the expanding subdural effusion. For this reason, the content of subdural hygroma is frequently mixed with blood or is xanthochromic. Microhemorrhages may occur with little or no trauma (Kleinman & Barnes, 1998; Lee et al., 1998). Ultimately, repeated hemorrhages can transform an enlarging subdural hygroma into an expanding chronic subdural hematoma—a collection of liquefied blood within a membrane (Fujisawa et al. 1991, 1995; Ito et al., 1976, 1987; Markwalder, 1989; Nakamura & Tsubokawa, 1989; Yamashima, Yamamoto, & Friede, 1983).

Chronic subdural hematoma fluid does not coagulate. Even with the addition of thrombin, a fibrin clot often does not form. All laboratory measures of coagulation indicate a dysfunction of the coagulation system in chronic subdural hematomas (Kawakami, Chikama, Tamiya, & Shimamura, 1989). A vicious cycle of rebleeding and hyperfibrinolysis is believed to ultimately result in hematoma enlargement (Toyosawa et al., 1997).

TABLE 3: Differentiation Between Subdural Hygroma and Chronic Subdural Hematoma

Feature	*Subdural Hygroma*	*Chronic Subdural Hematoma*
Appearance on CT/MRI	Similar to CSF	Similar to CSF
Mean interval from injury to diagnosis	1 to 3 weeks	Over 3 weeks
Head circumference	Static or decreasing	Enlarged or increasing
Fluid collection	Clear, xanthochromic, or blood-tinged	Dark brown "crank case oil"
Total protein, albumin and hemoglobin content	Substantially lower than blood	Similar to blood
Neomembrane	Often lacking	Usually present
Mass lesion	Rarely	Potentially

The distinction between subdural hygroma and chronic subdural hematoma may be difficult. Both subdural collections demonstrate density or intensity similar to cerebrospinal fluid on cranial CT or MR imaging. The mean interval from injury to diagnosis is 1 to 3 weeks for subdural hygroma and more than 3 weeks for chronic subdural hematoma (Lee et al., 1998). The fluid collection within a subdural hygroma is clear, xanthochromic, or blood-tinged. Subdural fluid within a chronic subdural hematoma may have a dark brown "crank case oil" appearance (Lee, 1998). The total protein, albumin, and hemoglobin contents in subdural hygroma are substantially lower than in chronic subdural hematoma or in blood (Weir, 1971). Neomembrane is usually present with chronic subdural hematoma, but subdural hygroma often lacks a membrane. In adults, subdural fluid within a chronic subdural hematoma may act as a mass lesion, whereas subdural hygroma rarely acts as a mass lesion and often disappears (Lee, 1998; Lee et al., 1994, 1998; Lee et al., 1996) (see Table 3).

During infancy, the differentiation between subdural hygroma and chronic subdural hematoma may be even more difficult or impossible. In the abused, head-injured infant, large, bilateral, subdural collections may lead to craniocerebral disproportion with an enlarging head, virtual disappearance of brain white matter, and massive subdural collections of CSF density (Duhaime & Sutton, 1992). With such intracranial findings, it is often difficult to determine whether a chronic subdural collection reflects a delayed space-filling lesion, a mass lesion, or both of these sequentially.

An infant's brain is very deformable. The skull is both unfused and elastic. Very likely, these characteristics facilitate the formation of posttraumatic subdural hygroma as a space-filling lesion. Thereafter, the extent and severity of the child's posttraumatic cerebral atrophy will significantly influence his/her propensity for development of an enlarged subarachnoid space and/or a chronic subdural hematoma. As the head-injured infant with cerebral atrophy grows older, the cranial sutures will fuse, the fontanels will close, and both skull elasticity and brain deformity will decrease. Under these circumstances, the child's chronic subdural hematoma may begin to act with mass effect, and subdural rebleeding from any etiology may be less well-tolerated (see Figure 3).

What Does Serial Cranial Imaging Reveal in Young Victims of Head Trauma?

We reviewed the available medical literature regarding the results of serial cranial CT imaging of head-injured children (Dias, Backstrom, Falk, & Li, 1998; Feldman, Brewer, & Shaw, 1995; Giangiacomo, Khan, Levine, & Thompson, 1988; Sinal & Ball, 1987; Stein, Spettell, Young, & Ross, 1993; Tabori et al., 2000). Our findings are summarized in Table 4. These reports were published between 1987 and 2000, representing a potentially wide range of CT imaging technology, technique and/or sensitivity for detection of subtle intracranial hemorrhage. Serial MR cranial imaging in young victims of head trauma has not been described. The specific timing of subsequent CT imaging varied widely across studies, dictated by clinical necessity rather than prospective research design. In addition, cases of potential delayed hemorrhage or rebleeding were not always described or interpreted in light of the associated clinical findings, making pathophysiologic extrapolations difficult. For example, initial intracranial hemorrhage may be self-limited by increased intracranial pressure.

Despite their limitations, these reports lead us to conclude the following:

1. The first appearance of intracranial hemorrhage in young, head-injured children can be delayed.
2. Serial CT cranial imaging frequently reveals progressive intracranial hemorrhage in young children hospitalized with head injury.
3. Some head-injured children with delayed and/or progressive intracranial hemorrhage will require neurosurgical intervention.

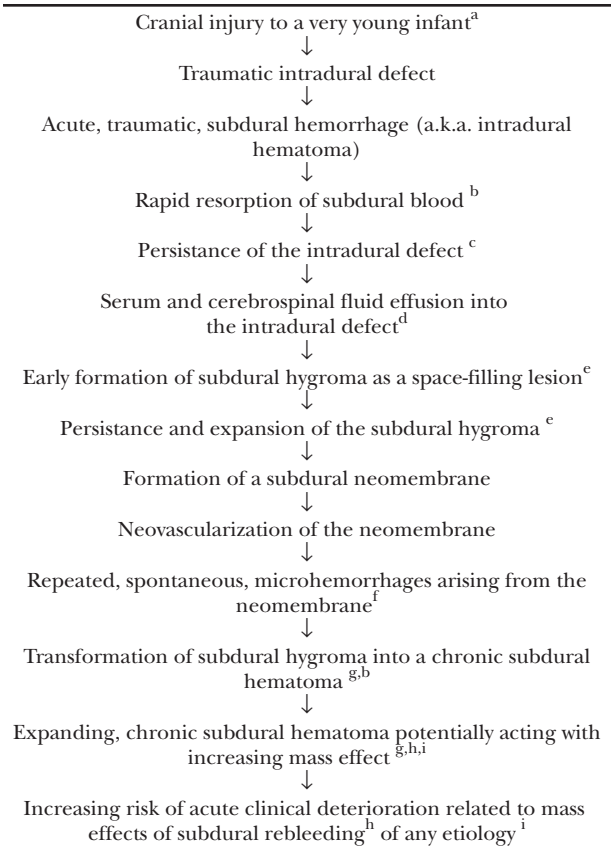

FIGURE 3: Pathophysiological Pathway Explaining Changing Clinical Manifestations of a Posttraumatic Subdural Collection in the Same Infant or Young Child Over Time

a. The acute clinical manifestations at the time of an acute, posttraumatic, subdural hemorrhage likely reflect the primary injury mechanism(s) and severity.
b. Facilitated by high levels of tissue thromboplastin in brain and cerebrospinal fluid.
c. Facilitated by decreasing intracranial pressure.
d. Facilitated by increased arachnoid and vascular permeability resulting from trauma.
e. Facilitated by evolving cerebral atrophy, open fontanels, and a young skull that is both unfused and elastic.
f. Spontaneously or as a consequence of minor closed head trauma.
g. Facilitated by the combination of rebleeding and hyperfibrinolysis.
h. Facilitated by skull maturation leading to decreasing elasticity, closed fontanels, and fused sutures.
i. Including spontaneous subdural rebleeding, rebleeding due to minor trauma, or major re-injury.

4. Spontaneous subdural rebleeding can and does occur in young children hospitalized with a head injury.
5. However, only a single case has been reported in the medical literature of a hospitalized, head-injured infant with spontaneous subdural rebleeding, documented on serial CT imaging and requiring neurosurgical intervention (Dias et al., 1998). Unfortunately, little or no details regarding this case are provided.

What Are the Expected Clinical Consequences of Subdural Bleeding or Rebleeding in an Infant or Young Child?

Acute subdural bleeding or rebleeding in an infant or young child has been linked directly or indirectly to a wide variety of acute and chronic clinical presentations (Alexander, Crabbe, Sato, Smith, & Bennett, 1990; Ewing-Cobbs et al., 1998; Gilles & Nelson, 1998; Jenny, Hymel, Ritzen, Reinert, & Hay, 1999; Johnson, Boal, & Baule, 1995; Willman, Bank, Senac, & Chadwick, 1997; Reece & Sege, 2000). Microhemorrhages originating from the neomembrane of a subdural hygroma may produce little or no clinical manifestations. On the other extreme, cranial acceleration injury mechanisms sufficient to cause subdural bleeding or rebleeding may precipitate traumatic axonal injuries at the craniocervical junction (Geddes et al., 2001; Geddes, Whitwell, & Graham, 2000) or more diffusely (Vowles, Scholtz, & Cameron, 1987). These primary brain injuries may manifest acutely as loss of consciousness, apnea, hypotension, prolonged traumatic coma, and/or death (Johnson et al., 1995; Willman et al., 1997).

Based on complex considerations, we may be able to predict the expected, acute, clinical consequences of subdural bleeding or rebleeding in young victims of head trauma (see Table 5). Adverse, acute clinical deterioration is *less* likely at the time of subdural bleeding or rebleeding if and when the subdural bleeding or rebleeding

- is microscopic and spontaneous (rather than induced by re-injury);
- occurs in a younger child with an elastic skull, unfused sutures, open fontanels, and a deformable brain;
- occurs into a space-filling subdural hygroma (as compared to chronic subdural hematoma acting with mass effect); and/or
- is induced by re-injury that does not precipitate clinically significant, primary brain injury (e.g., craniocervical or diffuse traumatic axonal injury).

On the other hand, adverse, acute clinical deterioration is *more* likely at the time of subdural bleeding or rebleeding if and when the subdural bleeding or rebleeding

- is induced by re-injury (rather than spontaneous and microscopic);
- occurs in an older child with decreasing skull elasticity, closed fontanels, fused sutures, and decreasing brain deformability;
- occurs into a chronic subdural hematoma acting with mass effect (as compared to a space-filling subdural hygroma); or

TABLE 4: Results of Serial Cranial CT Imaging in Young Victims of Head Trauma

Source	*Study Subjects*	*Changes on Follow-up Imaging Studies*
Stein, Spettell, Young, and Ross (1993)	351 children and adolescents with serious head injury	No spontaneous rebleeding; delayed or progressive intracranial lesions in 145 cases (41%); delayed parenchymal injury correlated with severity of initial injury, initial subarachnoid or intraventricular hemorrhage, and abnormal coagulation on admission.
Giangiacomo, Khan, Levine, and Thompson (1988)	5 whiplash shaken infants	All had abnormal imaging studies. One infant had bifrontal, hypodense subdural effusions. The child's CT at 13 days postadmission showed new, acute subdural hemorrhage.
Sinal and Ball (1987)	12 victims of abusive head trauma	3 developed new subdural hemorrhage seen 4 to 10 days after admission.
Feldman, Brewer, and Shaw (1995)	34 children with abusive head trauma	3 developed new, delayed abnormalities, including enlarging subdural effusion, evolving infarct, and delayed subdural and subarachnoid hemorrhage.
Tabori et al. (2000)	173 children with head trauma, Glascow Coma Score <12	47 (27%) of the repeat CT scans showed new lesions, including 6 new intracranial hemorrhages. 2 required surgical intervention.
Dias, Backstrom, Falk, and Li (1998)	21 infants with abusive head trauma	All initially had acute subdural hematoma. Four evolved to chronic subdural hematomas. One re-bled at 7 days and required surgical intervention.

TABLE 5: Expected Clinical Consequences of Subdural Bleeding or Rebleeding in Young Victims of Head Trauma

At the Time of Acute Subdural Bleeding or Rebleeding, Adverse Clinical Deterioration Is	
Less Likely	*More Likely*
If and When the Subdural Bleeding or Rebleeding	
. . . is microscopic and spontaneous	. . . induced by re-injury
. . . occurs in a younger child with increased skull elasticity, unfused sutures, open fontanels, and a very deformable brain	. . . occurs in an older child with decreasing skull elasticity, fused sutures, closed fontanels, and decreasing brain deformability
. . . occurs into a space-filling subdural hygroma	. . . occurs into a chronic subdural hematoma acting with mass effect
. . . is induced by re-injury that does not precipitate clinically significant primary brain injury	. . . is induced by re-injury that precipitates clinically significant primary brain injury

- is induced by re-injury that precipitates clinically significant, primary brain injury (e.g., craniocervical or diffuse traumatic axonal injury).

What Are the Potential Explanations for Heterogeneity in the Appearance of a Subdural Collection on CT or MR Imaging?

Pediatric victims of inflicted head trauma frequently suffer repetitive intracranial injuries (Alexander et al., 1990; Ewing-Cobbs et al., 2000; Jenny et al., 1999) (see Table 6). Therefore, discovery of intracranial hemorrhages of multiple ages during infancy should raise concern about the possibility of repetitive, inflicted cranial trauma.

In the majority of cases, an acute subdural hemorrhage is of uniform high density on CT imaging. However, exceptions do occur. Dias and colleagues described the appearance of a hypodense subdural collection 20 hours after head injury in an infant whose initial CT scan was considered normal (Dias et al., 1998). A hyperacute subdural hematoma may reveal low attenuation similar to the density of CSF or brain (Barnes & Robson, 2000). Leakage of cerebrospinal fluid into the subdural space may result from a traumatic disruption or tear of the arachnoid membrane (Borzone et al., 1983; Fobben et al., 1989; Gade et al., 1990; Miller, 1987; Murata, 1993). Rarely, this occurs as an isolated traumatic injury with little or no associated hemorrhage (Kleinman & Barnes, 1998). This is called an acute subdural hygroma—a lesion that may be misinterpreted radiologically as an older, chronic subdural hygroma or hematoma.

A hyperacute subdural may also reveal high- and low-density components that are arranged concentri-

TABLE 6: Potential Explanations for Heterogeneity in the Appearance of a Subdural Collection on CT or MR Imaging

Hyperacute subdural hematoma
Combined acute subdural hematoma and acute subdural hygroma
Heterogeneity within a single, evolving, subdural hemorrhage
Subdural rebleeding induced by acquired coagulopathy (e.g., DIC)
Spontaneous subdural rebleeding within a chronic subdural collection
Subdural rebleeding within a chronic subdural collection induced by minor closed head trauma
Repetitive and severe head trauma inducing multiple subdural hemorrhages

cally or intermixed. These mixed-density, hyperacute collections are frequently misinterpreted as chronic subdural hemorrhage, or chronic subdural hematoma with rehemorrhage (Sargent, Kennedy, & Kaplan, 1996). The low density component of a heterogenous, hyperacute subdural hematoma may represent active hemorrhaging, unclotted hemorrhage (particularly in a patient with low hematocrit or a clotting abnormality), or serum extrusion (low density) associated with early clot retraction (high density) (Kleinman & Barnes, 1998). Finally, an evolving subdural hemorrhage may demonstrate heterogeneity from one region to another over time within a single subdural collection.

Clinicians and radiologists should exercise considerable caution before estimating the age of a subdural collection or concluding with certainty that heterogeneity within a subdural collection represents rehemorrhage or re-injury (Kleinman & Barnes, 1998). Magnetic resonance imaging is the preferable modality for differentiating between aging hematoma and cerebrospinal fluid and for estimating the age of subdural bleeding (Alexander et al., 1986; Ball, 1989; Fobben et al., 1989; Gomori, Grossman, Goldberg, Zimmerman, & Billaniuk, 1985; Han et al., 1984; Langfitt et al., 1986; Sato et al., 1989; Snow, Zimmerman, Gandy, & Deck, 1986). Consultation with an experienced pediatric radiologist or neuroradiologist is strongly recommended.

On the other hand, the CT appearance of focal or multifocal, high-density, acute hemorrhage(s) in association with a moderate or large, low-density, subdural collection provides compelling evidence of subdural rehemorrhage or re-injury. Additional radiological manifestations of rehemorrhage or re-injury include septations, loculations, areas of varying density or intensity, and layering within the subdural collection (Kleinman & Barnes, 1998).

Intracranial hemorrhage or rebleeding may occur as a complication of an acquired coagulopathy—a frequent and potentially severe complication of traumatic brain injury in both adults (Kaufman et al., 1984; Olson et al., 1989; Stein, Young, Taluchi, Greenbaum, & Ross, 1992) and children (Hymel, Abshire, Luckey, & Jenny, 1997; Miner, Kaufman, Graham, Haar, & Gildenberg, 1982; Stein & Spettell, 1995). Tissue factor released from damaged brain cells may bind to factor VII and initiate activated coagulation, resulting in prothrombin time (PT) prolongation and occasionally disseminated intravascular coagulation (DIC) with ongoing or recurrent hemorrhage. More simply stated, the injured brain may protectively activate blood clotting throughout the body, ultimately consuming excessive blood clotting factors and leaving the patient vulnerable to rebleeding. In young victims of inflicted head trauma, PT prolongation and activated coagulation are strongly related to the presence of parenchymal brain damage (Hymel, Abshire, et al., 1997).

Because so many conditions may be confused with bleeding and rebleeding on CT or MR cranial imaging, it is highly likely that subdural bleeding with rebleeding has been overdiagnosed in the past. If this is true, then current opinions regarding the frequency, consequences, or forensic significance of subdural rebleeding should be considered speculative.

Under What Circumstances Do Subdural Hemorrhages Rebleed?

Nonacute subdural collections can rebleed spontaneously. As we have discussed previously, the transformation of a delayed, space-filling, posttraumatic, subdural hygroma into a chronic subdural hematoma with potential mass effect requires rebleeding. Nonacute subdural collections also rebleed as a consequence of minor cranial impact.

Consider this case example. An 11-month-old boy was admitted to the hospital with nausea, recurrent vomiting, and irritability. He was treated with intravenous fluids and then discharged. He was readmitted one month later with persistent, chronic irritability. His neurological examination upon readmission was nonfocal and his parents denied any knowledge of closed head trauma. Cranial CT scan at that time revealed a nonacute subdural hematoma overlying the right cerebral hemisphere anteriorly, swelling of the right cerebral hemisphere with midline shifting,

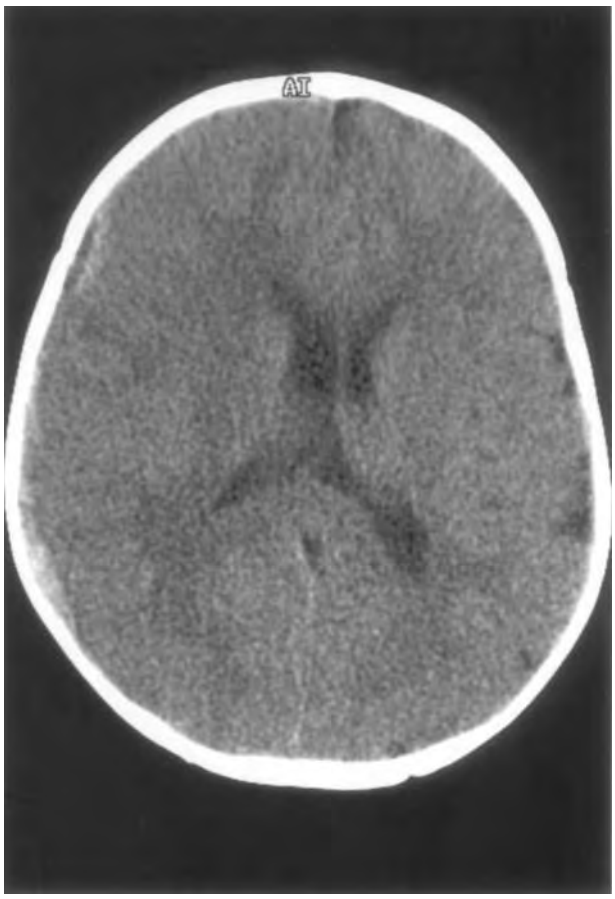

FIGURE 4: **This 11-Month-Old Boy Was Hospitalized and Treated With Intravenous Fluids for His Recurrent Vomiting and Irritability. He Was Readmitted One Month Later With Persistent Clinical Signs. Neurological Exam at That Time Was Nonfocal and His Caregivers Denied Knowledge of Closed Head Trauma. Nevertheless, CT Scan Revealed a Nonacute Subdural Hematoma Overlying the Right Cerebral Hemisphere, Swelling of the Right Cerebral Hemisphere With Midline Shifting, and Probable Atrophy of the Left Cerebral Hemisphere.**

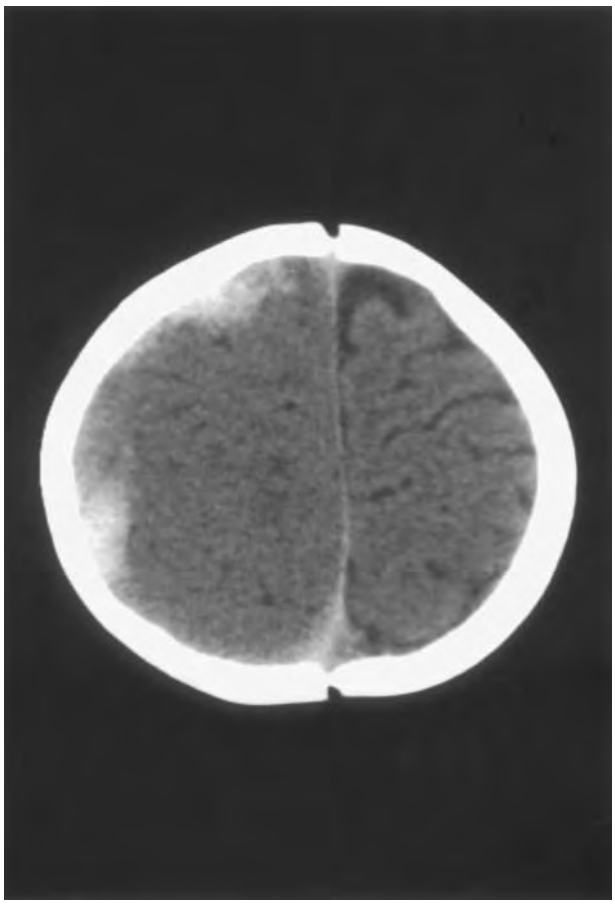

FIGURE 5: **On the Evening His Nonacute Subdural Collection Was Identified, This 11-Month-Old Child Fell From His Hospital Bed. Although He Suffered No Acute Loss of Consciousness, He Appeared Less Active Following the Fall and Became More Lethargic Over the Next Few Hours. Late That Evening, He Manifested Intermittent Periodic Breathing and Bradycardia. Urgent Cranial CT Scan Revealed Increased Swelling of the Right Hemisphere, Acute Subdural Rebleeding on the Right, and Worsening Mass Effect With Increased Midline Shifting. Neurosurgical Intervention Confirmed Acute Subdural Rebleeding.**

and probable atrophy of the left cerebral hemisphere (see Figure 4).

On the evening of admission, his nurse forgot to lower his top bed railing. Although his fall was not directly witnessed, his nurse found the child on his back on the floor next to his bed. He suffered no acute loss of consciousness and was consolable but appeared overall less active following the fall. Over the next few hours, he became more lethargic and late that evening manifested intermittent periodic breathing and bradycardia. An urgent cranial CT scan revealed increased swelling of the right hemisphere, increased volume of the acute, right-sided subdural collection, and worsening mass effect with increased midline shifting (see Figure 5). The child underwent acute neurosurgical intervention. A large, acute subdural hematoma was evacuated, along with an older component of subdural hematoma. Ophthalmologic examination revealed scattered intraretinal hemorrhages. After his neurosurgery, this young child improved clinically and had a favorable long-term outcome.

We postulate the following explanation for the clinical findings in this case. At one year of age, skull and brain maturation had facilitated his older subdural collection to begin to act with mass effect. Clinically, these changes manifested as persistent irritability. Acute subdural rebleeding triggered by his minor fall on the evening of his hospitalization accelerated these adverse mass effects and resulted in clinical deterioration over several hours. Because the fall

from his hospital bed did not represent a clinically significant cranial deceleration event, this young child did not suffer immediate loss of consciousness, apnea, seizure, hypotension, or prolonged traumatic coma.

To date, no prospective, comparative studies have measured the frequency of subdural rebleeding—or its clinical consequences, specifically in young children with known chronic subdural collections. Acute rebleeding within a chronic subdural collection during infancy may represent inflicted re-injury. Because the history in such cases is frequently unreliable, a prospective study of this type may be impossible to accomplish (Kleinman & Barnes, 1998).

CONCLUSIONS

To conclude that an infant's intracranial hemorrhage or rebleeding resulted from inflicted cranial injury or re-injury may have serious forensic consequences. How can we objectively make this determination? We recommend the following approach.

1. Thoroughly image the affected child's extra-axial collection(s). In most cases, a combination of CT and MR cranial imaging will be required.
2. In consultation with the pediatric radiologist or neuroradiologist, objectively characterize the extra-axial collection as an expansion of the subdural space, the subarachnoid space, or both.
3. If applicable, consider the differential diagnosis for expansion of the subarachnoid space (see Table 1).
4. If applicable, consider the differential diagnosis for subdural hemorrhage (see Table 2).
5. Exclude nontraumatic causes for subdural bleeding by appropriate history, physical examination, radiological imaging, and/or laboratory evaluation (see Table 2).
6. In consultation with the pediatric radiologist or neuroradiologist, objectively estimate the age(s) of the child's subdural collection(s).
7. In cases demonstrating heterogeneity in the radiological appearance of a subdural collection, consult with a pediatric radiologist or neuroradiologist to differentiate between hyperacute subdural hemorrhage, subdural rebleeding (occurring spontaneously or induced by re-injury), or other potential explanations for the heterogeneity (see Table 6).
8. If applicable, attempt to differentiate between subdural hygroma (acting as a space-filling lesion) and chronic subdural hematoma (potentially acting with mass effect) (see Table 3).
9. If neurosurgical intervention is required, make sure that the drained subdural fluid or clot, and the surrounding tissues, are carefully collected for biochemical and histopathological analysis. Review these results to ascertain the composition of the subdural collection and to verify the presence or absence of a neomembrane.
10. Finally, attempt to differentiate between subdural rebleeding and re-injury by considering the child's acute clinical presentation (see Table 5 and Figure 3). Was the acute clinical presentation predictable? In the majority of cases, severe cranial re-injury is associated with acute neurological deterioration, brain swelling, and/or retinal hemorrhages—findings not otherwise associated with spontaneous rebleeding within a chronic subdural hematoma (Kleinman & Barnes, 1998).

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P171

CEREBRAL OXYGENATION CHANGES FOLLOWING SEVERE BRAIN TRAUMA COULD BE INDICATIVE OF NEUROVASCULAR UNCOUPLING IN A MODEL OF LATERAL FLUID PERCUSSION BRAIN INJURY USING JUVENILE AND ADULT PIGS

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Aim: To evaluate disturbances in cerebral oxygen consumption and perfusion following varying degrees of traumatic brain injury in a porcine model of fluid percussion brain injury. To identify if these changes are a function of age.
Methods: 24 pigs were divided in 2 groups (Group A, 8 to 10kg n = 12, Group B, above 30kg n = 12). Injury was created using lateral fluid percussion. Three levels of injury were used in each group. Mild (0.5 to 1.5 atm.), Moderate (1.5 to 3 atm.) and severe (more than 3 atm.). Cerebral oxygenation was studied using Near infrared spectroscopy (NIRS) and perfusion was estimated using Laser Doppler flowmetry. 30 minutes of baseline data was collected and used as control followed by 90 minutes of data collection after TBI.
Results: The graphs at the bottom represent 2 patterns observed.
Fig. 1 represents NIRS data observed following an injury of 1.7atm. in a 10kg piglet. The corresponding laser Doppler flowmetry indicated an increase in cerebral perfusion. Fig 2 represents NIRS data observed following an injury of 4.5 atm. In a 10kg piglet. The corresponding laser Doppler flowmetry indicated a decrease in cerebral perfusion. There were no significant differences observed in these patterns between adult and juvenile pigs.
Discussion:
Lesser levels of brain injury(less than 3 atm.) was characterised by an early increase in deoxyhemoglobin followed by an increase in oxyhemoglobin with a corresponding increase in cerebral microvascular perfusion. Higher levels of brain injury (more than 3atm.) were characterised by a steep increase in deoxyhemoglobin which remained high and was not matched by a corresponding increase in oxyhemoglobin. Laser Doppler flowmetry indicated a decrease in cerebral microvascular perfusion. With lesser levels of injury the increased metabolic demand of the brain is well compensated and matched by an increase in oxygenated haemoglobin following an increase in cerebral perfusion. With higher trauma the increase in metabolic demand doesn't appear to be compensated by an increase in oxygen delivery and cerebral perfusion and could represent neurovascular uncoupling. This phenomenon was equally divided between both the porcine age groups studied.

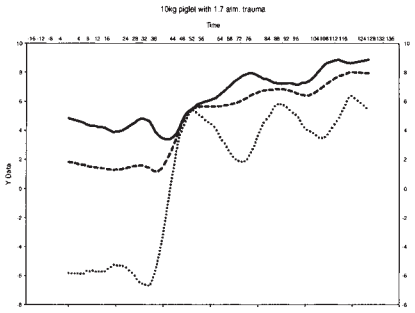


Fig. 1

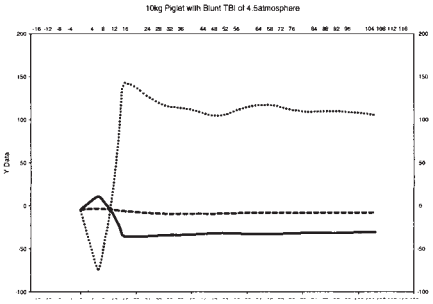
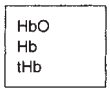


Fig. 2



HbO(Oxyhemoglobin),Hb(deoxyhemoglobin),tHb(total Hemoglobin)

P172

BIOMECHANICAL PREDICTORS FOR MILD TRAUMATIC BRAIN INJURY

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ABSTRACT WITHDRAWN

P173

THE RESPONSE OF TODDLER AND INFANT HEADS DURING VIGOROUS SHAKING

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Introduction: Shaking is a commonly reported history provided to clinicians in cases of abuse in young children. However, it remains unclear whether shaking without impact can cause severe brain injury. By gaining a better understanding of the biomechanics of pediatric head injury, clinicians can better evaluate, treat and prevent inflicted head trauma. Previously, we determined shaking frequency, angular accelerations, and event durations of a 1.5 month old anthropomorphic dummy head during abusive shaking conditions (Prange et al. ***J Neurosurg***, 2003). The current study expands on this research and determines shaking frequency, angular acceleration and shaking event duration of an 18 month-old "toddler" dummy we have created using published anthropometry and mechanical properties of skull and neck.
Methods: Volunteers were instructed to grasp the dummy's torso firmly, hold it at chest level and shake back and forth forcefully. Linear and angular accelerations of the head were measured and calculated, respectively, using a 9-accelerometer array. Linear accelerations of the body were measured using a 3-accelerometer array.
Results: Average shaking cycle frequency (mean $\pm$ sd) of the toddler dummy head (2.18 $\pm$ 0.23 Hz) was similar to that of the infant (3.08 $\pm$ 1.0 Hz). However, the peak sagittal angular acceleration of the toddler head (0.48 $\pm$ 0.17 krad/s^2) was significantly lower ($p < 0.005$) than that of the infant (4.14 $\pm$ 2.47 krad/s^2), likely because the infant head experienced a full range of motion during a shake while the toddler head did not. Despite the reduced motion range, the duration of a single shake was significantly longer ($p < 0.04$) in the toddler (485 $\pm$ 96 msec) than in the infant (359 $\pm$ 119 msec).
Conclusions: Differences in the shaking responses of the two models are likely the result of variations in body weight and neck properties. The toddler neck was stiffer than the infant neck to account for developmental changes (Nuckley et al. ***Bone***, 2004; Pintar et al. ***Stapp Car Crash***, 2000) , and the overall weight of the toddler (11 kg) was more than twice that of the infant (4.8 kg). These data indicate that it may be difficult to deliver large inertial forces to the toddler head by shaking alone. Future work will explore other mechanisms of head injury for this age group, including impact-related events. Supported by NIH RO1-NS39679.

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Evidence based case report

Perimacular retinal folds from childhood head trauma

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Editorials by Geddes and Plunkett and Harding et al

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A previously healthy 14 month old child was transferred to our medical centre with a severe head injury. The father had collected the boy and his 3 year old brother from their mother at his workplace car park and taken them home while their mother went to work. The children had been watching television while the father prepared dinner. After hearing something fall, the father found the boy on the floor with the television covering the right side of the head and anterior chest. A homemade television stand was partially across the child's lower legs. His older brother stated, "television fell." As soon as the father removed the television, he noticed the child's head beginning to swell. A neighbour drove them to the local hospital. According to the father and the neighbour, the child never stopped breathing and no resuscitative efforts were attempted.

Cranial computed tomography showed extensive head injuries. He had soft tissue swelling of the scalp, diffuse cerebral oedema with a subdural haematoma overlying the frontal convexities and layering along the falx cerebri, a left sided skull fracture adjacent to a widely diastatic coronal suture, cerebral contusions beneath the fracture, and a rightward midline shift measuring 8 mm. The paediatric ophthalmologist described bilateral dot and blot intraretinal haemorrhages, preretinal haemorrhages, and perimacular retinal folds (fig 1).

The child's condition deteriorated, and he died 18 hours after the incident. Child Protective Services removed the 3 year old sibling from the home because the retinal haemorrhages and retinal folds were considered diagnostic of abusive head trauma from shaking. This action was taken despite the father's repeated detailed, consistent account provided to emergency staff, the paediatric child abuse specialist, paediatric intensive care doctors, and law enforcement authorities.

Postmortem evidence

A forensic autopsy showed no direct trauma to the orbits or eyes. There were prominent bilateral scalp contusions with soft tissue and intramuscular haemorrhage, symmetrical parietal skull fractures with coronal sutural diastasis, and a lacerated dura mater with extrusion of brain and blood. In addition to bilateral subdural and subarachnoid haemorrhages, a thin epidural haematoma partially covered the frontoparietal, calvarial lamina interna. The brain showed bilateral cortical contusions, severe cerebral oedema, and diffuse anoxic-ischemic injury. Postmortem ocular examination showed haemorrhages of the optic nerve sheaths with subdural haemorrhage greater than subarachnoid haemorrhage. Both eyes had extensive retinal haemorrhages with perimacular retinal folds (fig 2). Retinoschisis and peripapillary intrascleral haemorrhages were evident, and the retinal haemorrhages extended from the posterior pole to the ora serrata affecting the preretinal, intraretinal, and subretinal layers.

When investigators went to the house to recover the television before the family returned home, it was still on the carpeted floor. The 480 mm screen television with built in videocassette recorder weighed 19.5 kg. The homemade television stand measured 762 mm (height)×635 mm (width)×508 mm (depth) and had a bottom drawer that held videotapes. A greasy smudged area on the glass of the television corresponded with the impact site on the child's head.

A re-enactment in which a 11.4 kg weight (similar to the child's weight at autopsy of 11.8 kg) was placed on the partially opened drawer caused the television and

P+ *Details of the included studies are on bmj.com*

television stand to readily topple forward. According to investigators, the family home was 7.8 km from the workplace and about 6 km from the local hospital. Based on the distance and estimated driving times plus workplace time clock records, the father was home with the children about 20 minutes when the incident happened. The day after the incident, while in foster care, the 3 year old sibling corroborated the father's account. Despite all this evidence, the paediatric ophthalmologist repeated that perimacular retinal folds coincident with retinal haemorrhages were considered specific for shaken baby syndrome secondary to retinal traction exerted by the oscillating vitreous.

Search for published evidence

We were unable to find a published report of perimacular retinal folds in a childhood non-abusive head injury. We therefore did a systematic review of the medical literature on perimacular retinal folds associated with abusive head trauma in infants and young children. Our background question became: "In infants and young children with an acute intracranial injury, are perimacular retinal folds specific for head injury from vitreoretinal traction occurring during cycles of acceleration and deceleration (shaken baby syndrome)?"

We searched the Medline (1966-2003) database using the terms retinal folds and child abuse and uncovered seven non-comparative case series articles.[1–7] We also examined references cited in these articles plus review articles and book chapters on ocular findings in child abuse mentioning or discussing perimacular retinal folds relative to non-accidental head injury. Similar searches in the Cochrane Library, ISI Web of Science, and Ovid found no additional articles.

Results

We found 42 articles and book chapters discussing perimacular retinal folds in childhood abusive head trauma. Seventeen mentioned the presence of retinal folds in non-accidental head injury but did not comment on specificity or formative mechanism. A table on bmj.com gives details of the remaining articles. All but two of the articles are non-comparative clinical or autopsy case series, case reports, review articles, or book chapters.

The two studies that included controls both showed bias in selection of controls and contained no cases with perimacular retinal folds but discussed the postulated causal mechanism.[8 9] In the prospective controlled study, the authors reported on 79 children younger than 3 years who had sustained head injuries.[8] The manner of injury in one case was indeterminate. Three children, including one who died, had non-accidental head injury diagnosed, all of whom had retinal haemorrhages; 72 of the 75 children with non-abusive injuries were managed by observation alone. No perimacular retinal folds were observed; however, the presumed causative mechanism of traumatic retinoschisis and retinal folds was discussed.

The second controlled study was a prospective autopsy study that examined the presence and location of ocular findings in 169 childhood deaths.[9] Ocular haemorrhages (retinal, peripheral retinal, optic nerve sheath and intrascleral) were more likely in craniocerebral trauma than in non-head injuries and natural diseases. Although case selection was purportedly random, the study contained a disproportionately high number of deaths from child abuse compared with natural and non-abusive causes. Case selection depended on the pathologist's willingness to participate in the study, and we were told by one of the authors that pathologists were more willing to participate when they believed that the deaths were abusive or suspicious (M Gilliland, personal communication, 2002). Perimacular retinal folds were not noted, but the authors concluded that acceleration-deceleration injury to the retina accounts for peripheral retinal haemorrhages and retinal folds.

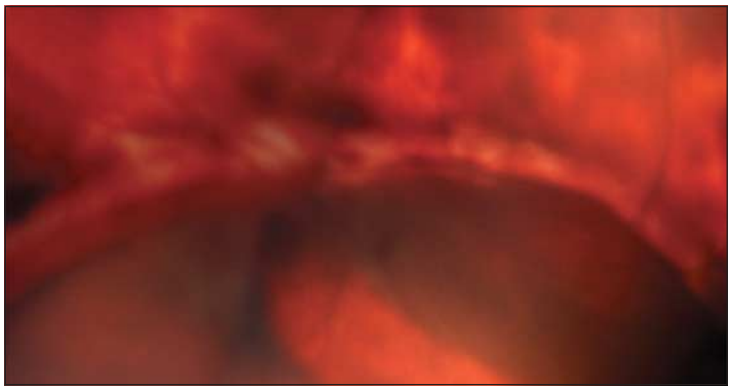

Fig 1 Clinical image highlighting temporal portion of perimacular retinal fold at 2-3 o'clock area in left eye with a blood vessel bending over the fold (magnification ×6)

Supporting evidence

The references cited to support statements about the specificity or causal mechanism of perimacular retinal folds and abusive head injury in the articles we found are all non-comparative observational reports, unsystematic review articles, and book chapters. Seventy per cent of the articles cited four non-comparative case series.[1 2 3 10] We assessed the quality of this evidence.

Gaynon et al reported on two infants with presumed shaken baby syndrome who had retinal folds and concluded that these folds may be a hallmark

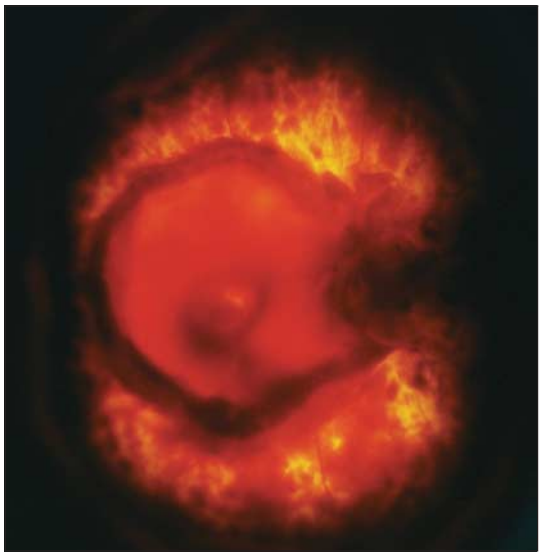

Fig 2 Transilluminated retinal image of right eye at autopsy showing circinate, elevated, perimacular retinal fold and extensive retinal haemorrhages

of shaking injuries in child abuse victims.[1] One infant reportedly fell 1.5 m to the floor while being carried down a stairway.

Massicotte et al reported the ocular findings at autopsy of three children with perimacular retinal folds.[2] Two infants had sustained direct head trauma, but in the other there was no physical or forensic evidence of direct head trauma. They observed that the vitreous had partially separated from the retina but remained attached to the internal limiting membrane at the apices of the folds and the vitreous base. They concluded that their study confirmed the role of vitreous traction in formation of perimacular folds and proved that shaking alone caused these folds and shaking was never an accidental phenomenon.

Elner et al reviewed the ocular and autopsy findings in 10 consecutive children who died of suspected child abuse.[3] Perimacular retinal folds were observed in three children, all of whom had evidence of blunt head injuries.

Greenwald et al reported five cases of children in whom definite or probable physical abuse during infancy was associated with traumatic retinoschisis.[10] They hypothesised that when an infant is shaken, the head is subjected to repetitive accelerations and decelerations causing the relatively dense lens to move forward and back within the ocular fluids. Transmission of force through firm attachments between the lens, vitreous gel, and particularly the macular retina presumably would result in appreciable traction on the retina causing it to split and creating the surrounding folds.

Discussion

Statements in the medical literature that perimacular retinal folds are diagnostic of shaken baby syndrome are not supported by objective scientific evidence. Non-comparative observational reports and unsystematic narrative review articles contain insufficient evidence to provide unbiased support for or against diagnostic specificity, and inferences about associations, causal or otherwise, cannot be determined. Clinical and autopsy evidence of ocular lesions must therefore be considered alongside other physical findings and a thorough investigation before concluding whether a head injury is caused by abuse. The child in our case had ocular haemorrhages (peripheral retinal, optic nerve sheath and intrascleral) and retinoschisis, which again some people consider specific for child abuse. Unfortunately, the evidence for these assumptions has similar problems to that for perimacular retinal folds. An evidence based analyis of indexed medical publications on shaken baby syndrome from 1966-1998 uncovered a weak scientific evidence base.[11] Selection bias, inappropriate controls, and the lack of precise criteria for case definition were identified as important problems with the data. Many studies committed a fallacy of assumption, selecting cases by the presence of the clinical findings that were sought as diagnostically valid. Unsystematic reviews and consensus statements often mingled opinion with facts and added no original supporting evidence.

Perimacular retinal folds are associated with increased neurological morbidity and mortality in infants and children with abusive head injuries.[6] The reported incidence of perimacular retinal folds in shaken baby syndrome varies from 6% in a consecutive clinical case series to 50% in a sequential autopsy case series.[5 12] Clinical and autopsy studies with appropriately matched controls are needed to determine the causal mechanism of perimacular retinal folds and their specificity for abusive head injury. Until good evidence is available, we urge caution in interpreting eye findings out of context.

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Severe retinal hemorrhages in infants with aggressive, fatal *Streptococcus pneumoniae* meningitis

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Retinal hemorrhages in children occasionally accompany bacterial meningitis, usually due to *hemophilus* or meningococcal organisms. The hemorrhages may be intraretinal, usually in the posterior pole of the eye and few in number, or, more uncommonly, subhyaloid or vitreous. Pathogenesis may include vasculitis, disseminated intravascular coagulation, or intracranial hypertension. We report 2 cases of bilateral severe retinal hemorrhages in fatal *Streptococcus pneumoniae* meningitis.

Case 1

A 16-day-old girl born at term by spontaneous vaginal delivery following uncomplicated pregnancy was well and breastfeeding. After 3 days of decreasing feeding, intermittent fever, and irritability, she was admitted to a community hospital for observation. Initial urinalysis, blood and urine cultures, blood count, and serum electrolytes were normal. On the third day she became acutely ill over a period of approximately 2 hours with a bulging fontanel, pallor, and lethargy. A lumbar puncture was performed and intravenous ceftriaxone was given. Seizures and apnea developed and she was intubated. Intravenous ampicillin, fresh frozen plasma, and red blood cell transfusion were given prior to the child's transfer to the Hospital for Sick Children in Toronto, Canada.

On arrival, the baby was afebrile and hypotensive, with dilated fixed pupils. Head circumference was 50th percentile. Hemoglobin was 9.4 g/dL, platelets 100,000, and white cell count 3.1 with normal differential. Electrolytes were normal. Coagulation studies were not done. Neurological assessment showed no evidence of cerebral activity. Physical examination was otherwise normal. Lumbar puncture results from the community hospital revealed cloudy fluid with 901 white blood cells, 7 red blood cells, protein 3.66 g/L, and glucose 11 mg%, with Gram-positive cocci. The blood culture and spinal fluid cultures were subsequently positive for *S. pneumoniae* sensitive to all antibiotics.

Ophthalmology consultation revealed predominantly intraretinal hemorrhages too numerous to count throughout the retina bilaterally in a radiating pattern from the optic nerve to the ora serrata consistent with central retinal vein occlusion (Figure 1A). Retinal veins were dilated but often obscured by the massive hemorrhaging. The macula in each eye was flat and appeared completely avascular with no cherry-red spot, suggesting central retinal artery occlusion combined with infarction of the choroidal circulation (Figure 1B). Retinal arteries were extremely attenuated. There was no papilledema.

The child died within 24 hours. Autopsy revealed grossly purulent cerebrospinal fluid, including around the optic nerves. The ocular pathology slides and report were not made available for this study.

Case 2

A boy born at 38 weeks following a normal pregnancy was diagnosed with cystic fibrosis and primary immune neutropenia at 4 months old following *Pseudomonas aeruginosa* sepsis. At 13 months of age, after 3 days of upper respiratory symptoms, fever, and 1 day of vomiting, he presented stuporous with a respiratory rate of 14/min, tachycardia, and delayed capillary refill. There was flexor upper limb reaction to painful stimuli and bilateral, unreactive to light, fixed dilated pupils. The computed tomographic scan of head, neck, chest, and abdomen was normal. Myocardiopathy, pulmonary edema, metabolic acidosis, and neutropenia (3300/mm^3) ensued. Electrolytes (including sodium 134.8 mm/L) were normal. Hemoglobin was 13.7 g/dL, prothrombin 46 (normal range, 70-120), and activated partial thromboplastin time 30 (normal range, 23-36). Platelet count was normal.

On the same day a pediatric ophthalmologist (JPL) performed indirect ophthalmoscopy and found bilateral edematous (right greater than left) optic disks, numerous right eye nerve fiber layer hemorrhages, and left eye dot intraretinal hemorrhages. Hemorrhages extended from the optic disk to the ora serrata bilaterally. In the right eye a large subinternal limiting membrane macular hemorrhage was present without retinal folds.

Lumbar puncture showed opening pressure of 76 cm H_2O, grossly purulent cerebrospinal fluid, total proteins of 1.380 g/L, and glucose of 4 mg%. Culture was positive for *S. pneumoniae*. The patient died 12 hours after presentation. Autopsy was not performed.

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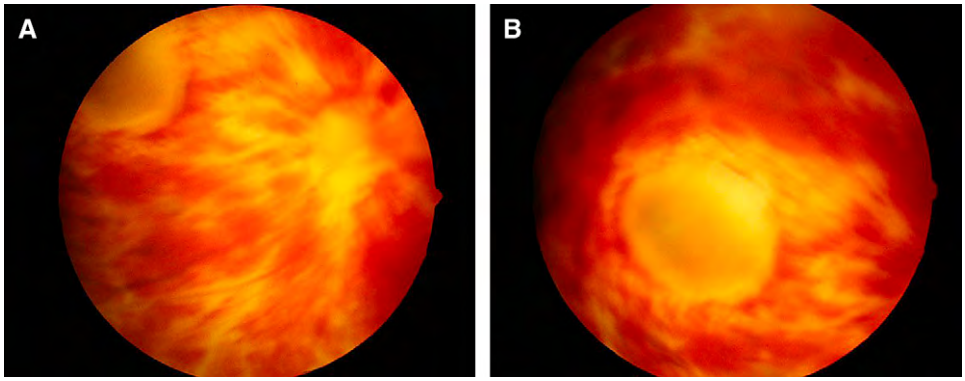

FIG 1. Fundus photographs obtained from Case 1 after admission. A, Severe radiating pattern of intraretinal hemorrhage consistent with central retinal vein obstruction in right eye. Optic nerve is to the right and nonperfused macula on the upper left. Note attenuated and nonperfused (not visible) retinal arteries. A single engorged retinal vein is seen running through the bottom half of the image. B, Nonperfused macula of right eye without cherry-red spot, consistent with combined central retinal artery occlusion and infarction of choroidal circulation.

Discussion

Dinakaran and colleagues[1] found retinal hemorrhages in 5 (42%) of 12 children with meningococcal septicemia and disseminated intravascular coagulation, but the authors did not mention the number of children who had meningitis. Hemorrhages were less than 20, intraretinal, and posterior to the equator. Subhyaloid retinal hemorrhages have also been reported in children with *hemophilus* meningitis.[2] Fraser and colleagues[3] described large unilateral subretinal and vitreous hemorrhage in a 12-year-old girl with meningococcal meningitis and normal clotting studies. To the best of our knowledge, ours is the first report of retinal hemorrhages in *S. pneumoniae* meningitis and the first report of extensive retinal hemorrhages in meningitis of any kind.

Our cases had fulminant fatal pneumococcal meningitis. In Case 1 the child clinically had a combined central retinal vein and artery occlusion with choroidal infarction. It appears this resulted from arrest of the circulation from the purulent cerebrospinal fluid. Central retinal vein obstruction in meningitis has been reported.[4] Unlike the typical central retinal vein obstruction pattern of retinal hemorrhage in Case 1, children with severe hemorrhagic retinopathy due to abusive head injury have a more random distribution of hemorrhages at all levels.[6]

Case 2 also had grossly purulent cerebrospinal fluid but a hemorrhage pattern not consistent with vein or artery occlusion. Papilledema, hypercoagulability, and sepsis may have contributed. The pathogenesis of the subinternal limiting membrane blood remains unclear, although we suspect that peripheral and macular intraretinal vessels may have necrosed from the infection.

The extensive retinal hemorrhages, in particular, the macular schisis-like lesion in Case 2, are reminiscent of abusive head injury.[5,6] Such patients can also present with no history of trauma and a sudden change in mental status. Evaluation for meningitis may be an important part of the evaluation of patients with extensive retinal hemorrhages, particularly when there is underlying predisposition to infection and no evidence of intracranial trauma.

S. pneumoniae is the most common cause of community-acquired pneumonia, meningitis, and bacteremia in children and adults. Invasive pneumococcal disease primarily affects young children, older adults, and individuals with comorbidities or impaired immune systems. Case fatality rates range from 10% to 30% in adults but are much lower (<3%) in children.[7] Most patients with pneumococcal meningitis present nonacutely after hours or days of developing signs and symptoms that may be nonspecific. Pneumococcal meningitis carries a greater risk of death and significant neurological disability than other causes of bacterial meningitis.[8]

Literature Search

MEDLINE was searched for the period from 1960 to the present for the following terms: *retinal hemorrhages/haemorrhages*, *meningitis*, *bacterial*, *pneumococcus*, and *sepsis*.

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Why is ZOVIRAX so effective, yet virtually nontoxic?

Selectivity.

ZOVIRAX (acyclovir) selectively stops the virus from replicating and very rarely affects the normal, healthy cell's metabolism. Since the prevention of herpes genitalis infections is presently unattainable, the most effective treatment is early intervention with ZOVIRAX Ointment 5%.

The importance of stopping viral activity (the virus from replicating) cannot be overemphasized. It is the mandatory first step in noticeably altering the clinical course of herpes simplex infections. In many ways, stopping viral activity is analogous to therapy of bacterial infections in which the primary objective is to eradicate offending pathogens.

Studies have demonstrated that the potent antiviral effect of ZOVIRAX significantly decreases the number of days that the virus is actively present at the lesion site. Studies in first episodes of genital herpes showed that following the eradication of the viral activity there is a subsequent and significant amelioration of the other clinical signs and symptoms such as sores, pain and itching.

ZOVIRAX selectively inhibits viral replication

ZOVIRAX is preferentially taken up by virus-infected cells because it is selectively activated by a virus-specified enzyme (thymidine kinase-TK). Once activated by the virus itself, ZOVIRAX is rapidly converted by cellular enzymes to its active form (acyclovir triphosphate). Conversion of ZOVIRAX is reported to be 30 to 120 times faster in the virus-infected cell than in the uninfected cell.[1]

In its active form, ZOVIRAX is capable of interfering with viral replication by either (1) competitively inhibiting the viral DNA polymerase, or (2) being incorporated into the viral DNA chain and thereby causing chain termination.[1-4] (See schematic.)

"Because virus-specified TK is manyfold more potent in activating acyclovir than is cell-specified TK, acyclovir is highly selective. It inhibits viral replication in infected cells but spares uninfected host cells and their functions."[5]

ZOVIRAX rarely affects normal cell metabolism

As noted above, the key to the effectiveness and safety of ZOVIRAX is the *preferential uptake* and *selective activation* of the drug by the virus-infected cell. The virus itself acts as the catalyst in this activation process. Therefore, in the healthy, uninfected cell, ZOVIRAX is virtually nontoxic.

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INDICATIONS AND USAGE: Zovirax (Acyclovir) Ointment 5% is indicated in the management of initial herpes genitalis and in limited nonlife-threatening cutaneous Herpes simplex virus infections in immunocompromised patients. In clinical trials of initial herpes genitalis, Zovirax Ointment 5% has shown a decrease in healing time and in some cases a decrease in duration of viral shedding and duration of pain. In studies in immunocompromised patients with mainly herpes labialis, there was a decrease in duration of viral shedding and a slight decrease in duration of pain.

By contrast, in studies of recurrent herpes genitalis and of herpes labialis in nonimmunocompromised patients, there was no evidence of clinical benefit; there was some decrease in duration of viral shedding.

Diagnosis: Whereas cutaneous lesions associated with Herpes simplex infections are often characteristic, the finding of multinucleated giant cells in smears prepared from lesion exudate or scrapings may assist in the diagnosis! Positive cultures for Herpes simplex virus offer a reliable means for confirmation of the diagnosis. In genital herpes, appropriate examinations should be performed to rule out other sexually transmitted diseases.

CONTRAINDICATIONS: Zovirax Ointment 5% is contraindicated for patients who develop hypersensitivity or chemical intolerance to the components of the formulation.

WARNINGS: Zovirax Ointment 5% is intended for cutaneous use only and should not be used in the eye.

PRECAUTIONS:
General: The recommended dosage, frequency of applications, and length of treatment should not be exceeded (see DOSAGE AND ADMINISTRATION). There exist no data which demonstrate that the use of Zovirax Ointment 5% will either prevent transmission of infection to other persons or prevent recurrent infections when applied in the absence of signs and symptoms. Zovirax Ointment 5% should not be used for the prevention of recurrent HSV infections. Although clinically significant viral resistance associated with the use of Zovirax Ointment 5% has not been observed, this possibility exists.

Drug Interactions: Clinical experience has identified no interactions resulting from topical or systemic administration of other drugs concomitantly with Zovirax Ointment 5%.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Acyclovir was tested in lifetime bioassays in rats and mice at single daily doses of 50, 150 and 450 mg/kg/day given by gavage. These studies showed no statistically significant difference in the incidence of benign and malignant tumors produced in drug-treated as compared to control animals, nor did acyclovir induce the occurrence of tumors earlier in drug-treated animals as compared to controls. In 2 *in vitro* cell transformation assays, used to provide preliminary assessment of potential oncogenicity in advance of these more definitive lifetime bioassays in rodents, conflicting results were obtained. Acyclovir was positive at the highest dose used in one system and the resulting morphologically transformed cells formed tumors when inoculated into immunosuppressed, syngeneic, weanling mice. Acyclovir was negative in another transformation system.

No chromosome damage was observed at maximum tolerated parenteral doses of 100 mg/kg acyclovir in rats or Chinese hamsters; higher doses of 500 and 1000 mg/kg were clastogenic in Chinese hamsters. In addition, no activity was found in a dominant lethal study in mice. In 9 of 11 microbial and mammalian cell assays, no evidence of mutagenicity was observed. In 2 mammalian cell assays (human lymphocytes and L5178Y mouse lymphoma cells *in vitro*), positive response for mutagenicity and chromosomal damage occurred, but only at concentrations at least 1000 times the plasma levels achieved in man following topical application.

Acyclovir does not impair fertility or reproduction in mice at oral doses up to 450 mg/kg/day or in rats at subcutaneous doses up to 25 mg/kg/day. In rabbits given a high dose of acyclovir (50 mg/kg/day, s.c.), there was a statistically significant decrease in implantation efficiency.

ORIGINAL CONTRIBUTION
child abuse, shaken baby syndrome; pediatrics, shaken baby syndrome; shaken baby syndrome

Shaken Baby Syndrome: A Review of 20 Cases

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Twenty cases of shaken baby syndrome are reviewed to determine important signs, symptoms, physical findings, laboratory parameters, and prognosis. The signs and symptoms of this form of head trauma are nonspecific. The findings may mimic infection, intoxication, or metabolic abnormalities. Diagnosis depends on a high index of suspicion and the physical findings of a bulging fontanelle, head circumference greater than the 90th percentile, and retinal hemorrhage. The finding of bloody fluid from a lumbar or subdural tap is also highly suggestive. Computed cranial tomography findings confirm the diagnosis. The prognosis in the shaken baby syndrome is poor. Three of our 20 patients died and ten others sustained significant morbidity. The emergency physician must be alert to making this diagnosis in order to promptly institute therapy for acute head trauma. [Ludwig S, Warman M: Shaken baby syndrome: A review of 20 cases. Ann Emerg Med February 1984;13:104-107.]

INTRODUCTION

Child abuse and neglect occur frequently in our society. In the United States, there continues to be a steady increase in the number of abuse cases reported each year. The manifestations of abuse and neglect are many, often making the diagnosis difficult. Early case recognition and reporting by the emergency physician are key to the overall success of case management.

Caffey's[1] first descriptions of child abuse included children who had long bone metaphyseal fractures and intracranial hemorrhages without external signs of abuse. Years later, he called this syndrome the whiplash shaken infant syndrome or the shaken baby syndrome (SBS), as it was realized that such injuries were caused by severe shaking of infants by parents as a form of discipline.[2,3] Other authors have confirmed Caffey's observations and the likely mechanism and pathogenesis of the injuries.[4-8]

With the advent of computed cranial tomography (CCT), the SBS can be more accurately diagnosed, yet it was our impression that often there is a delay in suspecting and making the diagnosis. The purposes of this review were the following: 1) to document the frequency of children with known shake injuries; 2) to determine important signs, symptoms, physical findings, and laboratory parameters; 3) to document the type and severity of injury caused by shaking; and 4) to increase the emergency physician's suspicion for and ability to detect this form of child abuse.

METHODS

All official child abuse reporting forms sent to the County Child Protective Services Agency from the Children's Hospital of Philadelphia during the years 1977 to 1982 were reviewed. Cases of possible shake injury were selected based on parent's admission of shaking or suspicion by medical staff of this form of abuse when the history and evaluation could not account for the patient's injuries.

Only children injured solely by being shaken were included in the study. Therefore, patients with other evidence of abuse (external head trauma, skull fracture, multiple skeletal fractures, burns, or patterned or severe bruising) were excluded despite the possibility of their having concomitant shake injuries. Information regarding the patient's history, clinical findings, and diag-

nostic evaluation was collected from emergency department documents, the in-patient chart, and the social work department records.

RESULTS

Demographics

Twenty of the 1,250 records reviewed met our criteria for SBS, including those of 14 boys and 6 girls. The age range was one month to 15 months (Table 1). The mean age was 5.8 months, and the median age was 3 months.

Signs and Symptoms

The most frequent chief complaint (given by the parent in 12 cases) was a respiratory problem. Other signs and symptoms included apparent central nervous abnormalities such as irritability, lethargy, seizures, or decreased muscle tone. The relative frequencies of these complaints are shown (Table 2). There were a number of associated gastrointestinal symptoms, including poor appetite, vomiting, and constipation. Shaking seldom was mentioned by the parent. In three cases, however, parents did admit to shaking the infant in an effort to perform resuscitation. Recent accidental injury was described in eight cases.

Physical Findings

The incidence of common physical findings is shown (Table 3). Significant alterations in the vital signs occurred commonly, including bradycardia in 13 cases, apnea/bradypnea in nine cases, and hypothermia in nine cases.

The anterior fontanelle was enlarged or bulging in 11 cases (55%), and in nine of 16 (56%) the head circumference was greater than the 90th percentile. Retinal hemorrhages were present in 12 of 18 cases examined by ophthalmoscopy. Three children had nuchal rigidity. Nonspecific bruising was noted in seven cases and, although suggestive of trauma, neither the pattern nor the severity was considered strongly indicative of abuse. Observed neurologic signs included irritability and lethargy. Other neurologic findings included anisocoria, seizure, posturing, gaze preference, and nystagmus.

Laboratory

Abnormal laboratory findings are summarized (Table 4). Leukocytosis was present in nine children. Twelve children underwent lumbar puncture, and in ten cases the spinal fluid was bloody. Ten patients had subdural taps, and eight were positive. Other studies were not positive in a significant number of children and were not helpful in making the diagnosis.

When CCT was performed, the findings were diagnostic of SBS. The findings included subdural hemorrhage (ten cases), cerebral contusion (eight cases), and subarachnoid hemorrhage (five cases).

Outcome

The patient outcomes are shown (Table 5). Three children (15%) died. Of those who survived, ten (50%) had significant morbidity. These deficits included blindness, visual impairment, motor impairment, seizures, and developmental delay. Seven children (35%) survived with no apparent morbidity.

DISCUSSION

In this retrospective look at the shaken baby syndrome (SBS), the most striking finding is the high rate of mortality and morbidity. The mortality rate (15%) and the morbidity rate (50%) emphasize the importance of early identification and prompt institution of treatment. Even those children (35%) who apparently escaped without sequelae may manifest some abnormality if followed for a longer period. Caffey[2,3] has suggested that undetected cases of shake injury may be responsible for many cases of mental retardation with no apparent etiology. Long-term follow-up studies may demonstrate an even higher rate of significant morbidity.

Important to reducing the morbidity and mortality is the early detection of SBS. The emergency physician should keep in mind a number of important indicators. First, the age of the child. As shown in this series and in previous reports,[2,4,8,9] SBS occurs in young children; the median age in our study was 5.8 months. Young children are most vulnerable to this form of injury because of several factors: 1) relatively large head size and weight; 2) weak neck muscles; 3) thin, friable central nervous system vasculature; and 4) a soft brain that is less myelinated than in the adult. Younger children may also be more vulnerable because of their proclivity to cry, at times for no apparent reason. This leaves the parents feeling frustrated, angry, and inadequate. Older children are also shaken, but their stronger neck muscles may modify the acceleration-deceleration forces that result in SBS.

Parents are rarely forthright in admitting their loss of control. In eight cases in this series, parents did give a history of prior accidental injury. Three parents also described shaking the child in an effort to resuscitate. In this latter group, it was impossible to determine what caused the child's need for resuscitation. Perhaps aspiration, seizure, dysrhythmia, or primary apnea were the root causes. Still other parents when questioned claimed only to have played roughly with their child, and described activities in which the head was not supported. Rough play may be implicated in rare cases.

Rather than a history of abuse, most parents presented the problem in terms of respiratory abnormality. The finding of respiratory distress in the absence of stridor or lower airway sounds should cause the physician to look for a central cause of respiratory distress. Other parental complaints were also varied and quite nonspecific. When the physician did not suspect SBS, infectious diagnoses such as meningitis and bacterial sepsis were invoked. Other diagnostic considerations included metabolic diseases, electrolyte disturbances, seizure disorders, and infantile botulism.

The physician's examination more often led to a diagnosis of SBS than did the chief complaint or the history. In particular, the finding of a full or bulging fontanelle was telling, as described previously.[7-9] Till's series[10] of infants with subdural hematomas and effusions demonstrated a 63% incidence of tense anterior fontanelle, which correlates well with the 55% incidence we observed. Head circumference measurements and funduscopic examinations, when performed, were also important indicators. These parts of the physical examination were not uniformly completed in the emergency department, perhaps due to lack of experience on the part of the emergency physician or the technical difficulty in examining the infant's fundi. There also appeared to be some reluctance to use mydriatics in a situation in which pupillary response was an important sign to monitor.

Funduscopic examinations revealed a high incidence of retinal hemorrhages in our cases. This finding has been described by several authors[11-15] as being strongly suggestive of trauma and intracranial hemorrhage. Zimmerman[4] reported 13 cases of abuse that met our criteria for SBS with a 77% incidence of retinal hemorrhage. Till[10] observed a 50% incidence, but noted that this figure would have been higher had more time been spent on the examination. With the most frequently made misdiagnosis, 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.

It has been suggested that violent shaking may cause extreme rises in intraocular and intracranial venous pressure with resultant retinal hemorrhaging likened to that of Purtscher's retinopathy.[14] However, Ober[11] and others have described ocular hemorrhage in the absence of intracranial hemorrhage or pressure increases. Ober suggested that the acceleration-deceleration forces associated with shaking are the cause of the retinal hemorrhage.

In seven children (35%) nonspecific bruises were seen. Although nonspecific bruising on older children is very common, we think that any bruising on a young infant (less than six months) should be considered with suspicion.

Leukocytosis was noted in 45% of cases but was not particularly helpful in making the diagnosis. In some instances, it supported the physician's erroneous suspicion that he was treating an infection. Several other laboratory studies were helpful in establishing the diagnosis. Blood in the cerebrospinal fluid (CSF) was a helpful finding. However, it was noted in sev-

TABLE 1. *Shaken baby syndrome age distribution (N = 20)*

Age (mo)	No.
1	2
2	6
3	3
4	1
5	2
6	1
7	0
8 or older	5

TABLE 2. *Shaken baby syndrome signs and symptoms*

Signs/Symptoms	No.*
Respiratory problem	12
CNS problem	16
Lethargic	5
Irritable	4
Seizure	4
Floppy/limp	3
Gastrointestinal problem	11
Diminished appetite	6
Vomiting	3
Constipation	2
Trauma history	11
Recent minor accident	8
Shaking to resuscitate	3

*More than one sign or symptom given for each patient.

TABLE 3. *Shaken baby syndrome physical findings*

Physical Finding	No. (%)*
Vital sign changes	
Bradycardia	13/20 (65)
Apnea/bradycardia	9/20 (45)
Hypothermia	9/20 (45)
Cranial examination	
Full or bulging fontanelle	11/20 (55)
Head circumference 90%	9/16 (56)
Retinal hemorrhages	
Unilateral	3/15 (20)
Bilateral	9/15 (60)
Unilateral/bilateral	12/15 (80)
Neurologic findings	
Lethargy/coma	9/20 (45)
Irritable	6/20 (30)
Nuchal rigidity	3/10 (33)
Anisocoria	3/10 (33)
Seizure	6/20 (30)
Posturing	3/20 (15)
Gaze disturbance	7/14 (50)
Integument	
Nonspecific bruising	7/20 (35)

*Number positive over number evaluated.

TABLE 4. *Shaken baby syndrome laboratory findings*

Laboratory Finding	No. (%)*
Leukocytosis	9/20 (45)
Bloody CSF	10/12 (83)
Bloody subdural tap	8/10 (80)
CCT	20/20 (100)
Subdural hemorrhage	10
Cerebral contusion	8
Subarachnoid hemorrhage	5

*Number positive over number evaluated.

TABLE 5. *Shaken baby syndrome outcome (N = 20)*

Outcome	No. (%)
Mortality	3 (15)
Morbidity	10 (50)
Blindness	4
Visual loss	3
Motor deficit	5
Seizure	7
Developmental delay	6
No apparent deficit	7 (35)

eral instances that the physician assumed that the CSF was bloody due to a traumatic lumbar puncture rather than from CNS hemorrhage. Subdural taps revealed bloody fluid in eight of ten cases. This finding was virtually diagnostic.

The CCT was the key diagnostic study in identifying SBS. A number of different lesions were observed, including subdural hematoma, cerebral contusion, and subarachnoid bleed. Zimmerman et al[4] reported the finding of a characteristic parieto-occipital acute interhemispheric subdural hematoma. There was often associated parenchymal injury. In long-term follow-up Zimmerman et al[4] found cerebral infarction in 50% of the cases and cerebral atrophy in 100%. Ellison and colleagues[16] reported cerebral contusion in children who did not have signs of extensive injury and were presumably shaken.

SUMMARY

We reviewed 20 cases of SBS and found a tragically high incidence of morbidity and mortality. Our findings indicate that the young infant manifesting respiratory alterations in the face of a normal pulmonary examination may have been shaken. Other children may develop nonspecific signs and symptoms. The physical findings of tense or bulging fontanelle, head circumference greater than 90th percentile for age, and retinal hemorrhage strengthen the diagnosis. Blood in the cerebral spinal fluid obtained on lumbar or subdural puncture indicates intracranial hemorrhage. Computed cranial tomography may specifically confirm SBS by showing acute interhemispheric subdural hematoma or cerebral contusion in the absence of external trauma. Early recognition of SBS will lead to prompt management of the intracranial hemorrhage and subsequent cerebral edema.

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Severity of Illness and Injury in Pediatric Air Transport

To assess severity of illness or injury in pediatric patients undergoing air transport, we prospectively evaluated 636 patients during 29 months of service. All patients were classified by age, diagnosis, and method and distance of transport. Therapeutic intervention scoring system (TISS) scores were calculated in all patients, Glasgow coma scale (GCS) scores were used in patients with altered level of consciousness, and Modified Injury Severity Scale (MISS) scores were used in patients with multiple trauma. A total of 57.5% of patients were transported by helicopter, 37.5% by fixed-wing aircraft, and 5% by ground transport. Mean distance of transport was 207 miles. Age ranged from 3 weeks to 16 years, with 45% of children under 1 year of age. Trauma (24.6%), neurologic disease (24.2%), and respiratory failure (20%) were the most common diagnoses. Eighty-one percent of patients were taken to surgery or admitted to the intensive care unit immediately on arrival at the regional children's hospital. Mean TISS score was 36.7, with 51% of patients having TISS scores greater than 30. The mean MISS score was 34.5, and 75% of patients had MISS scores greater than 25. Nineteen percent of patients had GCS scores less than or equal to 8. Overall mortality was 7%, with 9% mortality in patients with trauma versus 6.3% in nontraumatic diseases. TISS scores greater than 30, MISS scores greater than 25, and GCS scores less than or equal to 8 were associated with increased mortality ($P < .01$). [Mayer TA, Walker ML: Severity of illness and injury in pediatric air transport. Ann Emerg Med February 1984;13:108-111.]

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INTRODUCTION

In recent years, significant emphasis has been placed on regionalization of certain aspects of critical care, including major trauma, head injury, burns, spinal cord injury, and pediatrics. The need for rapid and expert transportation to specialized centers has led to the development of air transport programs in many areas of the country. While these programs have received popular support and have undoubtedly been beneficial to many of the patients who have been transported, detailed analyses of the costs and benefits from air transport programs are lacking. We have reported a cost analysis of one such program, indicating that transportation could be delivered without exorbitant cost to the patient or the hospital.[1]

Because air transport systems involve a great deal of time, effort, and expense, such systems should responsibly account for the type and number of patients transported. Ideally air transport and regionalization of pediatric care should be limited to those patients with serious illnesses or injuries.[2] To assess the severity of illness or injury in pediatric patients transported by an air transport system, we prospectively evaluated 636 patients treated during the initial 29 months of operation of the Pediatric LifeFlight service of the Primary Children's Medical Center, Salt Lake City.

METHODS

During a 29-month period (April 1979 through August 1981) all patients transported by the Pediatric LifeFlight program were classified prospectively according to age, diagnosis, and method and distance of transport. Depending on the patient's condition, either a physician and flight nurse or two flight nurses were sent on transport. All physicians and nurses had undergone in-

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Fatal Pediatric Head Injuries Caused by Short-Distance Falls

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Physicians disagree on several issues regarding head injury in infants and children, including the potential lethality of a short-distance fall, a lucid interval in an ultimately fatal head injury, and the specificity of retinal hemorrhage for inflicted trauma. There is scant objective evidence to resolve these questions, and more information is needed. The objective of this study was to determine whether there are witnessed or investigated fatal short-distance falls that were concluded to be accidental. The author reviewed the January 1, 1988 through June 30, 1999 United States Consumer Product Safety Commission database for head injury associated with the use of playground equipment. The author obtained and reviewed the primary source data (hospital and emergency medical services' records, law enforcement reports, and coroner or medical examiner records) for all fatalities involving a fall.

The results revealed 18 fall-related head injury fatalities in the database. The youngest child was 12 months old, the oldest 13 years. The falls were from 0.6 to 3 meters (2–10 feet). A noncaretaker witnessed 12 of the 18, and 12 had a lucid interval. Four of the six children in whom funduscopic examination was documented in the medical record had bilateral retinal hemorrhage. The author concludes that an infant or child may suffer a fatal head injury from a fall of less than 3 meters (10 feet). The injury may be associated with a lucid interval and bilateral retinal hemorrhage.

Key Words: Child abuse—Head injury—Lucid interval—Retinal hemorrhage—Subdural hematoma.

Many physicians believe that a simple fall cannot cause serious injury or death (1–9), that a lucid interval does not exist in an ultimately fatal pediatric head injury (7–13), and that retinal hemorrhage is highly suggestive if not diagnostic for inflicted trauma (7,12,14–21). However, several have questioned these conclusions or urged caution when interpreting head injury in a child (15,22–28). This controversy exists because most infant injuries occur in the home (29,30), and if there is history of a fall, it is usually not witnessed or is seen only by the caretaker. Objective data are needed to resolve this dispute. It would be helpful if there were a database of fatal falls that were witnessed or wherein medical and law enforcement investigation unequivocally concluded that the death was an accident.

The United States Consumer Product Safety Commission (CPSC) National Injury Information Clearinghouse uses four computerized data sources (31). The National Electronic Injury Surveillance System (NEISS) file collects current injury data associated with 15,000 categories of consumer products from 101 U.S. hospital emergency departments, including 9 pediatric hospitals. The file is a probability sample and is used to estimate the number and types of consumer product–related injuries each year (32). The Death Certificate (DC) file is a demographic summary created by information provided to the CPSC by selected U.S. State Health Departments. The Injury/Potential Injury Incident (IR) file contains summaries, indexed by consumer product, of reports to the CPSC from consumers, medical examiners and coroners (Medical Examiner and Coroner Alert Project [MECAP]), and newspaper accounts of product-related incidents discovered by local or regional CPSC staff (33). The In-Depth Investigations (AI) file contains summaries of investigations performed by CPSC staff based on reports received from the NEISS, DC, or IR files (34). The AI files provide details about the incident from victim and witness interviews, accident reconstruction, and review of law enforce-

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ment, health care facility, and coroner or medical examiner records (if a death occurred).

METHODS

I reviewed the CPSC, DC, IR, and AI files for all head and neck injuries involving playground equipment recorded by the CPSC from January 1, 1988 through June 30, 1999. There are 323 entries in the playground equipment IR file, 262 in the AI file, 47 in the DC file, and more than 75,000 in the NEISS file. All deaths in the NEISS file generated an IR or AI file. If the file indicated that a death had occurred from a fall, I obtained and reviewed each original source record from law enforcement, hospitals, emergency medical services (EMS), and coroner or medical examiner offices except for one autopsy report. However, I discussed the autopsy findings with the pathologist in this case.

RESULTS

There are 114 deaths in the Clearinghouse database, 18 of which were due to head injury from a fall. The following deaths were excluded from this study: those that involved equipment that broke or collapsed, striking a person on the head or neck (41); those in which a person became entangled in the equipment and suffocated or was strangled (45), those that involved equipment or incidents other than playground (6 [including a 13.7-meter fall from a homemade Ferris wheel and a 3-meter fall from a cyclone fence adjacent to a playground]); and falls in which the death was caused exclusively by neck (carotid vessel, airway, or cervical spinal cord) injury (4).

The falls were from horizontal ladders (4), swings (7), stationary platforms (3), a ladder attached to a slide, a "see-saw", a slide, and a retaining wall. Thirteen occurred on a school or public playground, and five occurred at home. The database is not limited to infants and children, but a 13-year-old was the oldest fatality (range, 12 months–13 years; mean, 5.2 years; median, 4.5 years). The distance of the fall, defined as the distance of the closest body part from the ground at the beginning of the fall, could be determined from CPSC or law enforcement reconstruction and actual measurement in 10 cases and was 0.6 to 3.0 meters (mean, 1.3 $\pm$ 0.77; median, 0.9). The distance could not be accurately determined in the seven fatalities involving swings and one of the falls from a horizontal ladder, and may have been from as little as 0.6 meters to as much as 2.4 meters. The maximum height for a fall from a swing was assumed to be the highest point of the arc. Twelve of the 18 falls were witnessed by a noncaretaker or were videotaped; 12 of the children had a lucid interval (5 minutes–48 hours); and 4 of the 6 in whom funduscopic examination was performed had bilateral retinal hemorrhage (Table 1).

CASES

Case 1

This 12-month-old was seated on a porch swing between her mother and father when the chain on her mother's side broke and all three fell sideways and backwards 1.5 to 1.8 meters (5–6 feet) onto decorative rocks in front of the porch. The mother fell first, then the child, then her father. It is not known if her father landed on top of her or if she struck only the ground. She was unconscious immediately. EMS was called; she was taken to a local hospital; and was ictal and had decerebrate posturing in the emergency room. She was intubated, hyperventilated, and treated with mannitol. A computed tomography (CT) scan indicated a subgaleal hematoma at the vertex of the skull, a comminuted fracture of the vault, parafalcine subdural hemorrhage, and right parietal subarachnoid hemorrhage. There was also acute cerebral edema with effacement of the right frontal horn and compression of the basal cisterns. She had a cardiopulmonary arrest while the CT scan was being done and could not be resuscitated.

Case 2

A 14-month-old was on a backyard "see-saw" and was being held in place by his grandmother. The grandmother said that she was distracted for a moment and he fell backward, striking the grass-covered ground 0.6 meters (22.5 inches) below the plastic seat. He was conscious but crying, and she carried him into the house. Within 10 to 15 minutes he became lethargic and limp, vomited, and was taken to the local hospital by EMS personnel. He was unconscious but purposefully moving all extremities when evaluated, and results of funduscopic examination were normal. A CT scan indicated an occipital subgaleal hematoma, left-sided cerebral edema with complete obliteration of the left frontal horn, and small punctate hemorrhages in the left frontal lobe. There was no fracture or subdural hematoma. He was treated with mannitol; his level of consciousness rapidly improved; and he was extubated. However, approximately 7 hours after admission he began to have difficulty breathing, both pupils suddenly dilated, and he was rein-

TABLE 1. *Summary of cases*

No.	CPSC No.	Age	Sex	Fall from	Distance M/F	Witnessed	Lucid interval	Retinal hemorrhage	Subdural hemorrhage	Autopsy	Cause of death	FP
1	DC 9108013330	12 mos	F	Swing	1.5–1.8/5.0–6.0	No	No	N/R	Yes +IHF	No	Complex calvarial fracture with edema and contusions	No
2	AI 890208HBC3088	14 mos	M	See-saw	0.6/2.0	No	10–15 minutes	No	No	No	Malignant cerebral edema with herniation	No
3	IR F910368A	17 mos	F	Swing	1.5–1.8/5.0–6.0	No	No	N/R	Yes +IHF	Yes	Acute subdural hematoma with secondary cerebral edema	Yes
4	AI 921001HCC2263	20 mos	F	Platform	1.1/3.5	No	5–10 minutes	Bilateral multilayered	Yes +IHF	Limited	Occipital fracture with subdural/subarachnoid hemorrhage progressing to cerebral edema and herniation	Yes
5[a]	DC 9312060661	23 mos	F	Platform	0.70/2.3	Yes	10 minutes	Bilateral, NOS	Yes	Yes	Acute subdural hematoma	Yes
6	DC 9451016513	26 mos	M	Swing	0.9–1.8/3.0–6.0	Yes	No	Bilateral multilayered	Yes +IHF	Yes	Subdural hematoma with associated cerebral edema	Yes
7[a]	AI 891215HcC2094	3 yrs	M	Platform	0.9/3.0	Yes	10 minutes	N/R	Yes	No	Acute cerebral edema with herniation	No
8	AI 910515HCC2182	3 yrs	F	Ladder	0.6/2.0	yes	15 minutes	N/R	Yes (autopsy only)	Yes	Complex calvarial fracture, contusions, cerebral edema with herniation	Yes
9	DC 9253024577	4 yrs	M	Slide	2.1/7.0	Yes	3 hours	N/R	No	Yes	Epidural hematoma	Yes
10	AI 920710HWE4014	5 yrs	M	Horizontal ladder	2.1/7.0	No	No	N/R	Yes	No	Acute subdural hematoma with acute cerebral edema	Yes
11	AI 960517HCC5175	6 yrs	M	Swing	0.6–2.4/2.0–8.0	No	10 minutes	No	Yes +IHF	No	Acute subdural hematoma	Yes
12	AI 970324HCC3040	6 yrs	M	Horizontal ladder	3.0/10.0	Yes	45 minutes	N/R	No	No	Malignant cerebral edema with herniation	Yes
13	AI 881229HCC3070	6 yrs	F	Horizontal ladder	0.9/3.0	Yes	1+ hour	N/R	Yes +IHF	Yes	Subdural and subarachnoid hemorrhage, cerebral infarct, and edema	Yes
14	AI 930930HWE5025	7 yrs	M	Horizontal ladder	1.2–2.4/4.0–8.0	Yes	48 hours	N/R	No	Yes	Cerebral infarct secondary to carotid/vertebral artery thrombosis	Yes
15	AI 970409HCC1096	8 yrs	F	Retaining wall	0.9/3.0	Yes	12+ hours	N/R	Yes (autopsy only)	Yes	Acute subdural hematoma	Yes
16	AI 890621HCC3195	10 yrs	M	Swing	0.9–1.5/3.0–5.0	Yes	10 minutes	Bilateral multilayered	Yes	Yes	Acute subdural hematoma contiguous with an AV malformation	No
17	AI 920428HCC1671	12 yrs	F	Swing	0.9–1.8/3.0–6.0	Yes	No	N/R	No	Yes	Occipital fracture with extensive contra-coup contusions	Yes
18	AI 891016HCC1511	13 yrs	F	Swing	0.6–1.8/2.0–6.0	Yes	No	N/R	Yes +IHF	Yes	Occipital fracture, subdural hemorrhage, cerebral edema	Yes

[a]The original CT scan for case #7 and the soft tissue CT windows for case #5 could not be located and were unavailable for review.

CPSC, Consumer Products Safety Commission; AI, accident investigation; IR, incident report; DC, death certificate; M, male; F, female; Distance, the distance of the closest body part from the ground at the start of the fall (see text); M/F, meters/feet; Witnessed, witnessed by a noncaretaker or videotaped; N/R, not recorded; IHF, including interhemispheric or falx; FP, forensic pathologist–directed death investigation system.

tubated. A second CT scan demonstrated progression of the left hemispheric edema despite medical management, and he was removed from life support 22 hours after admission.

Case 3

This 17-month-old had been placed in a baby carrier–type swing attached to an overhead tree limb at a daycare provider's home. A restraining bar held in place by a snap was across her waist. She was being pushed by the daycare provider to an estimated height of 1.5 to 1.8 meters (5–6 feet) when the snap came loose. The child fell from the swing on its downstroke, striking her back and head on the grassy surface. She was immediately unconscious and apneic but then started to breathe spontaneously. EMS took her to a pediatric hospital. A CT scan indicated a large left-sided subdural hematoma with extension to the interhemispheric fissure anteriorly and throughout the length of the falx. The hematoma was surgically evacuated, but she developed malignant cerebral edema and died the following day. A postmortem examination indicated symmetrical contusions on the buttock and midline posterior thorax, consistent with impact against a flat surface; a small residual left-sided subdural hematoma; cerebral edema with anoxic encephalopathy; and uncal and cerebellar tonsillar herniation. There were no cortical contusions.

Case 4

A 20-month-old was with other family members for a reunion at a public park. She was on the platform portion of a jungle gym when she fell from the side and struck her head on one of the support posts. The platform was 1.7 meters (67 inches) above the ground and 1.1 meters (42 inches) above the top of the support post that she struck. Only her father saw the actual fall, although there were a number of other people in the immediate area. She was initially conscious and talking, but within 5 to 10 minutes became comatose. She was taken to a nearby hospital, then transferred to a tertiary-care facility. A CT scan indicated a right occipital skull fracture with approximately 4-mm of depression and subarachnoid and subdural hemorrhage along the tentorium and posterior falx. Funduscopic examination indicated extensive bilateral retinal and preretinal hemorrhage. She died 2 days later because of uncontrollable increased intracranial pressure. A limited postmortem examination indicated an impact subgaleal hematoma overlying the fracture in the mid occiput.

Case 5

A 23-month-old was playing on a plastic gym set in the garage at her home with her older brother. She had climbed the attached ladder to the top rail above the platform and was straddling the rail, with her feet 0.70 meters (28 inches) above the floor. She lost her balance and fell headfirst onto a 1-cm (¾-inch) thick piece of plush carpet remnant covering the concrete floor. She struck the carpet first with her outstretched hands, then with the right front side of her forehead, followed by her right shoulder. Her grandmother had been watching the children play and videotaped the fall. She cried after the fall but was alert and talking. Her grandmother walked/carried her into the kitchen, where her mother gave her a baby analgesic with some water, which she drank. However, approximately 5 minutes later she vomited and became stuporous. EMS personnel airlifted her to a tertiary-care university hospital. A CT scan indicated a large right-sided subdural hematoma with effacement of the right lateral ventricle and minimal subfalcine herniation. (The soft tissue windows for the scan could not be located and were unavailable for review.) The hematoma was immediately evacuated. She remained comatose postoperatively, developed cerebral edema with herniation, and was removed from life support 36 hours after the fall. Bilateral retinal hemorrhage, not further described, was documented in a funduscopic examination performed 24 hours after admission. A postmortem examination confirmed the right frontal scalp impact injury. There was a small residual right subdural hematoma, a right parietal lobe contusion (secondary to the surgical intervention), and cerebral edema with cerebellar tonsillar herniation.

Case 6

A 26-month-old was on a playground swing being pushed by a 13-year-old cousin when he fell backward 0.9 to 1.8 meters (3–6 feet), striking his head on hard-packed soil. The 13-year-old and several other children saw the fall. He was immediately unconscious and was taken to a local emergency room, then transferred to a pediatric hospital. A CT scan indicated acute cerebral edema and a small subdural hematoma adjacent to the anterior interhemispheric falx. A funduscopic examination performed 4 hours after admission indicated extensive bilateral retinal hemorrhage, vitreous hemorrhage in the left eye, and papilledema. He had a subsequent cardiopulmonary arrest and could not be resuscitated. A postmortem examination confirmed the retinal hemorrhage and indicated a right parietal scalp impact injury but no calvarial frac-

," of bilateral subdural hemorrhage, ma with herniation, and focal hemor- right posterior midbrain and pons.

ear-old with a history of TAR (thrombo- absent radius) syndrome was playing children on playground equipment at his hen he stepped through an opening in a . He fell 0.9 meters (3 feet) to the hard- ground, striking his face. A teacher wit- he incident. He was initially conscious and walk. However, approximately 10 minutes e had projectile vomiting and became co- , was taken to a local hospital, and subse- y transferred to a pediatric hospital. A CT indicated a small subdural hematoma and dif- cerebral edema with uncal herniation, accord- o the admission history and physical examina- . (The original CT report and scan could not be ated and were unavailable for review.) His telet count was 24,000/mm^3, and he was treated pirically with platelet transfusions, although he d no evidence for an expanding extra-axial mass. esuscitation was discontinued in the emergency oom.

Case 8

This 3-year-old was at a city park with an adult neighbor and four other children, ages 6 to 10. She was standing on the third step of a slide ladder 0.6 meters (22 inches) above the ground when she fell forward onto compact dirt, striking her head. The other children but not the adult saw the fall. She was crying but did not appear to be seriously injured, and the neighbor picked her up and brought her to her parents' home. Approximately 15 minutes later she began to vomit, and her mother called EMS. She was taken to a local emergency room, then transferred to a pediatric hospital. She was initially lethargic but responded to hyperventilation and mannitol; she began to open her eyes with stimulation and to spontaneously move all extremities and was extubated. However, she developed malignant cerebral edema on the second hospital day and was reintubated and hyperventilated but died the following day. A postmortem examination indicated a subgaleal hematoma at the vertex of the skull associated with a complex fracture involving the left frontal bone and bilateral temporal bones. There were small epidural and subdural hematomas (not identifiable on the CT scan), bilateral "contra-coup" contusions of the inferior surfaces of the frontal and temporal lobes, and marked cerebral edema with uncal herniation.

Case 9

A 4-year-old fell approximately 2.1 meters (7 feet) from a playground slide at a state park, landing on the dirt ground on his buttock, then falling to his left side, striking his head. There was no loss of consciousness, but his family took him to a local emergency facility, where an evaluation was normal. However, he began vomiting and complained of left neck and head pain approximately 3 hours later. He was taken to a second hospital, where a CT scan indicated a large left parietal epidural hematoma with a midline shift. He was transferred to a pediatric hospital and the hematoma was evacuated, but he developed malignant cerebral edema with right occipital and left parietal infarcts and was removed from the respirator 10 days later. A postmortem examination indicated a small residual epidural hematoma, marked cerebral edema, bilateral cerebellar tonsillar and uncal herniation, and hypoxic encephalopathy. There was no identifiable skull fracture.

Case 10

A 5-year-old was apparently walking across the horizontal ladder of a "monkey bar," part of an interconnecting system of homemade playground equipment in his front yard, when his mother looked out one of the windows and saw him laying face down on the ground and not moving. The horizontal ladder was 2.1 meters (7 feet) above compacted dirt. EMS were called, he was taken to a local hospital, and then transferred to a pediatric hospital. A CT scan indicated a right posterior temporal linear fracture with a small underlying epidural hematoma, a 5-mm thick acute subdural hematoma along the right temporal and parietal lobes, and marked right-sided edema with a 10-mm midline shift. He was hyperventilated and treated with mannitol, but the hematoma continued to enlarge and was surgically evacuated. However, he developed uncontrollable cerebral edema and was removed from life support 10 days after the fall.

Case 11

A 6-year-old was on a playground swing at a p vate lodge with his 14-year-old sister. His sis heard a "thump," turned around, and saw him the grass-covered packed earth beneath the sw The actual fall was not witnessed. The seat o swing was 0.6 meters (2 feet) above the gr and the fall distance could have been from as as 2.4 meters (8 feet). He was initially con and talking but within 10 minutes becan matose and was taken to a local emergency then transferred to a tertiary-care hospital

scan indicated a large left frontoparietal subdural hematoma with extension into the anterior interhemispheric fissure and a significant midline shift with obliteration of the left lateral ventricle. There were no retinal hemorrhages. He was treated aggressively with dexamethasone and hyperventilation, but there was no surgical intervention. He died the following day.

Case 12

This 6-year-old was at school and was sitting on the top crossbar of a "monkey bar" approximately 3 meters (10 feet) above compacted clay soil when an unrelated noncaretaker adult saw him fall from the crossbar to the ground. He landed flat on his back and initially appeared to have the wind knocked out of him but was conscious and alert. He was taken to the school nurse who applied an ice pack to a contusion on the back of his head. He rested for approximately 30 minutes in the nurse's office and was being escorted back to class when he suddenly collapsed. EMS was called, and he was transported to a pediatric hospital. He was comatose on admission, the fundi could not be visualized, and a head CT scan was interpreted as normal. However, a CT scan performed the following morning approximately 20 hours after the fall indicated diffuse cerebral edema with effacement of the basilar cisterns and fourth ventricle. There was no identifiable subdural hemorrhage or calvarial fracture. He developed transtentorial herniation and died 48 hours after the fall.

Case 13

This 6-year-old was playing on a school playground with a 5th grade student/friend. She was hand-over-hand traversing the crossbar of a "monkey bar" 2.4 meters (7 feet 10 inches) above the ground with her feet approximately 1 meter (40 inches) above the surface. She attempted to slide down the pole when she reached the end of the crossbar but lost her grip and slid quickly to the ground, striking the compacted dirt first with her feet, then her buttock and back, and finally her head. The friend informed the school principal of the incident, but the child seemed fine and there was no intervention. She went to a relative's home for after-school care approximately 30 minutes after the fall, watched TV for a while, then complained of a headache and laid down for a nap. When her parents arrived at the home later that evening, 6 hours after the incident, they discovered that she was incoherent and "drooling." EMS transported her to a tertiary-care medical center. A CT scan indicated a right parieto-occipital skull fracture, subdural and subarachnoid hemorrhage, and a right cerebral hemisphere infarct. The infarct included the posterior cerebral territory and was thought most consistent with thrombosis or dissection of a right carotid artery that had a persistent fetal origin of the posterior cerebral artery. She remained comatose and was removed from the respirator 6 days after admission. A postmortem examination indicated superficial abrasions and contusions over the scapula, a prominent right parietotemporal subgaleal hematoma, and a right parietal skull fracture. She had a 50-ml subdural hematoma and cerebral edema with global hypoxic or ischemic injury ("respirator brain"), but the carotid vessels were normal.

Case 14

A 7-year-old was on the playground during school hours playing on the horizontal ladder of a "monkey bar" when he slipped and fell 1.2 to 2.4 meters (4–8 feet). According to one witness, he struck his forehead on the bars of the vertical ladder; according to another eyewitness he struck the rubber pad covering of the asphalt ground. There are conflicting stories as to whether he had an initial loss of consciousness. However, he walked back to the school, and EMS was called because of the history of the fall. He was taken to a local hospital, where evaluation indicated a Glasgow coma score of 15 and a normal CT scan except for an occipital subgaleal hematoma. He was kept overnight for observation because of the possible loss of consciousness but was released the following day. He was doing homework at home 2 days after the fall when his grandmother noticed that he was stumbling and had slurred speech, and she took him back to the hospital. A second CT scan indicated a left carotid artery occlusion and left temporal and parietal lobe infarcts. The infarcts and subsequent edema progressed; he had brainstem herniation; and he was removed from life support 3 days later (5 days after the initial fall). A postmortem examination indicated ischemic infarcts of the left parietal, temporal, and occipital lobes, acute cerebral edema with herniation, and thrombosis of the left vertebral artery. Occlusion of the carotid artery, suspected premortem, could not be confirmed.

Case 15

This 8-year-old was at a public playground near her home with several friends her age. She was hanging by her hands from the horizontal ladder of a "monkey bar" with her feet approximately 1.1 meters (3.5 feet) above the ground when she attempted to swing from the bars to a nearby 0.9-

meter (34-inch) retaining wall. She landed on the top of the wall but then lost her balance and fell to the ground, either to a hard-packed surface (one witness) or to a 5.1-cm (2-inch) thick resilient rubber mat (a second witness), striking her back and head. She initially cried and complained of a headache but continued playing, then later went home. Her mother said that she seemed normal and went to bed at her usual time. However, when her mother tried to awaken her at approximately 8:30 the following morning (12 hours after the fall) she complained of a headache and went back to sleep. She awoke at 11 a.m. and complained of a severe headache then became unresponsive and had a seizure. EMS took her to a nearby hospital, but she died in the emergency room. A postmortem examination indicated a right temporoparietal subdural hematoma, extending to the base of the brain in the middle and posterior fossae, with flattening of the gyri and narrowing of the sulci. (The presence or absence of herniation is not described in the autopsy report.) There was no calvarial fracture, and there was no identifiable injury in the scalp or galea.

Case 16

A 10-year-old was swinging on a swing at his school's playground during recess when the seat detached from the chain and he fell 0.9 to 1.5 meters (3–5 feet) to the asphalt surface, striking the back of his head. The other students but not the three adult playground supervisors saw him fall. He remained conscious although groggy and was carried to the school nurse's office, where an ice pack was placed on an occipital contusion. He suddenly lost consciousness approximately 10 minutes later, and EMS took him to a local hospital. He had decerebrate posturing when initially evaluated. Funduscopic examination indicated extensive bilateral confluent and stellate, posterior and peripheral preretinal and subhyaloid hemorrhage. A CT scan showed a large acute right frontoparietal subdural hematoma with transtentorial herniation. The hematoma was surgically removed, but he developed malignant cerebral edema and died 6 days later. A postmortem examination indicated a right parietal subarachnoid AV malformation, contiguous with a small amount of residual subdural hemorrhage, and cerebral edema with anoxic encephalopathy and herniation. There was no calvarial fracture.

Case 17

A 12-year-old was at a public playground with a sister and another friend and was standing on the seat of a swing when the swing began to twist. She lost her balance and fell 0.9 to1.8 meters (3–6 feet) to the asphalt surface, striking her posterior thorax and occipital scalp. She was immediately unconscious and was taken to a tertiary-care hospital emergency room, where she was pronounced dead. A postmortem examination indicated an occipital impact injury associated with an extensive comminuted occipital fracture extending into both middle cranial fossa and "contra-coup" contusions of both inferior frontal and temporal lobes.

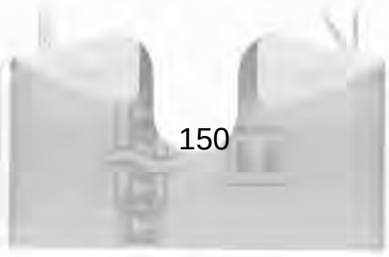
Case 18

This 13-year-old was at a public playground with a friend. She was standing on the seat of a swing with her friend seated between her legs when she lost her grip and fell backwards 0.6 to 1.8 meters (2–6 feet), striking either a concrete retaining wall adjacent to the playground or a resilient 5.1-cm (2 inch) thick rubber mat covering the ground. She was immediately unconscious and was given emergency first aid by a physician who was nearby when the fall occurred. She was taken to a nearby hospital and was purposefully moving all extremities and had reactive pupils when initially evaluated. A CT scan indicated interhemispheric subdural hemorrhage and generalized cerebral edema, which progressed rapidly to brain death. A postmortem examination indicated a linear nondepressed midline occipital skull fracture, subdural hemorrhage extending to the occiput, contusion of the left cerebellar hemisphere, bifrontal "contra-coup" contusions, and cerebral edema.

DISCUSSION

General

Traumatic brain injury (TBI) is caused by a force resulting in either strain (deformation/unit length) or stress (force/original cross-sectional area) of the scalp, skull, and brain (35–37). The extent of injury depends not only on the level and duration of force but also on the specific mechanical and geometric properties of the cranial system under loading (38–40). Different parts of the skull and brain have distinct biophysical characteristics, and calculating deformation and stress is complex. However, an applied force causes the skull and brain to move, and acceleration, the time required to reach peak acceleration, and the duration of acceleration may be measured at specific locations (36,41). These kinematic parameters do not cause the actual brain damage but are useful for analyzing TBI because they are easy to quantify. Research in TBI using physical models and animal experiments has shown that a force resulting in angular acceleration pro-



duces primarily diffuse brain damage, whereas a force causing exclusively translational acceleration produces only focal brain damage (36). A fall from a countertop or table is often considered to be exclusively translational and therefore assumed incapable of producing serious injury (3,7–9). However, sudden impact deceleration *must* have an angular vector unless the force is applied only through the center of mass (COM), and deformation of the skull during impact *must* be accompanied by a volume change (cavitation) in the subdural "space" tangential to the applied force (41). The angular and deformation factors produce tensile strains on the surface veins and mechanical distortions of the brain during impact and may cause a subdural hematoma without deep white matter injury or even unconsciousness (42–44).

Many authors state that a fall from less than 3 meters (10 feet) is rarely if ever fatal, especially if the distance is less than 1.5 meters (5 feet) (1–6,8,9). The few studies concluding that a short-distance fall may be fatal (22–24,26,27) have been criticized because the fall was not witnessed or was seen only by the caretaker. However, isolated reports of observed fatal falls and biomechanical analysis using experimental animals, adult human volunteers, and models indicate the potential for serious head injury or death from as little as a 0.6-meter (2-foot) fall (48–52). There are limited experimental studies on infants (cadaver skull fracture) (53,54) and none on living subadult nonhuman primates, but the adult data have been extrapolated to youngsters and used to develop the Hybrid II/III and Child Restraint–Air Bag Interaction (CRABI) models (55) and to propose standards for playground equipment (56,63). We simply do not know either kinematic or nonkinematic limits in the pediatric population (57,58).

Each of the falls in this study exceeded established adult kinematic thresholds for traumatic brain injury (41,48–52). Casual analysis of the falls suggests that most were primarily translational. However, deformation and *internal* angular acceleration of the skull and brain *caused by the impact* produce the injury. What happens during the impact, not during the fall, determines the outcome.

Subdural Hemorrhage

A "high strain" impact (short pulse duration and high rate for deceleration onset) typical for a fall is more likely to cause subdural hemorrhage than a "low strain" impact (long pulse duration and low rate for deceleration onset) that is typical of a motor vehicle accident (42,61). The duration of deceleration for a head-impact fall against a nonyielding surface is usually less than 5 milliseconds (39,59–61). Experimentally, impact duration longer than 5 milliseconds will not cause a subdural hematoma unless the level of angular acceleration is above 1.75×10^5 rad/s^2 (61). A body in motion with an angular acceleration of 1.75×10^5 rad/s^2 has a tangential acceleration of 17,500 m/s^2 at 0.1 meters (the distance from the midneck axis of rotation to the midbrain COM in the Duhaime model). A human cannot produce this level of acceleration by impulse ("shake") loading (62).

An injury resulting in a subdural hematoma in an infant may be caused by an accidental fall (43,44,64). A recent report documented the findings in seven children seen in a pediatric hospital emergency room after an accidental fall of 0.6 to 1.5 meters who had subdural hemorrhage, no loss of consciousness, and no symptoms (44). The characteristics of the hemorrhage, especially extension into the posterior interhemispheric fissure, have been used to suggest if not confirm that the injury was nonaccidental (9,62,65–68). The hemorrhage extended into the posterior interhemispheric fissure in 5 of the 10 children in this study (in whom the blood was identifiable on CT or magnetic resonance scans and the scans were available for review) and along the anterior falx or anterior interhemispheric fissure in an additional 2 of the 10.

Lucid Interval

Disruption of the diencephalic and midbrain portions of the reticular activating system (RAS) causes unconsciousness (36,69,70). "Shearing" or "diffuse axonal" injury (DAI) is thought to be the primary biophysical mechanism for immediate traumatic unconsciousness (36,71). Axonal injury has been confirmed at autopsy in persons who had a brief loss of consciousness after a head injury and who later died from other causes, such as coronary artery disease (72). However, if unconsciousness is momentary or brief ("concussion") subsequent deterioration *must* be due to a mechanism other than DAI. Apnea and catecholamine release have been suggested as significant factors in the outcome following head injury (73,74). In addition, the centripetal theory of traumatic unconsciousness states that primary disruption of the RAS will not occur in isolation and that structural brainstem damage from inertial (impulse) or impact (contact) loading *must* be accompanied by evidence for cortical and subcortical damage (36). This theory has been validated by magnetic resonance imaging and CT scans in adults and children (75,76). Only one of the children in this study (case 6) had evidence for any component of DAI. This child had focal hemor-

rhage in the posterior midbrain and pons, thought by the pathologist to be primary, although there was no skull fracture, only "a film" of subdural hemorrhage, no tears in the corpus callosum, and no lacerations of the cerebral white matter (grossly or microscopically).

The usual cause for delayed deterioration in infants and children is cerebral edema, whereas in adults it is an expanding extra-axial hematoma (77). If the mechanism for delayed deterioration (except for an expanding extra-axial mass) is venospasm, cerebral edema may be the only morphologic marker. The "talk and die or deteriorate (TADD)" syndrome is well characterized in adults (78). Two reports in the pediatric literature discuss TADD, documenting 4 fatalities among 105 children who had a lucid interval after head injury and subsequently deteriorated (77,79). Many physicians believe that a lucid interval in an ultimately fatal pediatric head injury is extremely unlikely or does not occur unless there is an epidural hematoma (7,8,11). Twelve children in this study had a lucid interval. A noncaretaker witnessed 9 of these 12 falls. One child had an epidural hematoma.

Retinal Hemorrhage

The majority of published studies conclude that retinal hemorrhage, especially if bilateral and posterior or associated with retinoschisis, is highly suggestive of, if not diagnostic for, nonaccidental injury (9,14–21). Rarely, retinal hemorrhage has been associated with an accidental head injury, but in these cases the bleeding was unilateral (80). It is also stated that traumatic retinal hemorrhage may be the direct mechanical effect of violent shaking (15). However, retinal hemorrhage may be caused experimentally either by ligating the central retinal vein or its tributaries or by suddenly increasing intracranial pressure (81,82); retinoschisis is the result of breakthrough bleeding and venous stasis not "violent shaking" (15,83). Any sudden increase in intracranial pressure may cause retinal hemorrhage (84–87). Deformation of the skull coincident to an impact nonselectively increases intracranial pressure. Venospasm secondary to traumatic brain injury selectively increases venous pressure. Either mechanism may cause retinal hemorrhage irrespective of whether the trauma was accidental or inflicted. Further, retinal and optic nerve sheath hemorrhages associated with a ruptured vascular malformation are due to an increase in venous pressure not extension of blood along extravascular spaces (81–83,88). Dilated eye examination with an indirect ophthalmoscope is thought to be more sensitive for detecting retinal bleeding than routine examination and has been recommended as part of the evaluation of any pediatric patient with head trauma (89). None of the children in this study had a formal retinal evaluation, and only six had funduscopic examination documented in the medical record. Four of the six had bilateral retinal hemorrhage.

Pre-existing Conditions

One of these children (case 16) had a subarachnoid AV malformation that contributed to development of the subdural hematoma, causing his death. One (case 7) had TAR syndrome (90), but his death was thought to be caused by malignant cerebral edema not an expanding extra-axial mass.

Cerebrovascular Thrombosis

Thrombosis or dissection of carotid or vertebral arteries as a cause of delayed deterioration after head or neck injuries is documented in both adults and children (91,92). Case 14 is the first report of a death due to traumatic cerebrovascular thrombosis in an infant or child. Internal carotid artery thrombosis was suggested radiographically in an additional death (case 13) but could not be confirmed at autopsy. However, this child died 6 days after admission to the hospital, and fibrinolysis may have removed any evidence for thrombosis at the time the autopsy was performed.

Limitations

1. Six of the 18 falls were not witnessed or were seen only by the adult caretaker, and it is possible that another person caused the nonobserved injuries.
2. The exact height of the fall could be determined in only 10 cases. The others (7 swing and 1 stationary platform) could have been from as little as 0.6 meters (2 feet) to as much as 2.4 meters (8 feet).
3. A minimum impact velocity sufficient to cause fatal brain injury cannot be inferred from this study. Likewise, the probability that an individual fall will have a fatal outcome cannot be stated because the database depends on voluntary reporting and contractual agreements with selected U.S. state agencies. The NEISS summaries for the study years estimated that there were more than 250 deaths due to head and neck injuries associated with playground equipment, but there are only 114 in the files. Further, this study does not include other nonplayground equipment–related fatal falls, witnessed or not witnessed, in the CPSC database (32).

CONCLUSIONS

1. Every fall is a complex event. There must be a biomechanical analysis for any incident in which the severity of the injury appears to be inconsistent with the history. The question is not "Can an infant or child be seriously injured or killed from a short-distance fall?" but rather "If a child falls (x) meters and strikes his or her head on a nonyielding surface, what will happen?"
2. Retinal hemorrhage may occur whenever intracranial pressure exceeds venous pressure or whenever there is venous obstruction. The characteristic of the bleeding cannot be used to determine the ultimate cause.
3. Axonal damage is unlikely to be the mechanism for lethal injury in a low-velocity impact such as from a fall.
4. Cerebrovascular thrombosis or dissection must be considered in any injury with apparent delayed deterioration, and especially in one with a cerebral infarct or an unusual distribution for cerebral edema.
5. A fall from less than 3 meters (10 feet) in an infant or child may cause fatal head injury and may not cause immediate symptoms. The injury may be associated with bilateral retinal hemorrhage, and an associated subdural hematoma may extend into the interhemispheric fissure. A history by the caretaker that the child may have fallen cannot be dismissed.

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APPENDIX

Newtonian mechanics involving constant acceleration may be used to determine the impact velocity in a gravitational fall. However, constant acceleration formulas cannot be used to calculate the relations among velocity, acceleration, and distance traveled *during* an impact because the deceleration is not uniform (45). This analysis requires awareness of the shape of the deceleration curve, knowledge of the mechanical properties and geometry of the cranial system, and comprehension of the stress and strain characteristics for the specific part of the skull and brain that strikes the ground. A purely translational fall requires that the body is rigid and that the external forces acting on the body pass only through the COM, i.e., there is no rotational component. A 1-meter-tall 3-year-old hanging by her knees from a horizontal ladder with the vertex of her skull 0.5 meters above hard-packed earth approximates this model. If she looses her grip and falls, striking the occipital scalp, her impact velocity is 3.1 m/second. An exclusively angular fall also requires that the body is rigid. In addition, the rotation must be about a fixed axis or a given point internal or external to the body, and the applied moment and the inertial moment must be at the identical point or axis. If this same child has a 0.5-meter COM and has a "matchstick" fall while standing on the ground, again striking her occiput, her angular velocity is 5.42 rad/second and tangential velocity 5.42 m/second at impact. The impact velocity is higher than predicted for an exclusively translational or external-axis angular fall when the applied moment and the inertial moment are at a different fixed point (slip and fall) or when the initial velocity is not zero (walking or running, then trip and fall), and the vectors are additive. However, the head, neck, limbs, and torso do not move uniformly during a fall because relative motion occurs with different velocities and accelerations for each component. Calculation of the impact velocity for an actual fall requires solutions of differential equations for each simultaneous translational and rotational motion (45). Further, inertial or impulse loading (whiplash) may cause head acceleration more than twice that of the midbody input force and may be important in a fall where the initial impact is to the feet, buttock, back, or shoulder, and the final impact is to the head (46,47).

The translational motion of a rigid body at constant gravitational acceleration (9.8 m/s^2) is calculated from:

$$F = ma \qquad v^2 = 2as \qquad v = at$$

where F = the sum of all forces acting on the body (newton), m = mass (kg), a = acceleration (m/s^2), v = velocity (m/s), s = distance (m), and t = time (s).

The angular motion of a rigid body about a fixed axis at a given point of the body under constant gravitational acceleration (9.8 m/s^2) is calculated from:

$$M = I\alpha \qquad \omega = v^1/r \qquad \alpha = a^1/r$$

where M = the applied moment about the COM or about the fixed point where the axis of rotation is located, I = the inertial moment about this same COM or fixed point, α = angular acceleration (rad/s^2), ω = angular velocity (rad/s), r = radius (m), v^t = tangential velocity (m/s), and a^t = tangential acceleration (m/s^2).

The angular velocity ω for a rigid body of length L rotating about a fixed point is calculated from:

$$\tfrac{1}{2}I_0\omega^2 = maL/2 \qquad I_0 = (1/3)\ mL^2$$

where I_0 = the initial inertial moment, ω = angular velocity (rad/s), m = mass (kg), a = gravitational acceleration (9.8 m/s^2), and L = length.

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Fatal Falls in Childhood

How Far Must Children Fall to Sustain Fatal Head Injury? Report of Cases and Review of the Literature

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The question of whether fatal head injuries may occur from short-distance falls is one that continues to cause controversy. The records of the Sacramento County Coroner's Office from 1983 to 1991 were reviewed for cases of fatal head injury in children aged ≤5 years, where a history of a fall was initially given. During this 9-year period, three cases of witnessed falls from heights of >10 ft (3 m) were found. At autopsy, all children had multiple complex calvarial skull fractures, basal fractures, or both; subdural and subarachnoid hemorrhage was found in all cases, and two showed severe cerebral contusion. None had retinal hemorrhage or axonal injury. These are compared with 19 fatalities initially alleged to have occurred from short falls of ≤5–6 ft (1.5–1.8 m). As others have found, most of these "minor fall" fatalities occurred under circumstances where there were no unrelated witnesses to corroborate the initial history. Autopsy findings in these cases tended to be of unexpected severity for the initially proposed mechanism of injury, and a number of cases showed evidence of accelerative injury (retinal hemorrhage and/or diffuse axonal injury) where no such mechanism was accounted for by initial history. After sufficient investigation, most of these cases (74%) have ultimately been proven to represent inflicted trauma. A thorough literature review on the subject identifies two major viewpoints. One is that short falls have a significant potential for fatality. The other, more widely espoused view is that short falls rarely, if ever, cause serious injury or death. These two views, and the data upon which they rest, are compared and contrasted.

Key Words: Child abuse—Head injury—Falls from heights.

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At times, medical examiners may be confronted with cases of severe head injury that present with a history of a minor accidental fall. Histories of falls down stairs, against pieces of furniture, in bathtubs, or from beds or couches are common examples. The suspicion of child abuse arises when the injuries do not correspond with the history. This necessitates critical comparison of the severity of the injury and the adequacy of the explanation. Additionally, one may be confronted in the courtroom with medical literature or expert opinion that attributes "severe head injury" to mechanisms typically held to be minor.

To address this question more fully, childhood fatalities from falls in Sacramento County, California, between 1983 and 1991 are reviewed. The related literature is also reviewed, exposing two apparently contradictory viewpoints. The first, more prevalent view is that major trauma does not result from minor falls. The other "minority" viewpoint is that, on occasion, fatal injury to children results from short falls. These two views, and their foundations, are explored and compared.

CASE PRESENTATION

Major-Fall Cases

In the period from January 1983 through December 1991, three cases of accidental injury in children under the age of 5 years, involving falls from heights of between 10 and 25 ft (3 and 7.5 m), were identified in the records of the Sacramento County Coroner's Office.

Case 1

Y.K., a 14-month-old girl, fell from a second-story window onto a concrete sidewalk after a screen came loose. The fall was witnessed by an unrelated bystander. The exact distance of the fall was not

measured, but is estimated at ~15 ft (4.5 m). The child was obtunded at the scene and had right otorrhea. The pupils were fixed but unequal. Aside from an area of right parietal swelling, no external trauma was seen.

The child was stabilized at a local hospital before transfer to the trauma center within an hour of injury. On admission, multiple skull fractures and an acute subdural hematoma were found. After a left parietal craniotomy, the child developed malignant brain swelling. She died ~1 week after injury.

An autopsy performed at the Sacramento County Coroner's morgue found no external injuries of note. Internal examination revealed a large left parietal craniotomy defect, through which softened hemorrhagic brain tissue had herniated. A left frontal skull fracture extended into the orbital plate. Residual subdural hematoma and focal subarachnoid hemorrhage were present.

Case 2

R.D., a 4-year-old girl, had allegedly climbed onto a heating/air-conditioning unit in a motel room and then fallen from the third-story room when a loose screen, covering the window above the unit, gave way. She struck a sheet-metal canopy over a first-story window before falling further to a concrete walkway. The total distance of the fall was estimated at 25–30 ft (7.5–9 m). The fall was not directly witnessed, but the desk clerk recalled hearing a "thump" at the time of the incident. Police investigation revealed several aspects of the scene that confirmed the story and uncovered no contradictory evidence.

The child was rushed by ambulance to the nearest hospital and survived for ~8 h after the injury, following laparotomy with repair of splenic lacerations. She ultimately died from complications of severe head trauma. External findings at autopsy included marked swelling of the head, bilateral periorbital ecchymoses, and facial contusions and abrasions. Superficial abrasions were found on both upper extremities. Internally there was severe subgaleal hemorrhage and multiple complex depressed skull fractures of the left frontotemporal region. Linear basal fractures were present, as were bilateral linear occipital fractures.

The brain showed extensive left temporal and parietal lacerations, deep contusions, and bilateral cerebral and cerebellar subarachnoid hemorrhages. Bilateral pulmonary contusions were also found.

Case 3

J.L., a 22-month-old boy, was in the care of his mother, who was working as a motel maid. The mother was cleaning a third-floor room when the child escaped her attention, crawled under a metal railing, and fell ~20 ft (6 m) onto a concrete surface. He lost consciousness after the impact, but awoke briefly and cried before paramedics arrived.

The child was unresponsive when emergency medical personnel arrived and was hospitalized within 1 h of the injury, with a Glasgow coma score of 6. Computed tomographic scan showed a right frontal acute subdural hematoma, underlying cerebral cortical contusion, and cerebral swelling with a right-to-left shift of ~1 cm. Multiple cranial fractures were present.

He was rushed to neurosurgery for evacuation of the subdural hematoma and partial right frontal lobectomy, but died 32 h after injury, with severe cerebral edema, diabetes insipidus, and disseminated intravascular coagulopathy. The body was transported to the Sacramento County Coroner's Office for postmortem examination.

The head had a 5-cm area of contusion above the right eye. Bilateral periorbital ecchymoses were present, with greater severity on the right. No scalp abrasions or lacerations were seen. Scalp reflection revealed extensive frontoparietal subgaleal hemorrhage. The frontal skull showed multiple basal and calvarial fractures. The midcoronal suture and anterior sagittal suture were diastatic. Basal fracture extended into the right lesser and greater sphenoid wings.

Clot and softened brain matter replaced the right frontal pole; an adjacent frontal subdural hematoma was present. Bilateral uncal and tonsillar herniation and mild right cingulate herniation were found. Microscopic examination revealed acute anoxic damage, but evidence of diffuse axonal injury was absent. Other autopsy evidence of trauma included dislocation of the left wrist and hip, and scattered bruises and abrasions of the back and lower extremities.

Minor-Fall Cases

During the 9 years covered by this report, the records of the Coroner's Office contain 19 fatalities with an initial history of a short fall, usually between 1 and 5 ft (0.3–1.5 m). The reason for a higher number of short-fall fatalities during this period is unclear. It could simply be a reflection of a much higher prevalence of minor falls as compared with higher-distance falls in the community as a whole. However, close examination of individual cases suggests other explanations.

The minor-fall cases break down into three groups: (a) cases with clear evidence of homicidal assault; (b) cases with inconsistencies between history and injury, but where gaps in the investigation or pau-

city of autopsy findings prevent one from reaching a conclusion; and (c) cases where the history and injuries appear consistent.

The first group includes 14 cases (74%) that investigation and injury analysis have shown to be homicides involving either blunt impact (eight cases) or a combination of shaking and blunt impact (six cases). These cases show vast inconsistencies between initial history and spectrum of injury. In nearly all cases, the histories were ultimately amended, either with additional inconsistencies or with a confession of what actually happened.

The second group includes three cases (15.5%). Two have highly inconsistent findings, such as unexplained retinal hemorrhages, but for various reasons cannot be classified as homicides. A third case shows clear evidence of battering, but the cause of death is uncertain and the manner of death remains undetermined.

The third group includes two cases (10.5%) where the histories appear genuine. In one case, a 21-month-old child fell from a top bunk to a carpeted floor. This history suggests a height of fall of between 5 and 6 ft (1.5–1.8 m)—certainly the farthest of the short-fall group. As a result, the child sustained an acute subdural hemorrhage accompanied by severe brain swelling. There was no skull fracture.

In the second case, a 17-month-old child fell backwards from a rocking chair and sustained acute left subdural hemorrhage, subarachnoid hemorrhage, and a left parietal cerebral contusion, without scalp contusion or skull fracture. The fall was perhaps 2–3 ft (0.6–0.9 m), but there may have been an initial angular velocity from movement of the rocking chair. Recalling the formula for free-fall velocity, one of the assumptions is zero initial velocity. If there is, in fact, a significant initial velocity, then the final speed at impact is significantly higher than distance alone might suggest.

CASE ANALYSIS

Presence of Skull Fracture (Fig. 1)

All of the major-fall cases included skull fracture. Of the minor-fall cases, six (31.5%) showed skull fracture. These included three depressed fractures, two complex (one of which was also depressed), one linear but gaping, and one narrow linear. Fewer than one third of the "minor fall" deaths involved skull fracture.

Landing Surface (Fig. 2)

All of the major-fall deaths resulted from falls onto concrete surfaces. Only three (16%) of the 19 minor-fall cases had initial histories of landing on a hard surface such as concrete; two of these cases included skull fractures. In one case (5%), the child reportedly fell onto a semihard surface and had a complex depressed fracture. Fifteen cases (79%) involved a carpeted surface; only one such case included skull fracture.

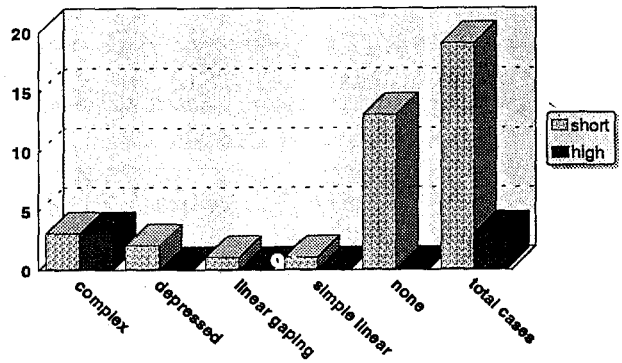

FIG. 1. Skull fractures in fatal falls to children <6 years of age, SAC county, 1983–91.

Unexpected Anatomic Findings (Fig. 3)

The most commonly encountered unexpected finding in the minor-fall group involved unexplained bruises of the trunk and/or extremities. These were found in 13 (68%) of 19 cases. One major-fall case (33%) had significant extremity injury, one (33%) had no extremity injury but had major internal abdominal trauma, and one (33%) had only minimal injury to one hand.

The next most common unexpected finding was retinal hemorrhage and/or diffuse axonal brain injury. None of the major-fall cases had evidence of retinal hemorrhage or diffuse axonal injury. Six (32%) of the minor-fall cases had autopsy evidence of either retinal hemorrhages, diffuse axonal injury, or both, although none of the presenting histories suggested acceleration injury.

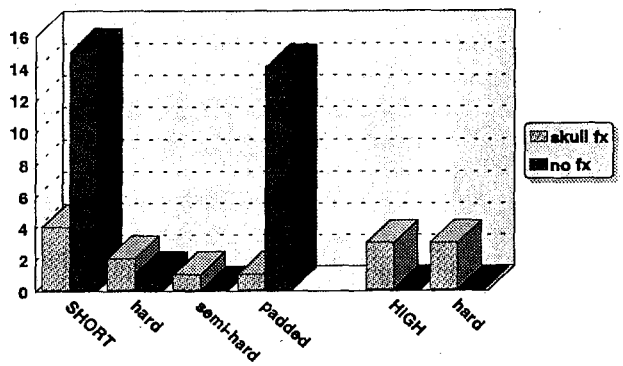

FIG. 2. Landing surfaces in fatal falls to children <6 years of age, SAC county, 1983–91.

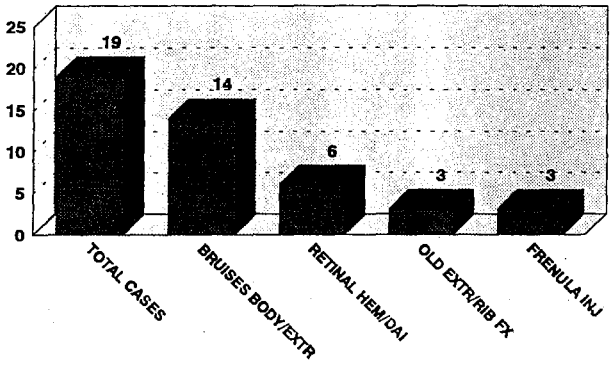

FIG. 3. Unexpected findings in alleged short falls, SAC county, 1983–91.

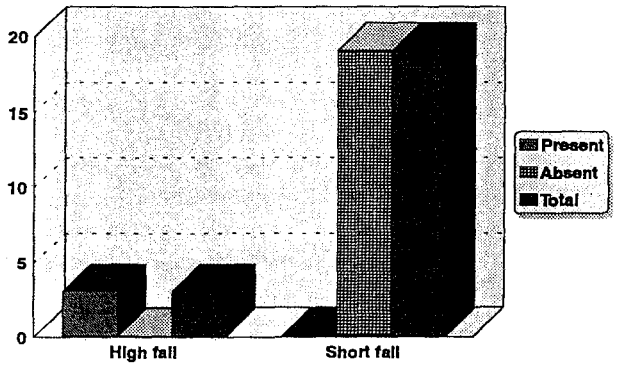

FIG. 4. Fatal falls in children <6 years of age, SAC county, 1983–91, observed by unrelated witnesses.

Torn or bruised frenula were absent in the major-fall cases and were found in only three (16%) of the minor-fall group. Old rib or extremity fractures were also found in three cases (16%).

Presence of Witnesses (Fig. 4)

Two of the three major-fall cases were directly witnessed by unrelated parties. One was indirectly witnessed. None of the 19 minor-fall cases were witnessed by an unrelated party.

LITERATURE REVIEW

A fairly large body of literature presents the concept that children do not typically sustain major injury from minor falls. This concept appears in studies based on series of only short falls without injury, or on series of falls from various heights but with serious injury only at substantial height. There is seeming contradiction in a smaller body of literature that presents cases of serious injury and death in children falling short distances.

The "Major Injury–Major Fall" Literature

This body of literature can be roughly separated into three major subgroups: (a) articles presenting the concept of minor injury from minor falls, (b) articles presenting the concept of major injuries from major falls, and (c) articles that contrast cases of major injury from alleged minor falls with cases of major injury from major falls.

The first subgroup presents the view, on a clinical basis, that short falls rarely, if ever, result in serious intracranial pathology. Helfer et al. (1), though sometimes criticized, represent an oft-quoted vanguard of this position. They present a group of 161 children with histories given by parents, and a second group of 85 children whose falls were witnessed in hospitals. Of the group reported by parents, only two had skull fractures; of the hospital-based cases, there was only one skull fracture. None of the fractures resulted in neurologic sequelae.

Nitimyongskul and Anderson (2) also conclude that minor falls lead to relatively minor injuries. In a series of 76 children who fell from low heights, there was only one questionable occipital skull fracture. Most injuries were cutaneous or soft tissue bruises or lacerations. No fatalities were encountered.

The second subgroup presents fatalities resulting from major falls. Barlow et al. (3) reviewed a series of 61 children aged ≥15 presenting with injuries from falls and found no fatalities from heights of three stories or less. The 50% mortality rate occurred between the fifth and sixth floors; most fatalities were from brain trauma.

Cummins and Potter (4) studied falls of ≥10 ft (3 m) by children and adults and found three fatalities—all adults falling onto concrete. Serious but nonfatal injuries, including four depressed compound skull fractures, were also found among children who had fallen onto hard surfaces from similar heights. Skull fractures in the group as a whole were more frequent in children of ≤5 years of age, but brain trauma was more frequent in older patients. Severity of brain injury was more significant in predicting outcome than was the presence or absence of skull fracture.

The third subgroup compares mortality and injury rates from alleged minor falls and from witnessed major falls. This group is represented by two recent studies. Chadwick et al. (5) address the issue from the standpoint of mortality rates. In their series, there was one death in 118 children who fell from between 10 and 45 ft (3–13.5 m; mortality rate, 0.85%), whereas seven of 100 children died whose caretakers gave a history of a fall of ≤4 ft (1.2 m; mortality rate, 7%). This would imply, if

most of the short-fall histories are accepted, that falls of <4 ft are not only dangerous, but are more than eight times as dangerous as falls from 10 to 45 ft. The odd results are attributed to falsification of history in the short-fall group, a conclusion that gains support from the high frequency of retinal hemorrhage in this group. The overall findings by Chadwick et al. generally correspond with the present author's analysis of Sacramento County cases.

Williams (6) echoes Chadwick et al. in comparing injury rates in falls of approximately known heights, and witnessed by unrelated parties, with injury rates in allegedly short falls (<5 ft or 1.5 m) where no independent verification was obtainable. The findings are quite similar to those reported by Chadwick et al.

The "Major Injury–Short Fall" Literature

Reichelderfer et al. (7), in discussing playground injuries, state that serious head injury may occur in falls where gravity (*g*) forces exceed 50 *g*. The authors list tabular data which indicate that a drop of 0.25 ft—a "fall" of 3 in. (7.5 cm)—onto a concrete surface may generate a force of 150–200 *g*. A drop of 1 ft (0.3 m) onto concrete produced a 475–525 *g* impact.

Weber (8) dropped infant cadavers and found that, in free-fall situations, skull fractures may occur in young (<1 year) infants at changing-table and countertop heights, especially with a hard landing surface. Most injuries were linear parietal fractures; complex fractures were rare.

Root (9) implies that, if skull fractures may occur with falls from relatively low heights, then *perhaps* serious head injury does not require a "serious" event or mechanism. He presents the image of someone running headlong into a brick wall, which would most certainly result in discomfort and could produce serious injury. Unfortunately, since no hard data result, it is difficult to compare such a mental "experiment" with the clinical and laboratory studies on children who fall or infant cadavers that are dropped.

A recent study of autopsied children by Hall and colleagues (10) reveals a potential for mortality from minor falls of ≤3 ft (0.9 m). Over a 4-year period, 18 children died after allegedly falling <3 ft. Two of the cases were witnessed by medical personnel. The remaining 16 were not independently witnessed, but the histories were never disproved.

Whereas the medical examiner's office involved in the study autopsied eight children who died after falls from five stories or higher, 18 children were autopsied who died after allegedly falling ≤3 ft. This could be simply a function of frequency of major versus minor falls in the community. There is also the possibility of falsified history, as our analysis of Sacramento cases indicated.

While the study by Hall and colleagues does not contain our type of case analysis, the lack of concurrent injuries to other parts of the body in their series contrasts with the high incidence of such unexpected trauma in our series. While not settling the issue, this may indicate a higher likelihood that his short-fall histories were true than in our cases.

DISCUSSION

Superficially, there is clear conflict in the literature on the subject of fatalities from childhood falls. On closer examination, however, the conflict is less than it initially seems. Much of the apparent contradiction arises not so much from the data contained in the literature as from the use of differing approaches to the data and the inherent strengths and weaknesses of those approaches. Table 1 summarizes these differences.

While there is disagreement, there is also much consensus in the literature on the subject. The major areas of consensus form a core of concepts that contain useful guidelines for the evaluation of one's individual cases.

One point of consensus is that the presence of skull fracture *alone* is not evidence of nonaccidental trauma, particularly when only a simple linear parietal fracture is present. Several clinically based studies (11,12) illustate the not uncommon occurrence of linear skull fractures from minor falls in children. While the greatest incidence involves infants of <1 year of age, a lower incidence is also seen in older children. Such fractures are usually uncomplicated and result in no neurologic sequelae. Other types of fracture, however, are more problematic. Complex fracture patterns and large depressed fractures, especially when combined with severe intracranial pathology, are generally inconsistent with minor falls (13–15). Unless there is history of a major fall or road accident, the possibility of abuse must be investigated in these cases. Small depressed fractures, however, have been seen in accidental short falls against edged surfaces (6).

Another important point over which there is no contradiction is the lack of major nonhead trauma in short falls. When falls of significant magnitude occur, injuries to parts of the body other than the head may be present and may be of major significance (3,4). However, such collateral trauma is not expected from a short fall (10). If severe trauma to

TABLE 1. *Comparison of literature on childhood falls*

Helfer et al. (1)	
Viewpoint	Clinical pediatrics; need for routine x-rays
Strengths	Size of series; use of clinical data
Weaknesses	Fall mechanics limited to landing surface and relative height; partial reliance on questionnaires may add reporting bias
Nitimyongskul and Anderson (2)	
Viewpoint	Orthopedic surgery (clinical study)
Strengths	Statistical analysis of direct clinical observations
Weaknesses	Little information on mechanics of falls; primary focus on skeletal trauma, not head trauma
Barlow et al. (3)	
Viewpoint	Pediatric surgery/injury prevention (clinical study)
Strengths	Includes some autopsy data; includes variety of distances
Weaknesses	Addresses only free-falls; small series
Cummins and Potter (4)	
Viewpoint	Neurosurgery/outcome oriented (clinical study)
Strengths	Cases well analyzed
Weaknesses	Small database
Chadwick et al. (5)	
Viewpoint	Pediatrics/child abuse (clinical study)
Strengths	Reasonably sized database; more detailed case analysis
Weaknesses	Overt orientation to child abuse diagnosis may lead to exclusion of some valid short-fall cases
Williams (6)	
Viewpoint	Pediatric pathology (clinical study)
Strengths	Reasonably sized database; good statistical analysis
Weaknesses	Many histories uncorroborated
Reichelderfer et al. (7)	
Viewpoint	Pediatrics/injury prevention (laboratory data presented)
Strengths	Laboratory-based "hard" data; tabular presentation clear
Weaknesses	Experimental technique not discussed leaves question of comparability to real-world falls
Weber (8)	
Viewpoint	Forensic science (laboratory study)
Strengths	Lab study with some control; varying heights and contact surfaces; used actual infants (cadavers)
Weaknesses	Use of cadavers prevents full injury evaluation; comparability with older children questionable
Root (9)	
Viewpoint	Forensic pathlogy (discussion article only)
Strengths	Appeal to conceptual framework
Weaknesses	No data; limited to discussion and opinion
Hall et al. (10)	
Viewpoint	Pediatric surgery/prevention (clinical study)
Strengths	Includes clinical and autopsy data
Weaknesses	Case breakdown unclear; autopsy findings abbreviated; evaluation of some histories may be too noncritical

Note: All of the articles except those by Weber (8) and Root (9) relied largely on hospitalized cases. This selects for the most serious cases and may overemphasize the commonality of injuries from falls of any height.

head *and* body is found, a presenting history of a minor fall is suspect.

One clear area of remaining uncertainty concerns the infant skull itself. The pliability and thinness are considered by some (4) to provide greater protection to the brain by virtue of a greater degree of shock absorption. Others (10) feel that the child's skull provides less protection. The true role of the child's skull in either lessening or exacerbating the magnitude of internal injury is a question that deserves additional study.

CONCLUSIONS

In our series, all fatalities from bona fide short falls resulted from intracranial hemorrhage without skull fracture or significant external head trauma. Hall et al. (10) also emphasize the possibility of potentially

lethal intracranial hemorrhage from seemingly "minor" falls. Cases of fatal malignant cerebral edema may also occur without signs of major mechanical trauma to the scalp or skull (16–20).

In contrast, cases with extensive skull fracture and brain contusion or laceration, or with major head *and* body injury, render a short-fall history highly questionable. Findings of retinal hemorrhage, interhemispheric subdural hemorrhage, and diffuse axonal injury indicate accelerative injury. Without a history of a major fall or high-speed motor vehicle accident, these findings typically point to shaking or shaken/impact syndrome.

The height from which children must fall to sustain fatal head trauma is a question that lacks a single, easy answer. One has to consider the possible mechanics of the fall, the age and condition of the child, and the shape and consistency of the contact surface. Correlation of fall mechanics and injury pattern should be considered.

The bulk of the studies on head injuries in children, regardless of viewpoint, are based on hospitalized cases. This selects for the most serious cases and is likely to give the impression that fatalities from short falls are more prevalent than they actually are. The conclusion that appears best at this time, with our current state of information, is that, while children on occasion suffer fatal injury from short falls, such events are an extreme rarity. Major injuries nearly always result from major impacts and serious falls. □

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TECHNICAL NOTE

Finite element analysis of impact and shaking inflicted to a child

Sébastien Roth · Jean-Sébastien Raul · Bertrand Ludes · Rémy Willinger

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Abstract This study compares a vigorous shaking and an inflicted impact, defined as the terminal portion of a vigorous shaking, using a finite element model of a 6-month-old child head. Whereas the calculated values in terms of shearing stress and brain pressure remain different and corroborate the previous studies based on angular and linear velocity and acceleration, the calculated relative brain and skull motions that can be considered at the origin of a subdural haematoma show similar results for the two simulated events. Finite element methods appear as an emerging tool in the study of the biomechanics of head injuries in children.

Keywords Child abuse · Finite element head model · Forensic medicine · Shaken baby syndrome

Introduction

The "shaken baby syndrome" is the leading cause of death or serious neurological injury resulting from child abuse. Injuries that characterize the shaken baby syndrome are subdural haemorrhage and retinal haemorrhage. For the past 20 years, child head injury biomechanics have been studied through the evaluation of rotational and linear velocities and acceleration. When comparing a shaking event with an impact, the results have shown significant differences. In addition to these global parameters, other biomechanical parameters have to be taken into account for a complete evaluation of the injury mechanisms. Brain injury can be caused either by vigorous shaking, by a shaking followed by an impact on a soft or hard surface or by direct blows to the head. Vigorous shaking will initiate for a certain frequency a relative motion of the brain and skull, tearing the bridging veins that extend from the cortex to the dural venous sinus and leading to a subdural haematoma. Vigorous shaking can also cause neck injuries, and the younger is the child, the greater is the effect of force exerted during shaking. Injuries to the neck will be either articular or ligamental injuries of the cervical spine as well as injuries to the spinal cord. When a significant amount of force is applied on the neck, apnoea and breathing problems will be the major clinical signs. In the other case, seizure or less specific signs, such as irritability, vomiting or lethargy, will dominate the clinical presentation. Since the article by Guthkelch in 1971, who postulated that a subdural haematoma as a feature of the "battered child syndrome" could be caused by shaking, biomechanical studies of falls, shakes or inflicted impacts to children have been widely described and studied in the literature especially for the past 20 years [1–5]. Studies using dummies or analytical models have compared child head injuries as a function of angular and linear acceleration, but this scientific approach is insufficient when the local behaviour of biological tissues must be taken into account. To evaluate the consequence of head injuries with a maximum biofidelity, finite element models are by far the most reliable tools. Finite element models have been recently used to investigate adult head injuries in forensic cases [6–8]. Whereas more than ten

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different three-dimensional finite element models of the adult head exist in the literature, very few finite element models of the child head have been developed. This is mainly due to the difficulties in validating such models against experimental data. However, child head material properties have been studied and are available in the literature [9–12]. In the present paper, we developed a 6-month-old child head model and simulated a vigorous shake and an inflicted impact, defined as the terminal portion of a vigorous shake, to study their consequences in terms of intracerebral pressure, shearing stress and relative motion of brain and skull. The aim of this paper is not to establish new pediatric injury criteria but to use a new finite element model to compare intracerebral mechanical response under two different loading conditions.

Materials and methods

The head of a child cannot be considered as a "small-scale model" of an adult. Therefore, the present study proposes a finite element model using computed tomography (CT) scan slices of a representative 6-month-old child who underwent a radiological examination after minor head trauma. Meshing was performed using the Hypermesh code (Altair Hyperworks 7.0 software, Michigan, USA). The main anatomical features modelled were the skull, tentorium, fontanels, falx, cerebro-spinal fluid (CSF), scalp, cerebrum and cerebellum. The finite element mesh is continuous. Falx, tentorium, fontanels, sutures and skull were simulated with one layer of shell elements; brain, CSF and scalp were modelled with brick elements. Bridging veins were modelled with springs. Globally, the model consists of 69,324 brick elements and 9,187 shell elements.

Material properties were taken from the literature, where several studies have been carried out on cranial sutures, cranial bone and brain [9–11, 13, 14]. In 1998, Thibault and Margulies [12] reported the mechanical properties of a porcine brain to find age-dependence in comparing viscoelastic properties of 1-year-old pigs (similar to a 4-year-old child) to those of 2- to 3-day-old pigs (similar to a 1-month-old human). They established a viscoelastic law that is used for paediatric brain models. Mechanical properties of the scalp, the CSF and the membranes (tentorium and falx) are issued from the adult finite element model and have already been published [7]. For the skull, fontanels and sutures, the material properties were taken from Margulies and Thibault [10]. The face is considered as a part of the skull and has the same constitutive law. Figure 1 shows the three-dimensional finite element model, and Table 1 summarizes the material properties.

Simulation of both inflicted impact and vigorous shaking is based on the work reported by Prange et al. [3] who recorded the angular velocity of the head of a dummy

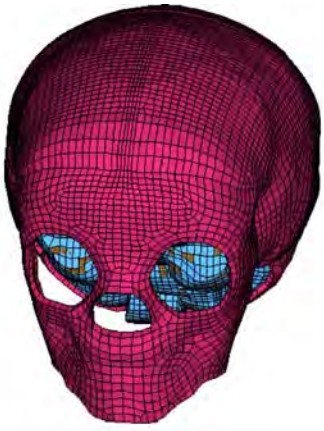

Fig. 1 Finite element model of a 6-month-old infant head

Tissue	Element type	Constitutive law		Tissue	Element type	Constitutive law	
Brain	Brick	Viscoelastic		Subarachnoid space	Brick	Elastic	
Membranes (Tentorium and falx)	Shell	Elastic		Facial bone	Shell	Elastic	
Skull	Shell	Elastic		Scalp	Brick	Elastic	
Fontanel and sutures	Shell	Elastic					

Table 1 Material properties of the 6-month child head model

	Young modulus (MPa)	Poisson's ratio	Density (kg/m^3)	References
Membranes	31.5	0.45	1,140	Zhou et al. [24]
CSF	0.012	0.49	1,040	Willinger and Taleb [25]
Scalp	16.7	0.42	1,200	Zhou et al. [24]
Skull	2,500	0.22	2,150	Margulies and Thibault [10]
Sutures and fontanels	1,500	0.22	2,150	
Brain	$G(t) = G_\infty + (G_0 - G_\infty)e^{-\beta t}$, where $G_0 = 5.99 \times 10^{-3}$MPa, $G_\infty = 2.32.10^{-3}$MPa and $\beta = 0.09248$ s^{-1}, with a bulk modulus of K=2,110 MPa			Thibault and Margulies [12]

submitted to these loadings. They performed several tests with perpetrators of different height and strength. Their experiments consisted of a shaking event followed by an inflicted impact on a concrete surface.

The simulation of the vigorous shake consists of one cycle extracted from Prange et al.'s experiments. The input of the vigorous shake lasts one cycle of the angular velocity curve as shown in Fig. 2 and is applied at the centre of rotation of the system, which is assumed to be located at the C5–C6 junction according to Swischuk [15].

For the simulation of inflicted impact, the input consisted of a linear velocity of 3 m/s against a rigid wall.

Intracerebral pressure and shearing in terms of Von Mises stress were computed throughout the brain. The CSF layer allows a relative motion of the brain and skull under dynamic loading. In the present study, relative displacement in the sagittal plane was computed to evaluate elongation of the bridging vein.

The strain of the bridging veins is a function of the initial and final lengths as illustrated in the following equation:

$$\varepsilon = \frac{l - l_0}{l_0} \quad (1)$$

where ε represents the strain and l and l_0 are the length and initial length, respectively.

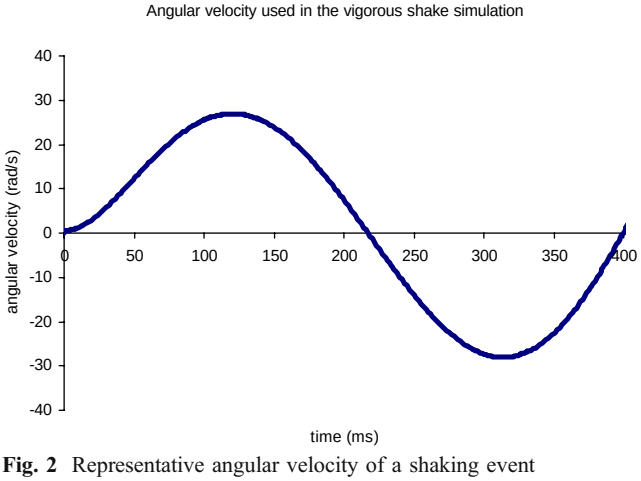

Fig. 2 Representative angular velocity of a shaking event

Results

For the inflicted impact simulation, maximum Von Mises stress was located in the occipital region and reached 14 kPa for an initial linear velocity of 3 m/s. In the vertex area, the stress reached 10 kPa.

Like Von Mises stress, maximum pressure was located in the impact area, and the computed value was 80 kPa. The minimum pressure was located in the frontal area for the contre-coup and reached −120 kPa.

Finally, the computed maximum bridging vein strain occurred at the time of 6 ms after the impact and reached 100%.

For the shaking simulation, the maximum calculated Von Mises stress was 3.2 kPa and was located in the vertex area. For the pressure, a maximum of 22 kPa located in the frontal area was observed.

Moreover, the maximum strain of the bridging veins reached 90% and occurred at the time of 140 ms after the beginning of the shaking motion.

Comparison of intracranial mechanical response under both loading conditions (shaking and impact) clearly showed that brain pressure and shearing stresses are significantly lower for shaking than for an impact as illustrated in Figs. 3 and 4. However, for brain–skull relative motion, the conclusions are different. Results illustrated in Fig. 5 show that the maximum strain value of the bridging veins is equivalent for shaking and for an impact. These results are in accordance with values found by Lee and Haut [16]. The rupture of a bridging vein can occur in both a shaking event and an inflicted impact, leading to the formation of a subdural haematoma.

Discussion

For the last two decades, the biomechanical evaluation of child head injury has been based on the comparison of angular and linear acceleration and velocity. Duhaime et al. [2] and Prange et al. [3] found a peak of angular and linear acceleration and velocity very different for inflicted impacts and shaking events using a dummy model. This led to the

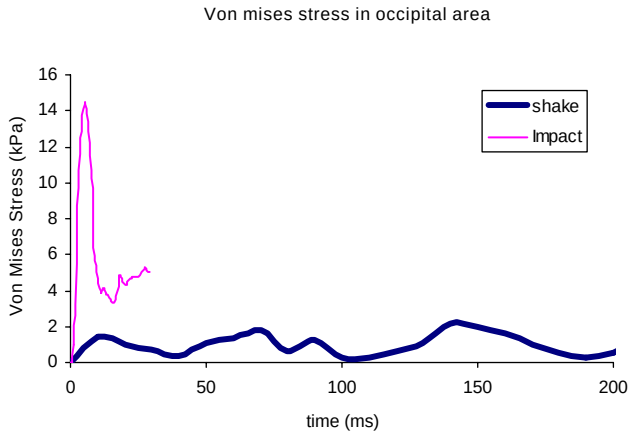

Fig. 3 Von Mises stress in the occipital area for the inflicted impact and the shaking event

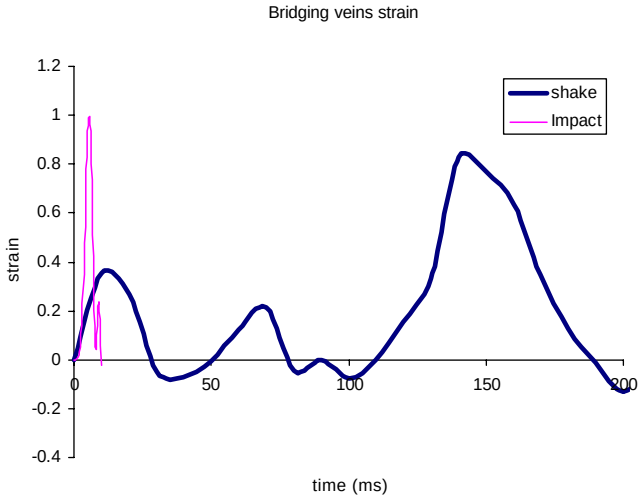

Fig. 5 Bridging veins strain evaluation as a function of time for a shaking event and an inflicted impact

idea that what was supposed to be a shaking event was in fact a shaken–impact event, and what was called the *whiplash shaken infant syndrome* by Caffey [17] would be an inappropriate description and should better be called *shaking impact syndrome*. This idea has recently been reinforced by Bandak [5] who tried to demonstrate that the force involved for creating intracranial injuries by shaking a baby exceeds the limit for failure of the spine.

The use of β-amyloid precursor protein, the most reliable marker of axonal damage, has recently helped to distinguish between hypoxic and traumatic axonal injury [18, 19]. These works have demonstrated that in cases of violent shaking, the initial brain injury is caused by hypoxia. Hypoxia is caused by respiratory difficulties such as apnoea, which is a usual presentation in cases of shaking

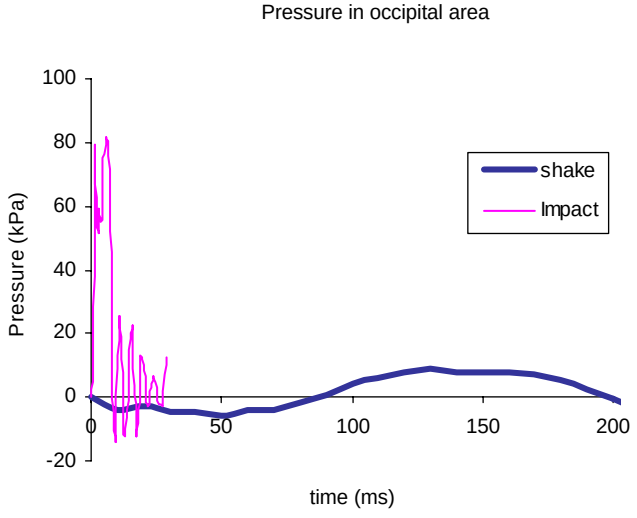

Fig. 4 Pressure in the occipital area for the inflicted impact and the shaking event

events. It is obvious that the primary lesion in cases of shaking leading to apnoea is a cervical spinal cord injury because the infant spine is immature, giving no protection to a whiplash event. It is of major importance to distinguish between two clinical presentation entities after a shaking event. The first is encountered when the child is very young and/or the perpetrator is very strong. In this case, the spinal injury is the *prime cause* leading to apnoea and hypoxic axonal injuries and accompanied by a subdural haematoma. The second is dominated by the consequence of the subdural bleeding leading to seizure and possible raised intracranial pressure, ischaemia and hypoxic axonal injury. In these two clinical presentations, a subdural haematoma and retinal haemorrhage can be encountered.

Finite element models allow the dynamic response of the brain and skull to different forces to be understood. The finite element method is used in biomechanics especially of the adult head for which different finite element models have been developed for 30 years. Recently, research interests in child head modelling appeared, but very few models have been proposed [13, 20–22]. De Santis Klinich and Hulbert [22] developed a finite element model of a 6-month-old child to simulate different accidental load cases (real-world cases) and to compute stress distribution. Their finite element model is based on a 27-week-old child geometry. Scalp, skull and sutures were modelled as shell elements, whereas brain, dura and CSF were modelled as solid elements. The face was modelled as a rigid body. They investigated the tolerance to skull fracture, studying the role of cranial suture. They found a negligible effect of sutures and correlated the response of the model in terms of deformation and stress distribution with the severity of the real-world cases. Prange and Kiralyfalvi [23] studied the

influence of mechanical properties, geometry and loading of the brain by creating a finite element model of a mid-coronal slice of the brain and skull. The geometry was obtained from a 2-week-old infant, and the adult model was obtained by scaling up the child's geometry. The aim of the study was to determine the influence of brain size and mechanical properties on pediatric inertial injuries, running several simulations to see the influence of those parameters (size and material laws).

We developed a realistic finite element model using CT scan slices and used to simulate a vigorous shaking and an impact. The difference in the values obtained in terms of Von Mises stress and pressure is obvious and higher for an inflicted impact than for a shaking event. In adult head injury cases, Von Mises stress and pressure have been correlated to severe traumatic neurological injuries, concussion and loss of consciousness [6–8]. This is usually the case when a child suffers an impact of any kind, whether it is an impact against a wall or a violent blow. However, vigorously shaken babies often present with no concussion but with subdural haematoma and the consequence of hypoxic neurological injuries. The differences we observed in our study between vigorous shaking and inflicted impact therefore appear to be relevant. In fact, what is of major importance when comparing the effect of shaking to an impact is to measure the relative motion of the brain and skull, which gives information on the chance of creating a subdural haematoma by the rupture of a bridging vein. In the two simulations, the brain moves in a sagittal plane. When compared to one another, shaking and impact cause similar relative displacement of the brain and skull but in a different time scale. A vigorous shaking can therefore have the same consequence as an impact in term of subdural bleeding. For an impact, the subdural bleeding will sometimes be associated with cerebral contusion or a skull fracture, and the shaking will lead to subdural bleeding and sometimes spinal injuries. In the two cases, the subdural bleeding will occur; the difference stands in the associated injuries.

Conclusion

In the past years, biomechanical studies have focused on the comparison of velocity and acceleration between different scenarios of head injuries to children. This extreme simplification has led to wrong ideas concerning the consequences of shaking a baby. Based on a detailed finite element model of the 6-month-old child head, it has been demonstrated that vigorous shaking can have the same consequence as an impact in terms of subdural bleeding. Finite element methods can be used as a complementary tool in understanding and analysing cases of child abuse.

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CALCULATING DISTANCE OF FALL FOR 3 METER/SECOND IMPACT

For an object that is initially not moving:[1]

V = AT or T = V/A

D = VT/2

V: velocity
A: acceleration
T: time
D: distance

Here, we want to know the distance an object has to fall to reach a speed of 3 meters/second. The acceleration due to gravity is 9.8 meters/second^2.[2]

Use the first equation to calculate the time it takes a falling object to reach a speed of 3 meters/second:

T = 3/9.8 = 0.306 seconds

Now use the second equation to calculate the distance a falling object moves in that time to reach that speed:

D = 3 x 0.306/2 = 0.459 meters = 18.1 inches

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[1] The equations can be found at http://www.engineeringtoolbox.com/acceleration-velocity-d_1769.html, equations 2 and 3 where the initial velocity is 0.

[2] See https://en.wikipedia.org/wiki/Standard_gravity.

question.

Lastly, is there any serious matter or concern bearing on your service as a juror in this case that any juror needs to bring to my attention at this time?

Again, no response from the jurors to that question.

Thank you very much, ladies and gentlemen. We are ready to begin with the evidence in this case.

Mr. Yeager, you may call your first witness.

MR. YEAGER: Thank you, Your Honor. The Commonwealth calls Dr. Alice Newton to the stand.

ALICE NEWTON, M.D., SWORN

THE CLERK: Would you please state your name and spell your name for the record, please.

THE WITNESS: Yes. Good morning. My name is Dr. Alice Newton, A-l-i-c-e Ne-w-t-o-n.

say: No. In fact this really make sense that this is consistent with a fall.

So it's not at all a given, and in fact if you look at the number of reports --

MR. D'ANGELO: Objection.

THE COURT: Stop there. Next question.

Q Doctor, directing your attention to June of 2003, did you begin treating a three-month-old baby girl named Mackenzie Corrigan?

A Yes, I did.

MR. YEAGER: May I approach, Your Honor?

THE COURT: You may.

Q Doctor, I'm placing one item in front of you. Do you recognize what that item is?

A This is the record from Children's Hospital.

MR. YEAGER: Your Honor, may that be entered?

THE COURT: Record of what, Doctor?

THE WITNESS: I'm sorry.

A The medical record -- let me just read this briefly. This is the medical record,

A Well, we certainly asked an ophthalmologist to do a formal examination.

Q And why did you do that?

A Because we wanted to be absolutely certain about what we were looking at. Generally, it's actually very difficult for pediatricians or even neurologists to see this bleeding in the back of the eyes. This was -- it was actually impressive. I remember being impressed with the fact that it was so severe that I could see it because generally it's not that easy to see. But certainly not being an ophthalmologist we wanted to have a specialist confirm that that is in fact what we had.

Q Directing your attention to the next day, June 5, 2003, did you evaluate Mackenzie Corrigan again?

A Yes.

Q And had the ophthalmology consultation been conducted at that time?

A Yes.

Q And did you review those findings?

A I did.

Q After your examination of Mackenzie

Corrigan on June 5, 2003 and your review of the ophthalmology consultation, did you reach an opinion based on your training and experience to a medical degree of certainty as to what was wrong with Mackenzie Corrigan?

A Yes.

Q What was your opinion?

A My opinion, which had been the suspicion from the previous day, was confirmed that she was the victim of Shaken Baby Syndrome.

Q Did Mackenzie Corrigan show any improvement on June 4, 2003 to June 5, 2003?

A No. Her condition deteriorated.

Q And if you could describe that for us please.

A As I said, when she arrived at the hospital she had some -- she already was clearly severely neurologically damaged but had some response to deep pain. By the day -- the next day after injury, she was not making any response to any type of stimulus and was not -- the other factor was that she had been trying sporadically to breathe a little bit on her

Before the Grand Jury of Middlesex County

Tuesday, May 11, 2010

Woburn, Massachusetts

In the Matter of: INVESTIGATION

By:

KATHARINE FOLGER, Assistant District Attorney
Middlesex County

Testimony of:

Q Doctor, are you able to estimate the number of infants and children you have personally examined in your role on the child protection teams at Mass. General and Children's?

A I'd say it's in the thousands.

Q Dr. Newton, are you aware that a baby boy by the name of Nathan Wilson was admitted to Children's Hospital in Boston on March 7th of this year?

A Yes, I am.

Q Were you actually involved in his care, treatment and work up?

A No. I was aware, being the medical director of the team, that there was a child who came through the emergency room but I was not directly involved in his care.

Q Have you had the opportunity to review his medical records?

A Yes, I have.

Q Specifically have you reviewed records from Children's Hospital?

A Yes.

Q Have you reviewed records from Nathan's pediatrician, Dr. Feldman?

A Yes, I have.

Q Have you had the opportunity to review lab work that was conducted at Melrose Wakefield Hospital?
A Yes.
Q As well as some medical records from Beth Israel Deaconess Medical Center where Nathan was born?
A Yes.
Q Have you also had the opportunity to briefly discuss the case with Dr. Peter Cummings at the medical examiner's office?
A Yes, I have.
Q During the course of your review and meetings in this case, did you learn that Nathan's date of birth was August 22, 2009?
A Yes.
Q And did you learn that he was delivered at 35 weeks due to C-section due to maternal preeclampsia and maternal recent hemorrhoid surgery?
A Yes.
Q Just very briefly could you explain what preeclampsia is please?
A Yes. It's a condition where it's a problem that some women have when they're pregnant. Their blood pressure starts to rise and it can lead to other problems if not treated. So women can have blood

He also had retinoscesis or retinal detachment basically. There was so much bleeding that the retina had actually lifted up. It's basically kind of a membrane and it lifted up and folded back down again. And he also had a bleeding, not only in the retina, but along the nerve that travels back from the eye to the brain. And it was quite dramatic in both eyes.

Q Doctor, based upon the history that was obtained at Nathan's admission to Children's Hospital on March 7th, the team's care and treatment of Nathan, your review of the medical records, diagnostic tests and studies, did you arrive at a diagnosis with regards to Nathan Wilson?

A Yes.

Q What was your diagnosis?

A I believe this child was a victim of multiple episodes of abusive head trauma, the last of which was fatal.

Q Could you please explain the basis of your diagnosis?

A Yes. When I say "abusive head trauma" I am speaking about something that used to be called shaken baby syndrome. Now in medicine we call it a more general

term, abusive head trauma. And it is something that we know from studies of cases and from cases where there have been confessions, occurs when a child is very violently shaken. And what happens when that occurs is that first of all they're just tearing the blood vessels that lead to the brain. So there's bleeding around the brain, which can sometimes even cause a high-pressure situation as in this child.

There's also a direct brain injury, which is, I believe, what ultimately led to this child's death. And there also are findings usually in the eyes with bleeding in the retina, something we call retinal hemorrhages, and in the most dramatic cases retinoscesis, or that folding of the retina.

Sometimes we see fractures or bruising, but we don't always see that. So the things we look for to make this diagnosis are a child who has bleeding around the brain, bleeding in the back of the eyes, and brain injury.

Q Could you please describe the types of force that are involved when a baby is shaken?

A Yes. And the way that we know about this is from people who have confessed. So what we understand is

A Oh, immediately after with the person that shook him?

Q Yes; the types of symptoms that would have been visible.

A Well, as I've talked about, I think given the severe nature of the injuries I think he would have become unconscious during being shaken and would have been unconscious with poor muscle tone, probably not breathing well, maybe appearing bluish because he wasn't getting enough oxygen and would not have appeared in any way normal after the injury; meaning that he wouldn't have recovered to any meaningful function. So from the time he was shaken I believe that he was very, very sick and his illness just progressed and worsened. There was no what we call lucid interval or awake time.

Q Doctor, you have mentioned that there was signs of older injuries?

A Yes.

Q If you could please elaborate.

A Yes. So when he had his autopsy, and also actually in the operating room, the neurosurgeon and the pathologist both noticed something called a subdural membrane. And so around-- once they

removed the dural, which is that membrane around the brain, they found not only the new blood that basically spilled out, they also found some old clotted blood, which I believe the pathologist has said was probably about three to four days old. And then they found this, almost like a scab or an area of membrane which is caused when subdural blood is starting to go away. Sometimes you can see an area of kind of thick clot. And so from my knowledge and understanding about inflicted brain injury, I believe this child very likely had at least three episodes where he was violently shaken: One immediately before presenting to the hospital leading to his death; one several days prior. And then--

Q Just for clarification.

A Yes.

Q It was Dr. Cummings opinion, in your conversations with him, that it was three to five days old?

A That's correct. So one three to five days prior and then one at least a week or more prior, which led to the membrane. So there were basically three types of blood clot or fresh blood surrounding the brain with three different ages.

Pages: 204
Exhibits: Nos. 1-3
Word Index: 20 pages

COMMONWEALTH OF MASSACHUSETTS

MIDDLESEX, ss **SUPERIOR COURT DEPARTMENT OF THE TRIAL COURT**

COMMONWEALTH OF MASSACHUSETTS,

Plaintiff

v.

AISLING BRADY MCCARTHY,

Defendant

MICR2013-00525

VOIR DIRE EXAMINATION OF DR. ALICE NEWTON
BEFORE THE HONORABLE MAUREEN HOGAN

APPEARANCES:
For the Commonwealth:
Middlesex County District Attorney's Office
15 Commonwealth Avenue
Woburn, Massachusetts 01801
By: Assistant District Attorney Patrick Fitzgerald
Assistant District Attorney Joseph Gentile
For the Defendant:
Attorney David Meier
Attorney Melinda L. Thompson
Todd & Weld
28 State Street, 31st Floor
Boston, Massachusetts 02109

Woburn, Massachusetts
Courtroom 630
Friday, September 5, 2014

Christina O'Neill, Official Court Reporter
(781) 939-2761

Pediatrics on Monday afternoon and all day Friday, and the remainder of my time was spent at Children's Hospital and a little bit at Massachusetts General Hospital. And I was responsible for consultation on certain days of the week.

Q And do you recall which days of the week you were assigned to be responsible for consultation?

A I believe it was Tuesday and Thursday, as well as Wednesday morning.

Q Did you become aware at some point in time on or before the morning of January 15th of 2013 about a consultation at Children's Hospital?

A Yes.

Q And what information did you have initially?

A I was contacted by my colleague, Celeste Wilson, who's another child-abuse pediatrician, because she was involved initially in the telephone consultation around a child named Rehma Sabir, who came into the hospital on the 14th with a very severe brain injury.

Q And when you arrived at the hospital on the morning of Tuesday, the 15th of January, what did you do with regards to that consultation first?

A Well, just because of the geographic location of the Intensive Care Unit and the Radiology Department, I actually went to the Radiology Department to review a

CAT scan and an MRI that had been obtained overnight on the child. And then I went to the Intensive Care Unit to evaluate the records, the computerized medical records, as well as to meet with the family and evaluate the child.

Q When you arrived at the Radiology Department to review what you knew to be a CT and MRI scan that had been done prior to your arrival, what did you learn from reviewing those scans?

A I reviewed the scans with the neuroradiologist and -- I'm sorry, can I clarify? Are you asking me about the scans or what I knew clinically?

Q What did you do when you first got there?

A I looked at the CT scans to understand the degree of the head injury and understand what findings there were.

Q And you say that you spoke with an individual, a radiologist there?

A Yes, a neuroradiolgist.

Q Do you recall who that was?

A I believe it was Dr. Michelle (sic) Silvera.

Q And what did you learn about the CT findings, CAT scan findings?

A I learned that the CT scan, which had been done, I believe, around seven o'clock on the 14th, showed that

apartment.
Q And there was no report of any accident or external trauma, other than those bruises, --
A That's correct.
Q -- to explain the internal head injury?
A Yes, that's correct.
Q Based on your education, training, experience, review of the relevant medical literature and review of the relevant medical records of Rehma Sabir in this case, as well as the imaging, and considering the reported histories and your physical examination of Rehma, do you have an opinion to a reasonable degree of medical certainty as to the mechanism of Rehma's head injuries?
A Yes.
Q And what is that opinion?
A I believe that she was shaken and slammed, had blunt force trauma to her head, leading to these injuries.
Q And what is the basis of that opinion?
A The basis for believing that she was shaken is the widespread nature of the subdural bleeding, as well as the widespread nature of the brain injury and the presence of retinal hemorrhaging. The reason for the belief that she had blunt force trauma was the presence

of a bruise in a subscapular region that was seen on autopsy.

Q And if you could explain for the Court, which of the symptoms that you considered in this opinion are particular to a blunt force impact versus that of a shaking?

A It's difficult to discriminate between the two, to be honest, because they happen together and they can both lead to similar symptoms of unconsciousness, vomiting or other signs of head injury.

Q Is there anything about the nature of the retinal hemorrhages noted in this case that are specific to shaking or blunt force impact?

A Well, so we don't necessarily see retinal hemorrhages with blunt force impact unless there's a crushing injury. So the fact that there are retinal hemorrhages supports the diagnosis of violent shaking injury.

Q Based on your education, training, experience, review of the relevant medical literature and review of the relevant medical records of Rehma Sabir in this case, as well as the imaging, and considering the reported histories and your physical examination of Rehma, do you have an opinion to a reasonable degree of medical certainty as to the amount of force used to

THE CLERK: Thank you.

THE COURT: Good afternoon.

THE WITNESS: Good afternoon.

CROSS EXAMINATION MS. THOMPSON, Resumed:

Q Good afternoon, Dr. Newton.

A Good afternoon.

Q I'd like to ask you a couple of questions about the differential diagnosis that you've done or, more generally, just a differential diagnosis.

A Yes.

Q You testified on direct examination that you would look at birth records; is that correct?

A Yes. If they're available, yes.

Q Well, you testified that you did look at the birth records, correct?

A I testified that I knew of the birth history, I believe.

Q Okay. And you knew of the birth history from whom?

A From the mother.

Q Okay. And we'll get back to that, but you would agree that the mother's not a doctor?

A No.

Q And birth records can be significant in a case where there's abusive head trauma?

A It's important to know about the birth history, yes.

Q Okay. And in this case did you look at the birth

records?
A No.
Q Okay. So that's one part of a differential diagnosis; is that correct? Birth records?
A Yes. Although --
Q Well, it's one piece of a differential diagnosis?
A Yes.
Q And, in fact, you testified that it's one piece of a differential diagnosis?
A Yes.
Q Okay. But in this case you didn't look at the birth records?
A No.
Q Do you know where the child was born?
A I believe at Beth Israel Hospital.
Q Are you sure?
A No, I'm not sure.
Q Okay.
A It becomes less relevant when a child is one year old, as opposed to a few months old.
Q However, it's relevant?
A Yes.
Q And part of a differential diagnosis is a complete family history, would you agree, --
A Yes.
Q -- going back generations?

Q Correct.
A -- and then go forward in looking for problems.
Q And the lab tests are based upon the clinical history?
A Yes.
Q Or the social history? Conversations with people, --
A Yes.
Q -- right?
A Or the genetic, yes.
Q Or the genetic history, right?
A Yes.
Q So let's just back up and walk through these in this case.
This child came into the hospital on January 14th; is that correct?
A Correct.
Q And on January 15th you suspected that this was abusive head trauma?
A Yes.
Q And during that week, as a matter of fact, you said this was abusive head trauma?
A Yes.
Q Okay. Now, the differential diagnosis has to be done within that time period because of course you do a differential diagnosis before you call it abusive head

COMMONWEALTH OF MASSACHUSETTS

MIDDLESEX, SS.

SUPERIOR COURT DEPARTMENT
DOCKET NO. 2010-596

COMMONWEALTH

V.

GEOFFREY WILSON

LOBBY COPY

NOLLE PROSEQUI

Now comes the Commonwealth and files a *nolle prosequi* in the above-captioned matter. As reasons therefore, the Commonwealth states:

The Medical Examiner originally ruled and testified before the grand jury that the victim's cause of death was "hypoxic-ischemic encephalopathy complicating blunt/shaking injuries of the head" and manner of death was "homicide." The Medical Examiner and Middlesex District Attorney's Office subsequently were provided information concerning a family medical history, which was unknown to the parents at the time of the victim's death and could possibly have played a role in the cause of the victim's death.

The Medical Examiner later opined that his review of the additional information would result in a change in his ruling regarding the cause and manner of death. The Middlesex District Attorney's Office then undertook an independent review of the "newly discovered" family medical history. Additional genetic testing of certain family members was requested, agreed to, and provided. A thorough review of the medical records and relevant medical literature was conducted and discussed with the Medical Examiner. Additionally, experts in the fields of clinical genetics and anatomic, clinical and forensic pathology were

consulted. Subsequently, the Medical Examiner officially revised his ruling in this case. The revised cause of death is "hypoxic-ischemic encephalopathy complicating subdural hematoma of uncertain etiology" and the manner of death is "could not be determined." Had this matter proceeded to trial, the Medical Examiner would not, to a reasonable degree of medical certainty, have been able to opine as to whether the cause of death was due to inflicted injury or natural causes.

Given the current state of the evidence, the Commonwealth cannot meet its burden of proving cause of death beyond a reasonable doubt when the revised ruling is considered in light of all the circumstances of this case. Consequently, this *nolle prosequi* is filed in the interests of justice.

Respectfully Submitted
For the Commonwealth,

MARIAN T. RYAN
DISTRICT ATTORNEY

By: Katharine B. Folger

Katharine B. Folger
Assistant District Attorney
Child Protection Unit
15 Commonwealth Avenue
Woburn, MA 01801
(781) 897-8400
BBO No. 630190

Dated: September 18, 2014

REGISTRY DIVISION OF THE CITY OF BOSTON

COUNTY OF SUFFOLK, COMMONWEALTH OF MASSACHUSETTS, UNITED STATES OF AMERICA

Certificate Number

№ 224189

I, the undersigned, hereby certify that I hold the office of _____ City Registrar of the City of Boston and I certify the following facts appear on the records of Births, Marriages and Deaths kept in said City as required by law.

AFFIDAVIT AND CORRECTION OF DEATH
REGISTRY OF VITAL RECORDS AND STATISTICS

REGISTERED NUMBER 1443 — STATE USE ONLY

RETURN ADDRESS BOSTON — DISPOSITION NO. 231

1. DECEDENT – NAME: FIRST NATHAN	MIDDLE GEOFFREY JEHAN	LAST WILSON	2. SEX: MALE	3. DATE OF DEATH (Mo., Day, Yr.): MARCH 7, 2010
4a. PLACE OF DEATH (City/Town): BOSTON	4b. COUNTY OF DEATH: SUFFOLK	4c. HOSPITAL OR OTHER INSTITUTION – Name (If not in either, give street and number): CHILDREN'S HOSPITAL		
5. PLACE OF DEATH (Check only one): HOSPITAL: Inpatient, ER/Outpatient, DOA; OTHER: Nursing Home, Residence, Other (specify)		6. SOCIAL SECURITY NUMBER: .026-92-8711	7. PLUS WAR VETERAN SPECIFY WAR: ---	
8a. WAS DECEDENT OF HISPANIC ORIGIN? If yes, Specify Puerto Rican, Dominican, Cuban, etc.: No, Yes	8b. RACE (e.g. White, Black, American Indian, etc.) (Specify): SRI LANKAN/AFRICAN AMERICAN	9. DECEDENT'S EDUCATION (Highest Grade Completed) Elementary/Sec (0-12); College (1-4, 5+): 0		
10a. AGE – Last Birthday (Yrs.): UNDER 1 YEAR MOS 6 DAYS 13	10b. UNDER 1 DAY HOURS MINS	10c. DATE OF BIRTH (Mo., Day, Yr.): AUGUST 22,2009	11. BIRTHPLACE (City and State or Foreign Country): BOSTON, MA	
12. MARRIED, NEVER MARRIED, WIDOWED, OR DIVORCED: NEVER MARRIED	13. LAST SPOUSE (If wife, give maiden name): - - - - - - - -	14a. USUAL OCCUPATION (Print, if military): - - - - - - - -	14b. KIND OF BUSINESS OR INDUSTRY: - - - - -	
15a. RESIDENCE – NO. & ST., CITY/TOWN, COUNTY, STATE/COUNTRY: 36 GREENLEAF ST., MALDEN, MIDDLESEX, MA				15b. ZIP CODE: 02148
16. FATHER – FULL NAME: GEOFFREY WILSON	17. STATE OF BIRTH (If not in U.S. name country): CA	18. MOTHER – NAME (MAIDEN): DILKUSHI WIJESINGHE	19. STATE OF BIRTH (If not in U.S. name country): SRI LANKA	
20. INFORMANT'S NAME: DILKUSHI WIJESINGHE	21. MAILING ADDRESS – NO. & ST., CITY/TOWN, STATE, ZIP CODE: 36 GREENLEAF ST., MALDEN, MA 02148		22. RELATIONSHIP: MOTHER	
23. METHOD OF IMMEDIATE DISPOSITION: Burial, Cremation, Entombment, Removal from State, Donation, OTH (SPEC):	24. FUNERAL SERVICE LICENSEE OR OTHER DESIGNEE: MARIANNE M BURKE		25. LICENSE #: 7535	
26a. PLACE OF DISPOSITION (Name of Cemetery, Crematory, or other): NEWTON CEMETERY	26b. LOCATION (City/Town, State): NEWTON, MA			
27. DATE OF DISPOSITION (Mo., Day, Yr.): MARCH 20,2010	28a. NAME AND ADDRESS OF FACILITY OR OTHER DESIGNEE: HENRY J BURKE & SONS FUNERAL HOME	28b. 56 WASHINGTON ST WELLESLEY MA 02481		

29. PART I – Enter the diseases, injuries, or complications that caused the death. Do not use only the words of dying, such as cardiac or respiratory arrest, shock or heart failure. List only one cause on each line (a through c). PRINT OR TYPE LEGIBLY. — Approximate Interval Between Onset and Death

IMMEDIATE CAUSE (Final disease or condition resulting in death): a. HYPOXIC-ISCHEMIC ENCEPHALOPATHY

DUE TO (OR AS A CONSEQUENCE OF):

Sequentially list conditions, if any, leading to immediate cause. Enter UNDERLYING CAUSE (disease or injury that initiated events resulting in death) LAST: b. COMPLICATING SUBDURAL HEMATOMA OF UNCERTAIN ETIOLOGY — HOURS

DUE TO (OR AS A CONSEQUENCE OF):

c.

DUE TO (OR AS A CONSEQUENCE OF):

d.

PART II – Other significant conditions contributing to death but not resulting in underlying cause given in Part I

30. WAS AUTOPSY PERFORMED? (Yes or No): 31 Yes

32. WERE AUTOPSY FINDINGS AVAILABLE PRIOR TO COMPLETION OF CAUSE OF DEATH? (Yes or No): 32 Yes

33. MEDICAL EXAM./CORONER? (Yes or No): Yes	34. MANNER OF DEATH: Natural, Accident, Homicide, Suicide, Could not be determined, Pending Investigation	35a. DATE OF INJURY (Mo., Day, Yr.): ---	35b. TIME OF INJURY: ---	35c. INJURY AT WORK? Yes, No: No

36a. DESCRIBE HOW INJURY OCCURRED: ---

36b. PLACE OF INJURY – (At home, farm, street, factory, office bldg., etc.) Specify: ---

36c. LOCATION No. & St., City/Town, State: ---

37a. DATE SIGNED (Mo., Day, Yr.):	37b. HOUR OF DEATH:	37c. DATE SIGNED (Mo., Day, Yr.): MAY 20, 2010	37d. HOUR OF DEATH: 10:25 PM
38a. NAME OF ATTENDING PHYSICIAN, IF NOT CERTIFIER:		38b. PRONOUNCED DEAD (Mo., Day, Yr.): MARCH 07, 2010	38c. PRONOUNCED DEAD (Hr.): 10:25 PM

39a. NAME AND ADDRESS OF CERTIFYING PHYSICIAN OR MEDICAL EXAMINER (Type or Print): Peter M.E. Cummings, M.Sc., MD720 ALBANY STREET, BOSTON, MA 02118

39b. LICENSE NO. OF CERTIFIER: 235452

40a. WAS BURIAL PERMIT ISSUED? (Yes or No): Yes	40b. IF YES, DATE PRONOUNCED:	40c. IF YES, TIME PRONOUNCED:	40d. NAME OF PRONOUNCER: NAME	TITLE: ORN DPA

41. DATE BURIAL PERMIT ISSUED: MARCH 19, 2010	42. RECEIVED IN CITY/TOWN OF: BOSTON	43. DATE OF ORIGINAL RECORD: MARCH 19, 2010

41. Patricia A McMahon — CLERK OR REGISTRAR — AUGUST 1, 2014

WITNESS my hand and the SEAL of the CITY REGISTRAR

SEP 16 2014

on this _____ day of _____ A.D. _____

Patricia A McMahon — City Registrar

By Chapter 314 of the Acts of 1892, "the certificates or attestations of the Assistant City Registrars shall have the same force and effect as that of City Registrar."

I hereby certify that the above and foregoing is a true copy of the record in the custody of the City Registrar of the City of Boston.

Attested:

East Boston	1852
South Boston	1804
Roxbury	1868
Dorchester	1870
Charlestown	1874
Brighton	1874
West Roxbury	1874
Hyde Park	1912

5/29/14

COMMONWEALTH OF MASSACHUSETTS

MIDDLESEX, ss

SUPERIOR COURT DEPARTMENT
NO. 2010-596

COMMONWEALTH	)
V.	)
GEOFFREY WILSON	)

DEFENDANT'S MOTION TO AMEND BAIL CONDITIONS

The defendant, Geoffrey Wilson, moves that this Court amend his bail conditions in the following respects:

1. Remove the 10 pm curfew, and

2. Authorize the defendant to travel out of the Commonwealth, provided that he has the prior approval of his probation officer and subject to all terms imposed by his probation officer.

As grounds for this motion, the defendant states the following:

Geoff Wilson is charged with murder of his infant son, Nathan, and is currently released on bail of $20,000 set on September 30, 2010 by Justice D. Lloyd MacDonald. He is monitored by the Middlesex County Probation Department and wears a GPS ankle bracelet to confirm his movements. He has no prior

2014 May 29 | Allowed. Attest: Jonathan B. Healy Mgr (Hamden)

Copies given to Probation Dept 5-30-14

criminal record whatsoever. He has fully complied with his bail conditions at all times since his release.

Geoff was born in 1978 and is 36 years old. He was raised in California, and moved to the Boston area in 1996 to attend college at M.I.T. He has been married to his wife, Dilkushi (Dilly) Wilson, since 2003. Dilly's immediate family lives in Buffalo, New York. Geoff's mother lives in California, and his father is deceased.

On August 22, 2009, Geoff and Dilly welcomed a son, Nathan, into their family. Geoff was thrilled to have a son, and was a loving father. Soon after Nathan's birth, it became clear that he had several medical problems. Nathan was admitted to the Neonatal Intensive Care Unit (NICU) after his birth, and spent nearly a week in the hospital. Nathan's health needs were attended to by his parents, who had twenty-three interactions with his pediatrician between August 31, 2009 and March 4, 2010.

On the morning of March 7, 2010, Mr. Wilson was home with Nathan while Dilly went to church. Geoff spent the morning playing with and feeding Nathan, then put him down for a nap. When Dilly returned from church, she and Geoff found Nathan unresponsive in his crib. After Nathan's condition did not improve significantly and they learned that his pediatrician was unavailable, Dilly and Geoff rushed Nathan to Children's Hospital emergency department. The doctors discovered that

Nathan had bleeding in his skull and performed emergency surgery, but tragically, Nathan could not be saved. Geoff was arrested that night for murder under the theory of Shaken Baby Syndrome, before any autopsy was conducted, based primarily on the fact that he had been alone with Nathan when the bleeding in the brain apparently began.

The defendant would present five experts at trial: a forensic pathologist, a neuropathologist, a pediatrician, a pediatric neurologist, and an ophthalmologist. Each of these specialists disputes the Shaken Baby Syndrome diagnosis - they believe that an underlying medical condition led to Nathan's death. The experts were convinced in large part by Nathan's family history evincing a pattern of genetic disorders related to collagen, the substance of which human veins and arteries are constructed. This family history information was not available to the medical examiner at the time of Nathan's autopsy.

After a hearing on September 9, 2013, the prosecutor asked defense counsel for more information on the defense's case. This led to a series of unusual events, including a meeting between the defendant's forensic pathologist and the medical examiner who performed the autopsy. The defendant's counsel also fully presented the anticipated medical testimony to the prosecutors in significant detail, both orally and in a binder containing specific references to the medical records. Defense counsel also

took the extraordinary step of allowing the prosecutor who leads the Child Abuse Unit, along with the prosecutor who heads the Homicide Unit, and a Massachusetts State Police detective assigned to the District Attorney's Office, to meet the defendant and freely question him. The District Attorney has informed the defense that the medical examiner who performed the autopsy has changed his conclusion of the cause of death from homicide to undetermined. The prosecutor has asked that this finding be reviewed by the Chief Medical Examiner. The trial has been postponed to an as-yet-undetermined date following these developments.

The defendant moves that the Court reconsider his bail terms. Geoffrey Wilson has been released on bail since 2010 with exemplary behavior, and a trial date has not been set. The defendant's wife has serious medical ailments that require frequent, and sometimes overnight, treatment. Geoff would like to be able to accompany his wife to all of her hospital visits, including those that last overnight. In addition, Geoff has had to forgo certain religious services associated with his practice as a Jehovah's Witness because of his curfew. The Kingdom Hall of Jehovah's Witnesses has always played an important role in Geoff's life. Prior to his arrest, he held a leadership role as a minister. Geoff and Dilly attended an annual multi-day religious convention in western Massachusetts since they began

dating. They have been unable to participate in this event since Nathan's death and Geoff's arrest, as the trip would require them to stay away from home overnight.

Family has always been very important to Geoff and Dilly, especially during the difficult time since they lost their son. Unfortunately, Dilly's mother is no longer healthy and cannot travel to Massachusetts from upstate New York. Geoff would like to be able to travel to Buffalo so that he and his wife can visit her and Dilly's father. Dilly cannot travel this far alone. Geoff was permitted by the Court to travel to California in 2012 to assist his mother as she recovered from cancer treatment. Defense counsel submits that Geoff should be allowed to travel outside of the Commonwealth with the prior approval of his probation officer, subject to the terms that his probation officer requires. These terms likely will include submission of the dates, times, and means of travel; daily phone check-ins; and the address where Geoff will stay while out of the Commonwealth.

WHEREFORE, the defendant moves that this Court amend his bail conditions to remove his daily curfew, and to allow him to travel outside of the Commonwealth with the prior approval of his probation officer.

Geoffrey Wilson
By His Attorneys,

CARNEY & ASSOCIATES

J. W. Carney, Jr.

J. W. Carney, Jr.
B.B.O. # 074760
Samir Zaganjori
B.B.O. # 663917
Danya Fullerton
B.B.O. #683134

Carney & Associates
20 Park Plaza, Suite 1405
Boston, MA 02116
617-933-0350 work
617-943-5178 cell
jcarney@CARNEYdefense.com

May 22, 2014

CERTIFICATE OF SERVICE

I hereby certify that a true copy of the above document was served upon the attorney of record for each other party by mail or email or fax or in hand on or before the above date.

J. W. Carney, Jr.

J. W. Carney, Jr.

COMMONWEALTH OF MASSACHUSETTS

MIDDLESEX, SS

SUPERIOR COURT
No. 2010-596

COMMONWEALTH

V.

GEOFFREY WILSON

AFFIDAVIT SUPPORTING
DEFENDANT'S MOTION TO AMEND BAIL CONDITIONS

I, J. W. Carney, Jr., state that the facts contained in the attached motion are true to the best of my information and belief.

Signed under the penalties of perjury.

J. W. Carney, Jr.

J. W. Carney, Jr.

May 22, 2014

COMMONWEALTH OF MASSACHUSETTS

MIDDLESEX, SS.

SUPERIOR COURT DEPARTMENT
DOCKET NO. 1381CR00525

COMMONWEALTH

V.

AISLING BRADY MCCARTHY

August 21, 2015
Filed in court

NOLLE PROSEQUI

Now comes the Commonwealth and files a *nolle prosequi* in the above-captioned case pursuant to Rule 16(a) of the Massachusetts Rules of Criminal Procedure.

As reasons therefore, the Commonwealth states as follows:

At approximately 4:30 p.m. on January 14, 2013, the deceased, Rehma Sabir, a 12-month old infant, was found unresponsive in her crib after having been in the sole care and custody of the defendant since 9:30 a.m. that day. Shortly thereafter, the child was transported by ambulance to Boston Children's Hospital. The child remained unconscious and was diagnosed with acute, extensive and severe injuries for which neurosurgical intervention was deemed futile. Other medical intervention was ultimately unsuccessful in preventing her death, which occurred at approximately 4:30 p.m. on January 16, 2013. Medical professionals in a variety of disciplines observed various specific injuries, the constellation of which, combined with the medical history acquired and absence of any reported history of a major trauma, were consistent with non-accidental trauma, specifically abusive head trauma.

The Medical Examiner who performed the autopsy originally ruled that the victim's death was caused by "complications of blunt force head injuries" and that the manner of death was

#186

"homicide." Based on that opinion and other evidence, the grand jury returned the indictment in this case. In April of 2015, the Medical Examiner undertook a review of additional newly-submitted materials.

On the afternoon of August 27, 2015, this office was informed that the Medical Examiner made a decision to change the cause and manner of death. The Medical Examiner has amended the cause of death on the death certificate to "complications of subdural hemorrhage of uncertain etiology" and has amended the manner of death to "undetermined." Additionally, the Medical Examiner has indicated that if this case proceeds to trial, she cannot opine to a reasonable degree of medical certainty that the cause of death was an inflicted injury as opposed to accident or natural causes.

Given this significant change in the state of the evidence, the Commonwealth believes that, at this time, it can no longer meet its burden of proving an essential element of the charged crime beyond a reasonable doubt. Consequently, this *nolle prosequi* is filed in the interests of justice.

Respectfully Submitted
For the Commonwealth,

MARIAN T. RYAN
DISTRICT ATTORNEY

By: _____

Patrick G. Fitzgerald, Esq.
Assistant District Attorney
15 Commonwealth Avenue
Woburn, MA 01801
(781) 897-8300
BBO No. 652018

Dated: August 31, 2015

Nanny's murder case dropped

Woman jailed 2 years won't face trial after state says it can't prove Cambridge infant's death was homicide

Boston Globe - Boston, Mass.
Subjects: Trials; Head injuries; Medical examiners; Press conferences
Author: Schworm, Peter; Wen, Patricia; Andersen, Travis
Date: Sep 1, 2015
Start Page: A.1
Section: Metro

Document Text

In a stunning reversal, prosecutors Monday dropped criminal charges against Aisling Brady McCarthy, the nanny accused of murdering a Cambridge infant in her care in 2013, after the medical examiner's office said it could no longer stand by its ruling that the child's death was a homicide.

The revised ruling, which followed a lengthy and highly unusual review of the shaken-baby case, found that 1-year-old Rehma Sabir's medical history "could have made her prone to easy bleeding with relatively minor trauma," raising serious doubts that the child was a victim of abusive head trauma.

"I believe that enough evidence has been presented to raise the possibility that the bleeding could have been related to an accidental injury in a child with a bleeding risk or possibly could have even been a result of an undefined natural disease," medical examiner Katherine Lindstrom wrote.

The ruling marked the second time in less than a year the medical examiner's office had changed its stance on a shaken-baby case. It made McCarthy, a 37-year-old native of Ireland who has steadfastly maintained her innocence, a free woman.

"Miss McCarthy was put in jail for 21/2 years over a crime that never occurred," said McCarthy's lawyer, Melinda Thompson. "Not just a crime that she did not commit, but a crime that did not occur. The life of an innocent woman was ruined."

At an afternoon news conference, Thompson said McCarthy "pretty much can't stop crying out of joy" over the news and hopes to return to Ireland soon. She has been living in the United States illegally since 2002.

"She's relieved, scared, and absolutely thankful" to the medical examiner's office, she said. Thompson described the prosecution's handling of the case and the charges against McCarthy as "a complete disgrace."

"I think there was a rush to judgment," she said.

McCarthy, a Quincy resident, did not attend the news conference, and Thompson said she was not available for comment Monday night.

The child's parents, Sameer Sabir and Nada Siddiqui, also could not be reached for comment.

In Ireland, where the case has received intense media attention, news that McCarthy was no longer on trial brought rejoicing.

"It's fantastic news, just fantastic news," said the Rev. Kevin Fay, a priest from Mc Carthy's hometown, Lavey, in County Cavan. "We all know the family very well. There's a sense of great relief for everybody."

The accusation that a nanny caused the death of an infant in her care generated widespread shock and prompted outrage on both sides of the Atlantic.

Prosecutors had alleged that McCarthy had assaulted Rehma on Jan. 14, 2013, causing massive brain injuries. Alice Newton, a specialist who determined that the child died of abusive head trauma, concluded the injuries could not have been inflicted before that day.

Tests found that the infant suffered from extensive bleeding in her brain and behind her eye, acute injuries that specialists said led directly to her death. There was "no other medical explanation for Rehma's injuries or death," Newton wrote.

But McCarthy's defense team, tapping into growing doubts about the science behind shaken-baby cases, submitted reports from a range of specialists challenging the cause of death. They asserted that the baby had been sick much of her short life, suffering from

a bleeding disorder and gastrointestinal problems, and that she sustained bone fractures weeks before her death, when she had no contact with McCarthy.

In April, the medical examiner's office said it was conducting a comprehensive review to determine whether the child was intentionally harmed, and prosecutors said they did not want to proceed to trial until it was complete. A short time later, McCarthy was released on bail to home confinement.

Only months before, prosecutors dropped murder charges against a Malden man when the medical examiner's office changed its ruling that his 6-month-old son had died from head injuries caused by shaking, saying the cause of death could not be determined.

Newton, the specialist who determined that the Malden child died of abusive head trauma, defended her initial diagnosis in the Sabir case, despite the medical examiner's shift.

"I stand by my comments in court," said Newton, now the head of the child protection team at Massachusetts General Hospital.

In a hearing in September 2014, Newton said Sabir sustained massive brain injuries that were consistent with a "violent shaking injury."

A search conducted the day after Rehma died uncovered blood stains on a pillow and blanket in her crib and on discarded baby wipes, police said. Investigators also found that part of the wall beside the changing table had been broken off, damage consistent with "forceful contact" from the table corner.

But in its review, the medical examiner's office noted that the girl's overall health and past medical issues "raise the possibility that she had some type of disorder that was not able to be completely diagnosed prior to her death."

"Given these uncertainties, I am no longer convinced that the subdural hemorrhage in this case could only have been caused by abusive/inflicted head trauma, and I can no longer rule the manner of death as a homicide," Lindstrom wrote.

Middlesex District Attorney Marian Ryan said prosecutors were notified late Thursday that the medical examiner's office had changed its opinion.

The office "could no longer opine to a reasonable degree of medical certainty that the injuries were inflicted," she said.

Ryan said prosecutors pursued the case "based on the evidence we had" and dropped it after concluding they could not "meet its burden of proof."

Gerry Leone, the then-Middlesex district attorney who initiated the case against McCarthy, said he did not know the details of prosecutors' decision to drop the charges and declined further comment.

Stephen Weymouth, a Boston criminal defense attorney who has followed media coverage of the case, said the revised finding could be seen as an exoneration of McCarthy, especially since prosecutors spent nearly three years working on the case.

But, he added, it does not mean that McCarthy can never face murder charges again, because there is no statute of limitations for murder.

Also, prosecutors dropped charges before a jury was sworn, meaning that double jeopardy constitutional protections do not apply to McCarthy.

"She could conceivably be charged with, and prosecuted for, murder in the future if other evidence that can be utilized is found," Weymouth said. "I think it's highly unlikely."

Robert Sege, a Boston pediatrician and a member of the American Academy of Pediatrics committee on child abuse and neglect, said shaken-baby syndrome remains a tragic cause of many baby deaths, and the legal dismissal of some cases should not undermine this point.

Thompson, meanwhile, said McCarthy could not rule out a lawsuit against the authorities for wrongful prosecution.

She said the detainer that federal immigration officials lodged against McCarthy remains in effect, keeping her confined in her home.

Thompson said McCarthy's time in jail was "a complete nightmare" and said she hopes to return home to Ireland soon.

Hopefully, she said, US immigration officials will "use their discretion and let her get on a plane and go home as soon as possible."

Eric Moskowitz and John R. Ellement of the Globe staff contributed to this report. Peter Schworm can be reached at schworm@globe.com. Follow him on Twitter @globepete.

Credit: By Peter Schworm, Patricia Wen, and Travis Andersen Globe Staff

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Abstract (Document Summary)

In a stunning reversal, prosecutors Monday dropped criminal charges against Aisling Brady McCarthy, the nanny accused of murdering a Cambridge infant in her care in 2013, after the medical examiner's office said it could no longer stand by its ruling that the child's death was a homicide. Only months before, prosecutors dropped murder charges against a Malden man when the medical examiner's office changed its ruling that his 6-month-old son had died from head injuries caused by shaking, saying the cause of death could not be determined.

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YVONNE ABRAHAM

Autopsy notes in baby's death raise questions about DA's role

Medical examiner says he was bullied

DINA RUDICK/GLOBE STAFF/FILE

A former medical examiner, in notes left in the case file of a 6-month-old, said Middlesex District Attorney Marian Ryan bullied him.

By Yvonne Abraham | GLOBE COLUMNIST OCTOBER 13, 2015

The medical examiner who reversed a homicide finding in the death of a 6-month-old Malden baby maintains in case notes that Middlesex District Attorney Marian Ryan and her office "bullied" him to stick with the original homicide ruling, even though new evidence made that conclusion not "honest."

Initially, forensic pathologist Peter Cummings ruled that the 2010 death of the baby, Nathan Wilson, was a homicide, the result of abusive head trauma, or shaken baby syndrome. His father, Geoffrey Wilson, was charged by Ryan's office with the baby's murder.

But experts hired by his defense counsel found a rare genetic defect that made members of the Wilson family

prone to ruptures of veins and arteries, providing an alternative explanation of the baby's death. Cummings decided to file an amended death certificate changing the cause of death from "homicide" to "could not be determined."

In supplemental case notes, handwritten by Cummings and placed in the baby's closed case file the day before the murder charges were dropped, Cummings said that the DA and her staff had not wanted him to change his finding. He called the way they dealt with his office on the case "unethical and unprofessional." He accused them of "M.E. shopping" in the hopes of getting a different opinion.

"I told them I felt bullied and at times as though I was being forced to sign the case out in a way I did not think was honest," Cummings wrote in the case notes, obtained by the Globe.

Charges dropped in baby's 2010 death

A former MIT employee was accused of killing his 6-month-old son, but charges were dropped after questions were raised about an expert's testimony.

Cambridge nanny's murder case dropped

The district attorney rejects the claims and said she is "puzzled" by them, since her dealings with Cummings and his colleagues were respectful.

"Did we ask for second opinions?" she said. "Yes we did, we absolutely did. Nobody does anything medical without a second opinion. And in the end we did agree with his conclusion, and we were bound by that."

In the Wilson case, Ryan said, her office was the only party speaking up for the child, since both parents were united in their denial that Geoffrey Wilson had hurt his baby. Ryan said she could not drop the case against him lightly.

"Who else would have cried foul here?" she asked. "If the claim is that I am very careful before I exercise that power then yes, I am."

But Cummings's notes suggest Ryan went beyond careful. The claim is that her office tried to pressure a medical expert to stick with his original conclusion even in the light of the new information.

Nonetheless, Cummings appears to have withstood the pressure and moved ahead with revising the baby's cause of death, amending the death certificate on Aug. 1, 2014. (The chief medical examiner backed that new finding.)

Ryan, who fought a heated primary election Sept. 9, dropped the murder charge against Geoffrey Wilson on Sept. 18.

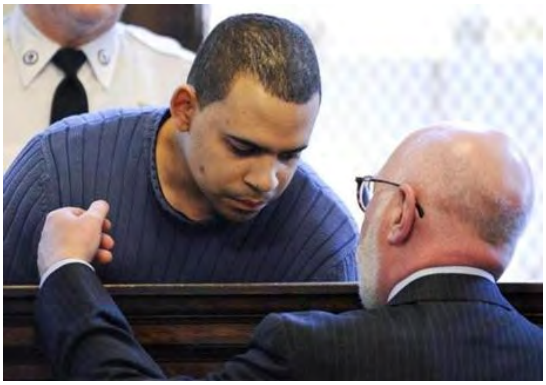
Cummings, who parted amicably with the Office of the Chief Medical Examiner in June, declined Monday to comment on the Wilson case.

"I do not feel it is appropriate for me to comment publicly on issues involving my former employer or cases

under the jurisdiction of the Medical Examiner's Office," he wrote in an e-mailed statement.

The Wilsons declined to comment, but their attorney said they are troubled by the allegations contained in Cummings's notes.

"This document raises very significant concerns about the extent to which an independent medical examiner could feel bullied," said Jeffrey Catalano. "We are looking deeply into this in order to determine what the Wilsons' rights are, and the best means of seeking justice in this case."



JOSH LONDON/ASSOCIATED PRESS/FILE

Geoffrey Wilson spoke to J.W. Carney Jr., his attorney, after his March 2010 arraignment in Malden District Court.

District attorneys and medical examiners work together on homicides: Medical examiners use information gathered by the prosecution team to inform their conclusions on the cause and manner of death, but not to influence them.

"We must have the independence and latitude to arrive at our medical opinions and conclusions," said Marcus Nashelsky, president of the National Association of Medical Examiners, who spoke only generally and is not familiar with the details of this case. "It is absolutely inappropriate for a law enforcement officer, a prosecutor, or a defense attorney to attempt to influence the diagnostic medical work of a forensic pathologist."

Ryan maintains neither she nor her prosecutors crossed that line.

She said her office maintained a good, "very professional" relationship with the medical examiner. Two people with knowledge of dealings between the two offices disagreed, calling their relationship toxic.

In a second, strikingly similar case involving a baby's death, concerns about possible interference arose again.

Attorneys for Aisling Brady McCarthy were sufficiently worried about Ryan trying to influence the medical examiner that they asked a judge to order the Middlesex district attorney and her office to refrain from contacting the examiner in the case of Rehma Sabir.

McCarthy, Sabir's nanny, was initially charged with murdering the 1-year-old in January 2013 after the medical examiner found that Sabir had died from abusive head trauma.

McCarthy's attorneys maintained their client's innocence, citing the baby's complicated medical history and findings that the baby had suffered bone injuries weeks earlier, when she was out of the country and away from McCarthy.

The medical examiner's office launched a review of the case in April. Melinda Thompson, one of McCarthy's attorneys, said she was not confident the review would be truly independent unless the judge required the Middlesex DA to stay away from the medical examiner.

The judge granted her request.

"I felt the interactions [so far] were inappropriate," Thompson said, citing e-mails showing heavy involvement

by the entire prosecution team with the medical examiner. “We believed the review, on behalf of Aisling, the child’s family, and the Commonwealth, should be independent, without interference from the Middlesex district attorney’s office.”

On Aug. 31, the medical examiner revised Rehma Sabir’s cause of death to “undetermined,” like Nathan Wilson’s.

The district attorney dropped the charges that same day.

Yvonne Abraham is a Globe columnist. She can be reached at yvonne.abraham@globe.com. Follow her on Twitter @GlobeAbraham.

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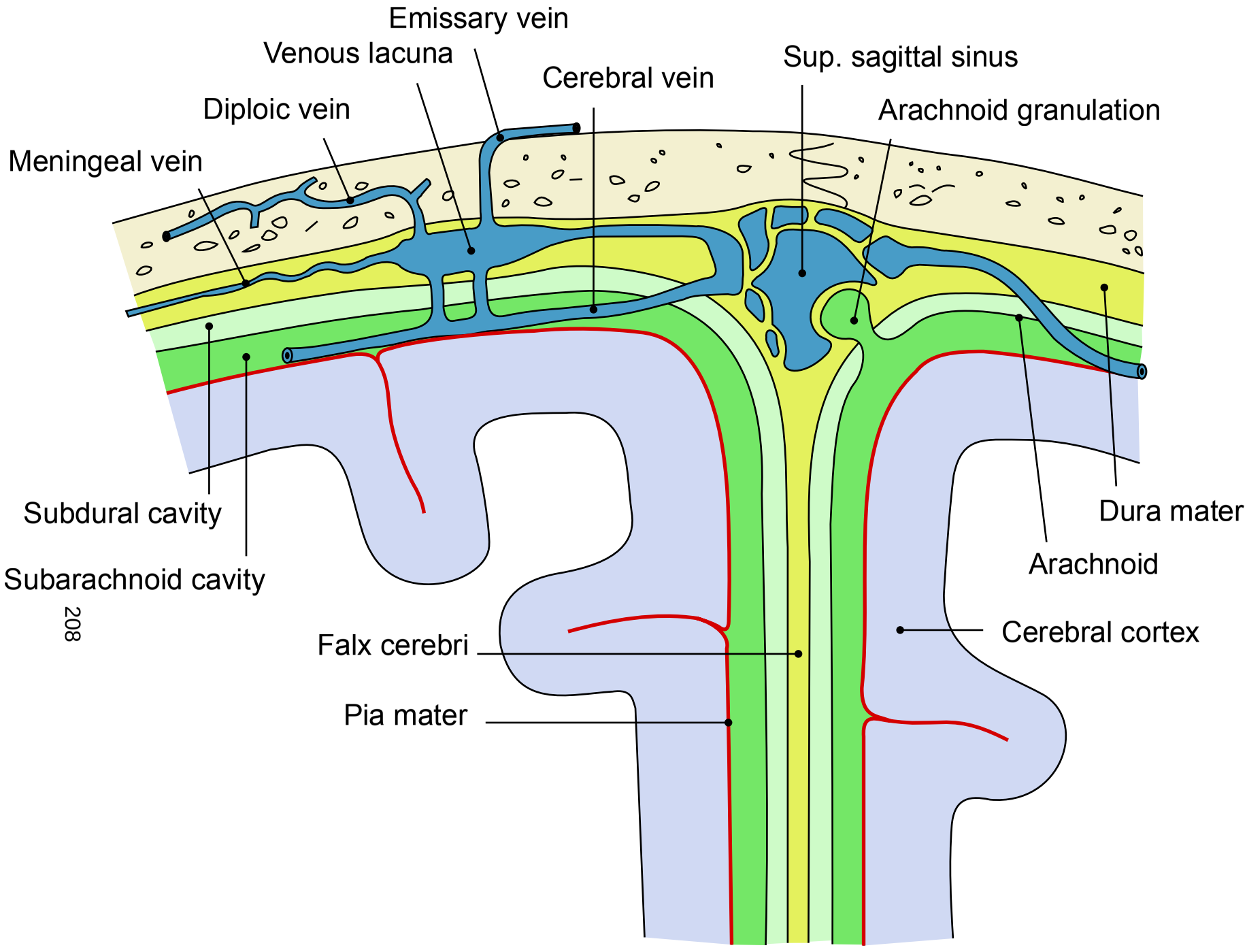
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Meningeal vein
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Arachnoid
Subarachnoid cavity
Cerebral cortex
Falx cerebri
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