Imaging of Nonaccidental Injury and the Mimics: Issues and Controversies in the Era of Evidence-Based Medicine

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KEYWORDS

- Evidence-based medicine • Nonaccidental injury
- Nonaccidental trauma • Nonaccidental head injury
- Child abuse

Nonaccidental injury (NAI) is reportedly the most frequent cause of traumatic injury in infants (peak incidence age 6 months; 80% of traumatic brain injury deaths under the age of 2 years).[1–4] NAI, nonaccidental trauma (NAT), and nonaccidental head injury are more recently used terms instead of the traditional labels, child abuse, battered child syndrome, and shaken baby syndrome (SBS). The traditional definition of NAI/SBS is intentional or inflicted physical injury to infants characterized by the triad of (1) subdural hemorrhage (SDH), (2) retinal hemorrhage (RH), and (3) encephalopathy (ie, diffuse axonal injury [DAI]) occurring in the context of inappropriate or inconsistent history (particularly when unwitnessed) and commonly accompanied by other apparently inflicted injuries (eg, skeletal).[1–4] This empirical formula is under challenge by evidence-based medical and legal principals.[4–14]

TRAUMATIC BRAIN INJURY

Traumatic brain injury has been categorized in several ways.[1,4] Primary injury directly results from the initial traumatic force and is immediate and irreversible (eg, contusion or shear injury). Secondary injury arises from or is associated with the primary injury and is potentially reversible (eg, swelling, hypoxia-ischemia, seizures, or herniation). Traditional biomechanics describes impact loading as linear forces that produce localized cranial deformation and focal injury (eg, fracture, contusion, or epidural hematoma). Accidental injury (AI) is considered typically associated with impact and, with the exception of epidural hematoma, is usually not life threatening. Impulsive loading refers to angular acceleration/deceleration forces resulting from sudden nonimpact motion of the head on the neck (ie, whiplash) and produces diffuse injury with tissue disruption (eg, bridging vein rupture with SDH and white matter shear with DAI). Young infants are thought particularly vulnerable to the latter mechanism (ie, SBS) because of weak neck muscles, a relatively large head, and an immature brain. SBS is traditionally postulated to result in the triad of primary traumatic injury (ie, SDH, RH, and DAI), which has been reportedly associated with the most severe and fatal CNS injuries. Stated assault mechanisms

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in NAI include battering, shaking, impact, shaking-impact, strangulation, suffocation, and combined assaults (shake-bang-choke).[1–4] Although the spectrum of injury in NAI overlaps that of AI, certain patterns have been previously reported as characteristic of or highly suspicious for NAI.[1–4] These include multiple or complex cranial fractures (**Fig. 1**), acute interhemispheric SDH (**Fig. 2**), acute-hyperacute SDH (**Fig. 3**), DAI, chronic SDH, and the combination of chronic and acute SDH (**Fig. 4**). The latter combination is thought indicative of more than one abusive event. Imaging evidence of brain injury may occur with or without other clinical findings of trauma (eg, bruising) or other traditionally higher-specificity imaging findings of abuse (eg, classic metaphyseal lesions or rib fractures) (**Fig. 5**).[1–4] Therefore, clinical and imaging findings of injury out of proportion to the history of trauma and injuries of different ages have been the basis of making a medical diagnosis and offer expert testimony that such "forensic" findings are "proof" of NAI/SBS, particularly when encountered in premobile, young infants.

EVIDENCE-BASED MEDICINE

Evidence-based medicine (EBM) is now the guiding principle as medicine moves from an authoritarian to an authoritative era to overcome bias and ideology.[4,15–20] EBM quality-of-evidence ratings of the literature (eg, classes I–IV) are based on levels of accepted scientific methodology and biostatistical significance (eg, *P* values) and apply to the formulation of standards and guidelines for every aspect of medicine, including diagnostics, therapeutics, and forensics. EBM analysis reveals that few published reports in the traditional NAI/SBS literature merit a quality-of-evidence rating above class IV (eg, expert opinion alone).[5] Such low ratings do not meet EBM recommendations for standards (eg, level A) or for guidelines (eg, level B). Difficulties exist in the rational formulation of a medical diagnosis or forensic determination of NAI/SBS based on an alleged event (eg, shaking) that is inferred from clinical, imaging, or pathology findings in the subjective context of (1) an unwitnessed event, (2) a noncredible history, or (3) an admission or confession under dubious circumstances.[6] This problem is further confounded by the lack of consistent and reliable criteria for the diagnosis of NAI/SBS and because much of the traditional literature on child abuse consists of anecdotal case series, case reports, reviews, opinions, and position papers.[5,6,10,11,21,22] Many reports include cases having impact injury, which

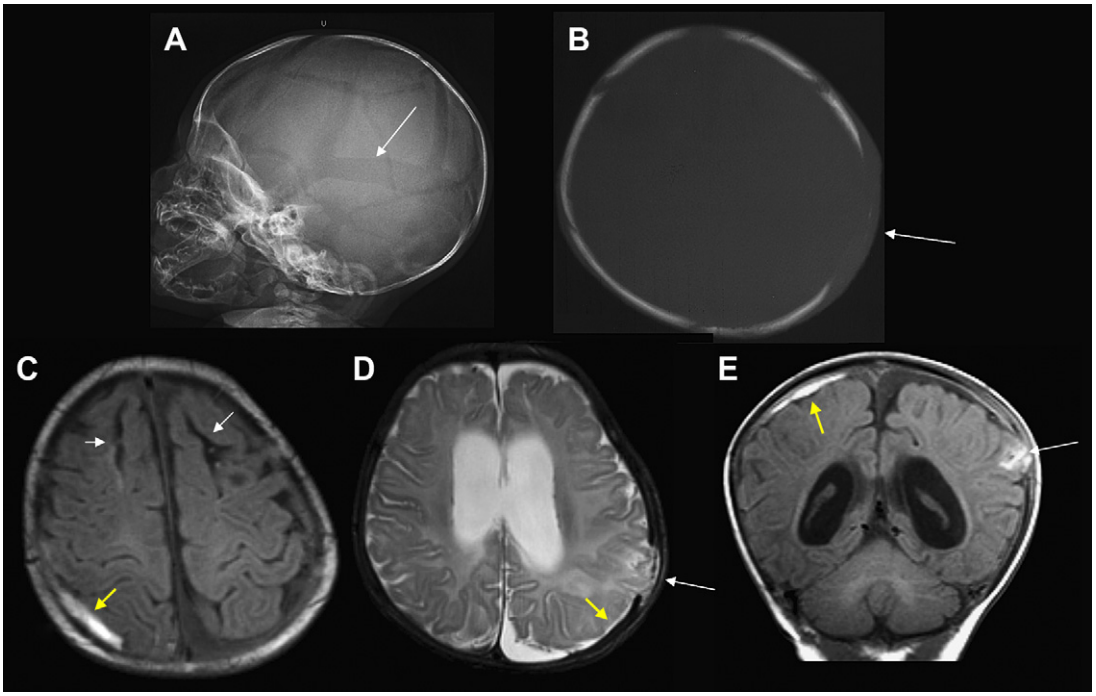

Fig. 1. Nine-week-old infant with triad and alleged NAI; also, history of traumatic labor and delivery. Skull film (*A*), CT (*B*) plus FLAIR (*C*), T2 (*D*), and T1 (*E*) MR imaging shows bilateral skull fractures with left growing fracture (*long white arrows*), chronic bifrontal cerebral white matter clefts (*short white arrows*) (*C*) plus acute, subacute, and chronic SDHs/rehemorrhages (*yellow arrows*).

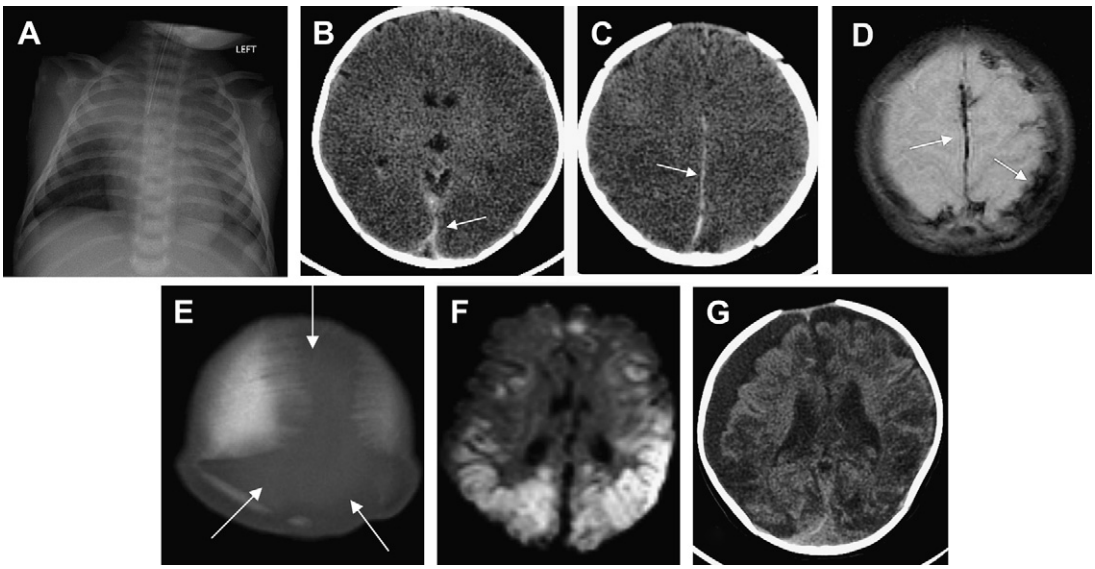

Fig. 2. Five-week-old infant with triad and alleged NAI; also, cold symptoms, vitamin D undersupplemented, acute choking episode during feeding, and status epilepticus. Chest film (*A*) shows bilateral lung opacities. CT (*B, C*) plus T2* MR imaging (*D*) shows bilateral cerebral edema with bilateral thin, acute-subacute hemorrhages (or thromboses) about the falx, tentorium, and convexities (*arrows*). Vertex CT (*E*) shows suture diastasis versus pseudodiastasis (*arrows*) (craniotabes?). DWI (*F*) shows global hypoxic-ischemic injury. Later CT (*G*) shows atrophy and chronic SDH.

undermines the SBS hypothesis by imposing a shaking-impact syndrome. Also, the inclusion criteria provided in many reports are criticized as arbitrary. Examples include suspected abuse, presumed abuse, likely abuse, and indeterminate.[21,22] Furthermore, the diagnostic criteria often seem to follow circular logic, such that the inclusion criteria (eg, the triad equals SBS/NAI) becomes the conclusion (ie, SBS/NAI equals the triad).

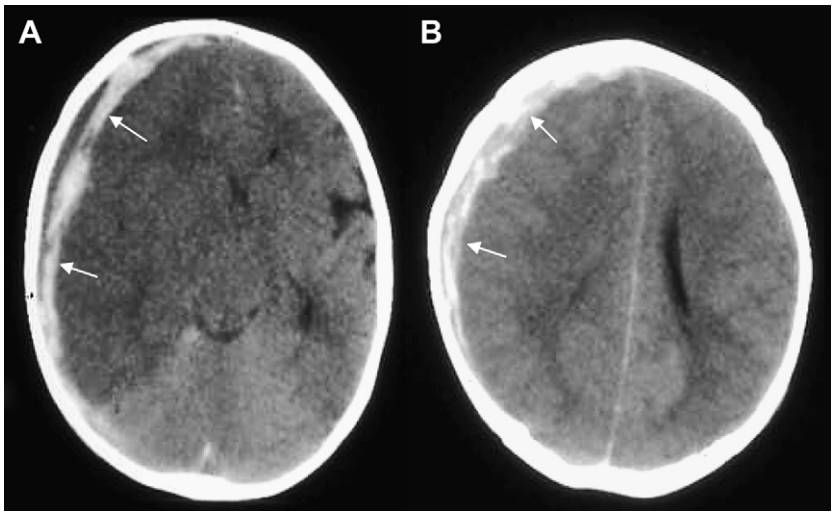

Fig. 3. Eight-month-old infant with triad and alleged NAI; also, right occipital skull fracture (age indeterminate; not shown) and 4- to 6-week-old wrist fracture. Hyperacute right SDH versus chronic SDH with rehemorrhage? CT (*A, B*) shows mixed high- plus low-density right extracerebral collection (*arrows*) with right cerebral edema, mass effect, and left shift. Question of subdural membrane on autopsy.

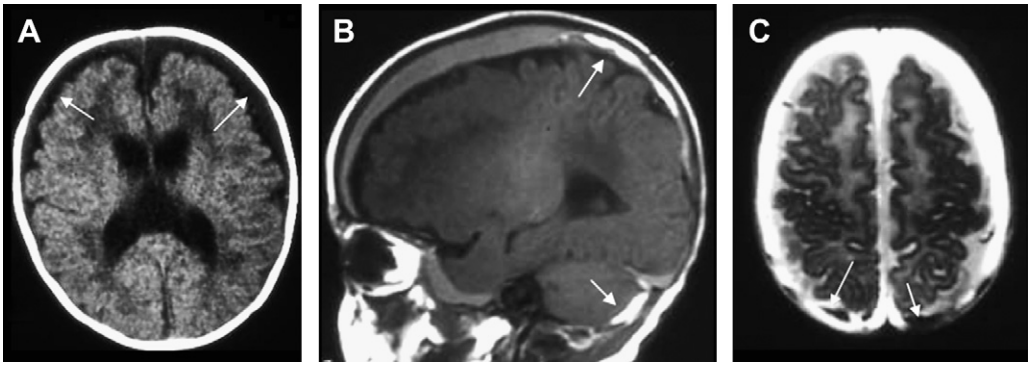

Fig. 4. Six-month-old infant with macrocephaly, the triad, and alleged NAI: BECC versus chronic SDH with rehemorrhage versus acute SDHG plus SDH? CT (*A*) shows bilateral frontal isohypodense extracerebral collections (*arrows*) with minute high densities (not shown). T1 MR imaging (*B*) shows smaller extracerebral high intensities (*arrows*) superimposed on larger isohypointensities. T2 MR imaging (*C*) shows small extracerebral T2 hypointensities (*arrows*) superimposed on large isohyperintensities.

RULES OF EVIDENCE AND EXPERT TESTIMONY

Regarding rules of evidence within the justice system, there are legal standards for the admissibility of expert testimony.[7,8,11,23] The Frye standard requires only that the testimony be generally accepted in the relevant scientific community. The Daubert standard requires assessment of the scientific reliability of the testimony. A criticism of the justice system is that the application of these standards varies with the jurisdiction (eg, according to state versus federal law). Additional legal standards regarding proof are also applied in order for the triar of fact (eg, judge or jury) to make the determination of civil liability or criminal guilt. In a civil action (eg, medical malpractice lawsuit), money is primarily at risk for the defendant health care provider, and proof of liability is based on a preponderance of the evidence (ie, at least 51% scientific or medical probability or certainty). In a criminal action, life or liberty is at stake for the defendant, including the permanent loss of child custody.[7,8,11,23,24] In such cases, the defendant has the constitutional protection of due process that requires a higher level of proof. This includes the principles of innocent until proved guilty beyond a reasonable doubt with the burden of proof on the prosecution and based on clear and convincing evidence. No percentage of level of certainty is provided, however, for these standards of proof in most jurisdictions. Furthermore, only a preponderance of the medical evidence (ie, minimum of 51% certainty) is required to support proof of guilt whether or not the medical expert testimony

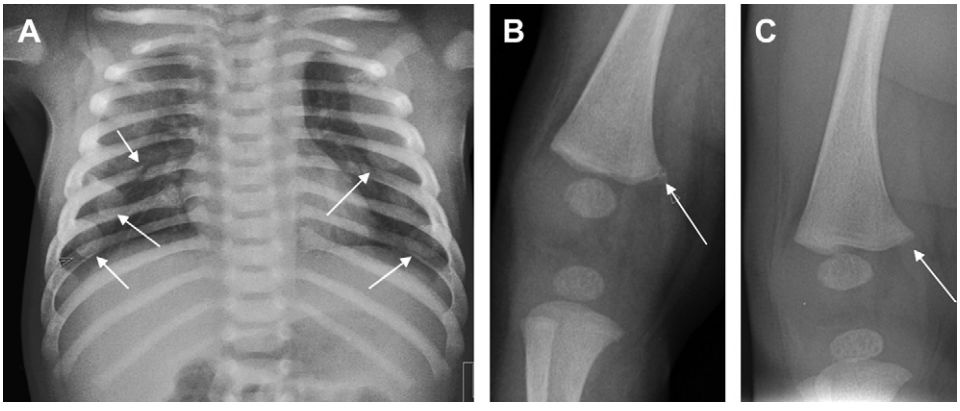

Fig. 5. Three-month-old infant with alleged NAI; also, history consistent with congenital rickets. Chest film (*A*) shows bilateral recent and old, healing rib fractures (pseudofractures? rachitic rosary? [*arrows*]). Knee films before (*B*) and after (*C*) vitamin D supplementation show healing classic metaphyseal lesions (*arrows*)?

complies with the Frye standard (ie, general acceptance requirement) or the Daubert standard (ie, scientific reliability requirement). Further criticism of the criminal justice process is that in NAI cases, medical experts have defined SBS/NAI as "the presence of injury (eg, the triad) without a sufficient historical explanation" and that this definition unduly shifts the burden to the defendant to establish innocence by proving the expert theory wrong.

THE MEDICAL PROSECUTION OF NAI AND ITS EBM CHALLENGES

Traditionally, the prosecution of NAI has been based on the presence of one or more aspects of the triad as supported by the premises that (1) shaking alone in an otherwise healthy child can cause SDH leading to death, (2) such injury can never occur on an accidental basis (eg, short-distance fall) because it requires a massive violent force equivalent to a motor vehicle accident or a fall from a multistory building, (3) such injury is immediately symptomatic and cannot be followed by a lucid interval, and (4) changing symptoms in a child with prior head injury indicates newly inflicted injury and not a spontaneous rebleed.[1–4,7,8,11] Using this reasoning, the last caretaker is automatically guilty of inflicted injury, especially if not witnessed by an independent observer. Also, it has been asserted that RHs of a particular pattern are diagnostic of SBS/NAI.

Reports from clinical, biomechanical, pathology, forensic, and legal disciplines, within and outside of the child maltreatment literature, have challenged the evidence base for NAI/SBS as the only cause for the triad.[5–12] Such reports indicate that the triad may also be seen with AI (including witnessed short-distance falls, lucid intervals, and rehemorrhage) (**Figs. 6** and **7**) as well as in medical conditions. These are the mimics of NAI and often present as acute life-threatening events (ALTEs).[25,26] The medical mimics include hypoxia-ischemia (eg, apnea, choking, or respiratory or cardiac arrest) (see **Figs. 2, 6,** and **7**), ischemic injury (eg, arterial versus venous occlusive disease) (**Fig. 8**), vascular anomalies (eg, arteriovenous malformation [AVM]) (**Fig. 9**), seizures (see **Fig. 2**), infectious or postinfectious conditions (**Fig. 10**), coagulopathies (**Fig. 11**), fluid-electrolyte derangement, and metabolic or connective tissue disorders, including vitamin deficiencies and depletions (eg, C, D, or K) (see **Figs. 1** and **5**; **Fig. 12**).[2,4]

Many ALTEs seem multifactorial and involve a combination, sequence, or cascade of predisposing and complicating events or conditions.[4,25] As an example, an infant may suffer a head impact, or choking spell, followed by seizures or apnea, and then undergo a series of interventions, including prolonged or difficult resuscitation and problematic airway management with subsequent hypoxia-ischemia and coagulopathy (see **Figs. 2, 6, 7,** and **11**). Another example is a young infant with a predisposing condition, such as infectious illness, fluid-electrolyte imbalance, metabolic disorder, or a coagulopathy, who then suffers seizures, respiratory arrest, and resuscitation with hypoxia-ischemia (see **Figs. 10–12; Fig. 13**). In many cases of alleged SBS/NAI, it is often assumed that nonspecific premorbid symptoms (eg, irritability, lethargy, and poor feeding) in an otherwise healthy infant are indicators of ongoing abuse or that such symptoms become the inciting factor for the abuse. A thorough and complete medical investigation in such cases may reveal that the child is not otherwise healthy and is suffering from a medical condition that progresses to an ALTE.[2,4,25]

BIOMECHANICAL CHALLENGES

The mechanical basis for SBS as hypothesized by Guthkelch, Caffey, and other investigators,[27] was originally extrapolated from Ommaya,[28] who used an animal whiplash model to determine the angular acceleration threshold (ie, 40 g) for head injury (ie, concussion, SDH, and shear injury). It was assumed that manual shaking of an infant could generate these same forces and produce the triad. Duhaime and colleagues[29] measured the angular accelerations associated with adult manual shaking (ie, 11 g) and impact (ie, 52 g) in a 1-month-old infant anthropormorphic test device (ATD). Only accelerations associated with impact (4 to 5 times that associated with shakes) on an unpadded or padded surface exceeded the injury thresholds determined by Ommaya. In the same study, the Duhaime and colleagues reported a series of 13 fatal cases of NAI/SBS in which all had evidence of blunt head impact (more than half noted only at autopsy).[29] The investigators concluded that CNS injury in SBS/NAI in its most severe form is usually not caused by shaking alone. Their results contradicted many of the original reports that had relied on the whiplash mechanism as causative of the triad. They suggested the use of the new term, shaken-impact syndrome. More recently, Prange and colleagues,[30] using a 1.5 month-old ATD, showed that inflicted impacts against hard surfaces were more likely associated with brain injury than falls from less than 1.5 m or from vigorous shaking. With further improvements in ATDs, more recent experiments indicate that maximum head

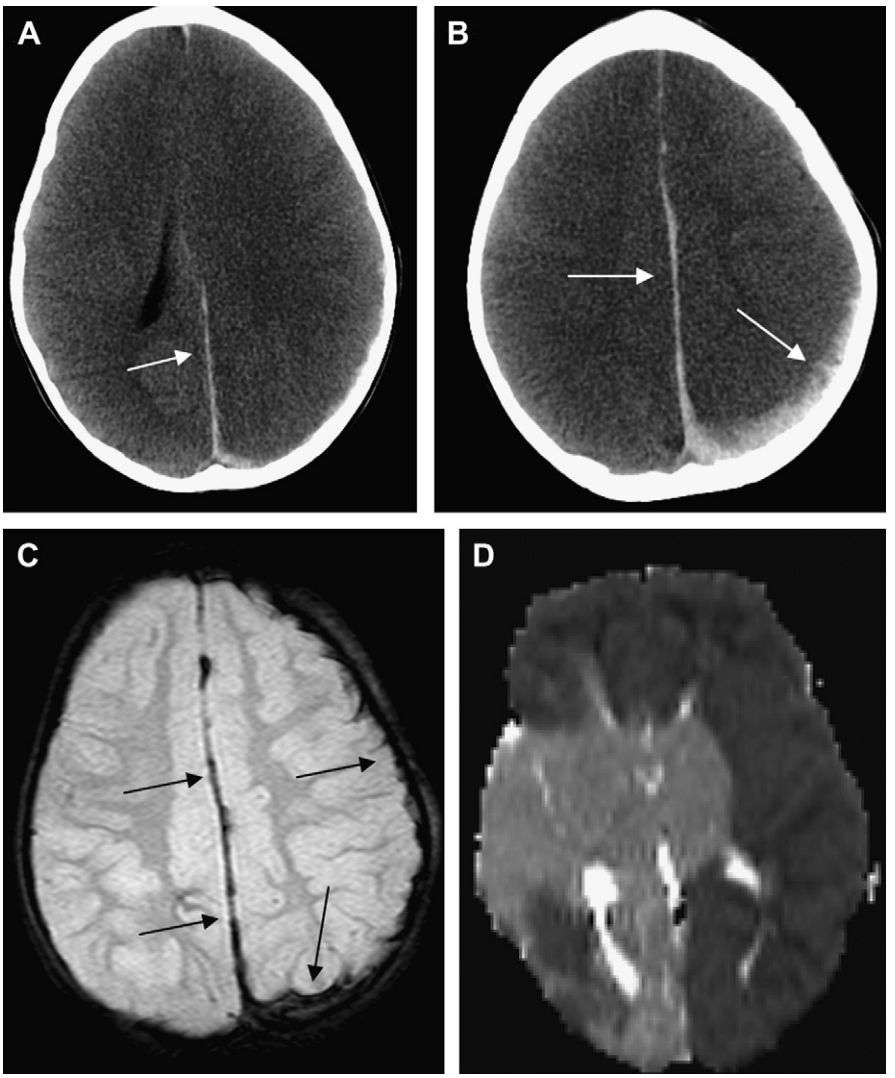

Fig. 6. Twenty-one–month–old toddler with triad and alleged NAI; also, history of prior head impact. Question prior injury with lucid interval versus hyperacute injury. CT (*A, B*) acute left convexity and interhemispheric SDH and SAH (*arrows*) with cerebral swelling, left more than right. T2* MR imaging (*C*) shows low intensity SDH (*arrows*) with T1/T2 isointensity (not shown). ADC map (*D*) shows asymmetric cerebral restricted diffusion (left > right). Autopsy confirms impact with acute SDH, SAH, and hypoxic-ischemic injury.

accelerations may exceed injury reference values at lower fall heights than previously determined (**Fig. 14**).[31] Critics of the Duhaime and Prange studies contend that there is no adequate human infant surrogate yet designed to properly test shaking versus impact.[32] Other reports also show that shaking alone cannot result in brain injury (ie, the triad) unless there is concomitant injury to the neck, cervical spinal column, or cervical spinal cord, because these are the weak links between the head and body of the infant.[33–35] Spinal cord injury without radiographic abnormality (SCIWORA), whether or not AI or NAI, is an important example of primary neck and spinal cord injury with secondary brain injury (see **Fig. 7**).[35] For example, a falling infant experiences a head-first impact with subsequent neck hyperextension (or hyperflexion) from the force of the trailing body mass. There is resultant upper spinal cord injury without detectable spinal column injury on plain films or CT. Compromise of the respiratory center at the cervicomedullary junction results in hypoxic brain injury, including the thin SDH (see **Fig. 7**). CT often shows the brain injury, but only MR imaging may show the additional neck or spinal cord injury.

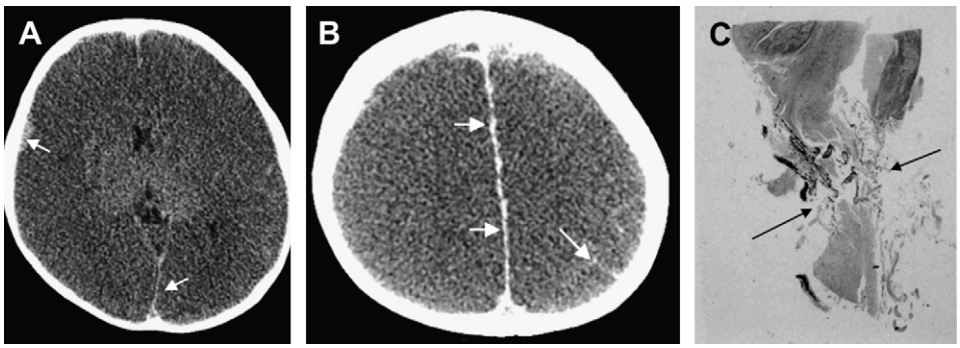

Fig. 7. Twenty-one–month–old with triad and alleged NAI; also, history of 4-ft fall. CT (*A, B*) with high-density SAH and thin SDH (*arrows*) plus cerebral edema. Sagittal plane photomicrograph (*C*) from autopsy shows upper cervical spinal cord disruption (*arrows*) resulting in global hypoxic-ischemic injury.

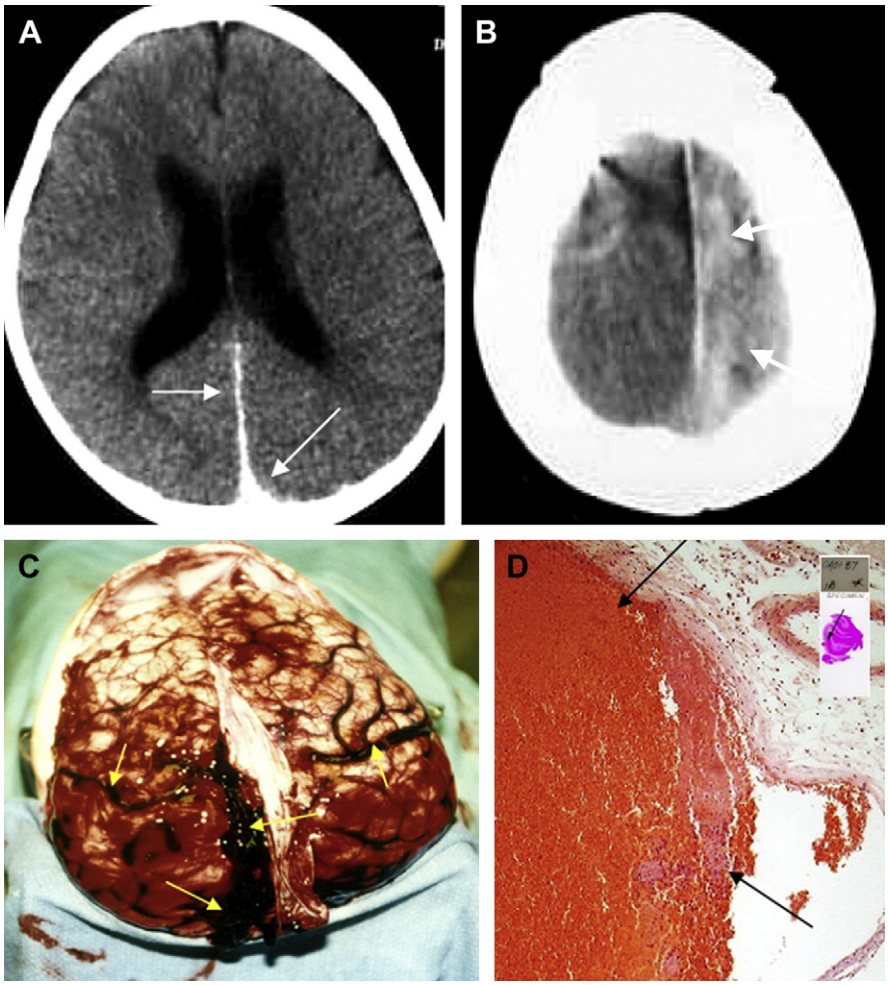

Fig. 8. Fourteen-month-old infant with triad and alleged NAI; also, recent infectious illness: dural and cortical venous sinus thrombosis with dural hemorrhage: CT (*A, B*) shows high densities along the falx and dural venous sinuses (*white arrows*). (*C*) Gross specimen—reflected superior sagittal sinus and cortical venous thromboses with distended veins (*yellow arrows*); (*D*) photomicrograph of cortical venous thrombus with inflammatory reaction (*black arrows*) plus SDH with neomembrane (7–14 days old; not shown). (*Pathology courtesy of* J. Leestma, MD.)

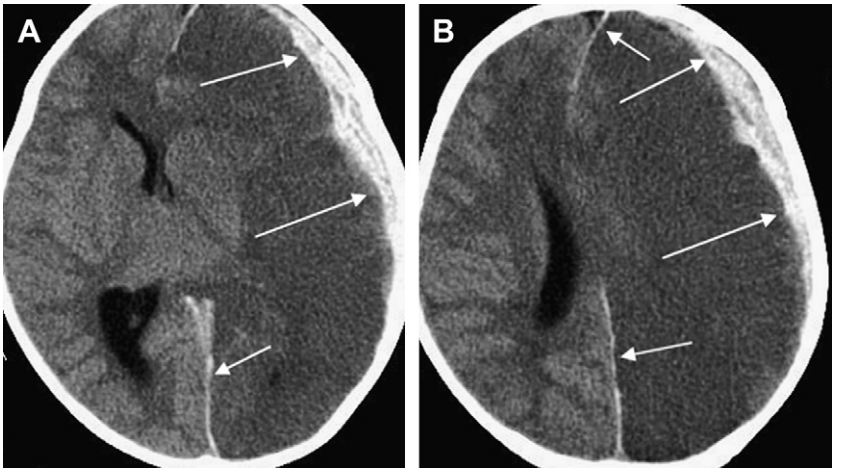

Fig. 9. Twenty-month-old infant with triad and alleged NAI. Left SDH with cerebral cortical and pial AVM at autopsy. CT (*A, B*) shows left mixed-density SDH and SAH (*long arrows*) plus interhemispheric hemorrhage (*short arrows*) with marked left cerebral swelling and shift.

The minimal force required to produce the triad has yet to be established. From the current biomechanical evidence base, however, it can be concluded that (1) shaking may not produce direct brain injury but may cause indirect brain injury if associated with neck and cervical spinal cord injury; (2) angular acceleration/deceleration injury forces clearly occur with impact trauma; (3) such injury on an accidental basis does not require a force that can only be associated with a motor vehicle accident or a multistory fall; (4) household (ie, short-distance) falls may produce direct or indirect brain injury; (5) in addition to fall height, impact surface and type of landing are important factors; and (6) head-first impacts in young infants not having developed a defensive reflex (eg, extension of a limb to break the fall) are the most dangerous and may result in direct or indirect brain injury (eg, SCIWORA).

NEUROPATHOLOGY CHALLENGES

In their landmark neuropathology study of 53 victims of alleged SBS/NAI,[36,37] Geddes and colleagues showed in 37 infants (ages <9 months) that (1) 29 had evidence of impact with only one case of admitted shaking; (2) cerebral swelling was more often due to DAI of hypoxic-ischemic encephalopathy (HIE) rather than shear or traumatic axonal injury (TAI); (2) although fracture, thin SDH (eg, dural vascular plexus origin), and RH are commonly present, the usual cause of

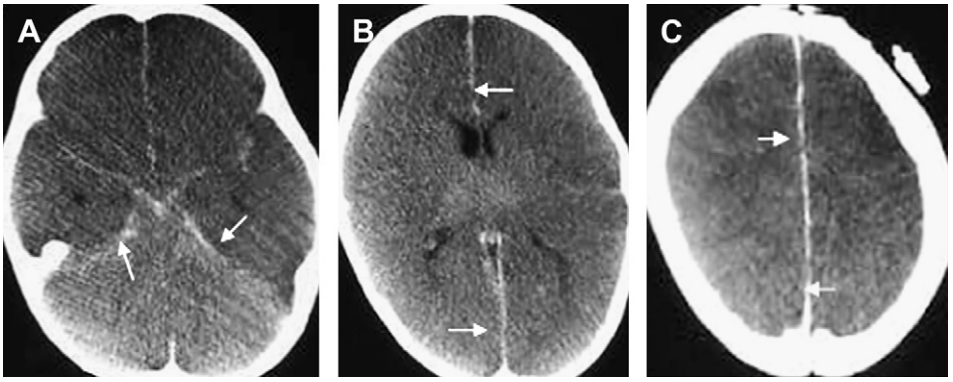

Fig. 10. Twenty-one–month–old infant with triad and alleged NAI. Pneumococcal meningitis, herniation, and hypoxic-ischemic injury confirmed at autopsy. CT (*A–C*) shows high-density thin SDH (*arrows*) plus cerebral edema.

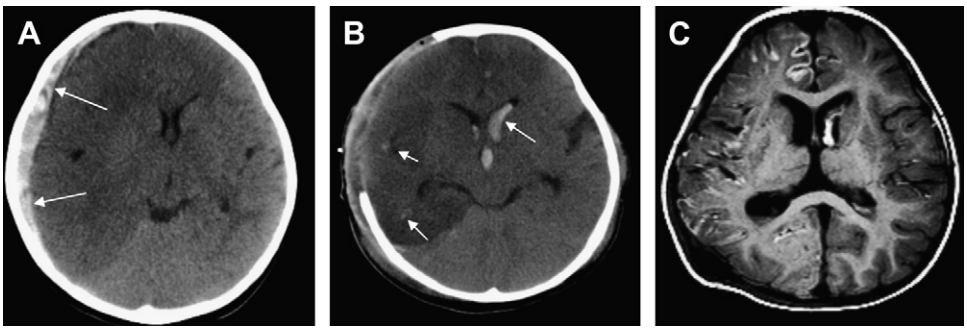

Fig. 11. Nine-month-old girl with triad and alleged NAI; also, recent fall and coagulopathy (later confirmed platelet disorder). Initial CT (*A*) shows mixed-density right SDH (*arrows*) with right cerebral edema. Postoperative CT 5 days later (*B*) shows other cerebral and intraventricular hemorrhages (*arrows*). T1 MR imaging (*C*) 11 days postoperatively shows evolving right cerebral high-intensity cortical injury and hemorrhages.

death was increased intracranial pressure from brain swelling associated with HIE (see **Fig. 2**); and (4) cervical epidural hemorrhage and focal axonal brainstem, cervical cord, and spinal nerve root injuries were characteristically seen in these infants (most with impact). Upper cervical cord/brainstem injury may result in apnea/respiratory arrest and be responsible for the HIE. In the 16 older victims (ages 13 months to 8 years), the pathology findings were primarily those of the battered child or adult trauma syndrome, including extracranial injuries (eg, abdominal), large SDH (ie, bridging vein rupture), and TAI. Additional neuropathology series by Geddes and colleagues[38] have shown that SDHs are also seen in nontraumatic fetal, neonatal, and infant brain injury cases and that such SDHs are actually of intradural vascular plexus origin rather than bridging cortical vein origin.

The common denominator in all these cases is likely a combination of vascular immaturity and fragility further compromised by HIE or infection, cerebral venous hypertension or congestion, arterial hypertension, and brain swelling (see **Fig. 2**). Although the unified hypothesis of Geddes and colleagues[13,14,39] has received criticism, their findings and conclusions have been validated by the research of Cohen and Scheimberg,[40] Croft and Reichard,[41] and others. In their postmortem series, Cohen and colleagues described 25 fetuses (26–41 weeks) and 30 neonates (1 hour–19 days) with HIE who also had macroscopic intradural hemorrhage (IDH), including frank parietal SDH in two-thirds. The IDH was most prominent along the posterior falcine and tentorial vascular plexuses (ie, interhemispheric fissure) (see **Fig. 2**). They concluded from their work, along with the findings of other cited

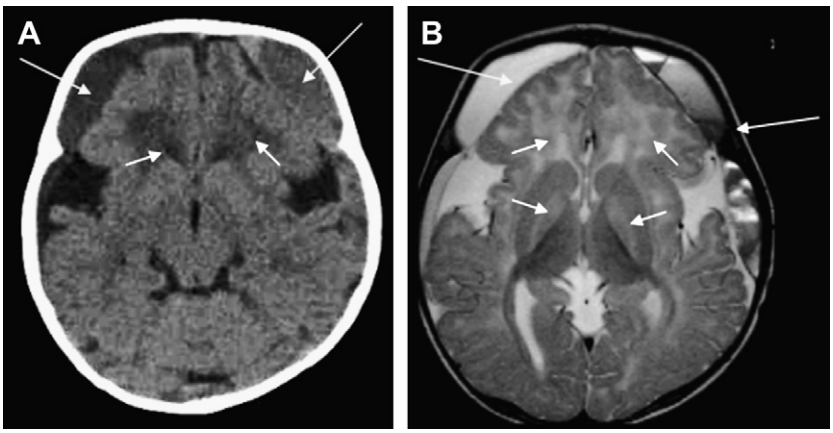

Fig. 12. Twelve-month-old infant with triad and alleged NAI. Glutaric acidopathy type 1. CT (*A*) and T2 MR imaging (*B*) shows bilateral SDH of varying age (*long arrows*), wide sylvian fissures plus basal ganglia, and cerebral white matter abnormalities (*short arrows*).

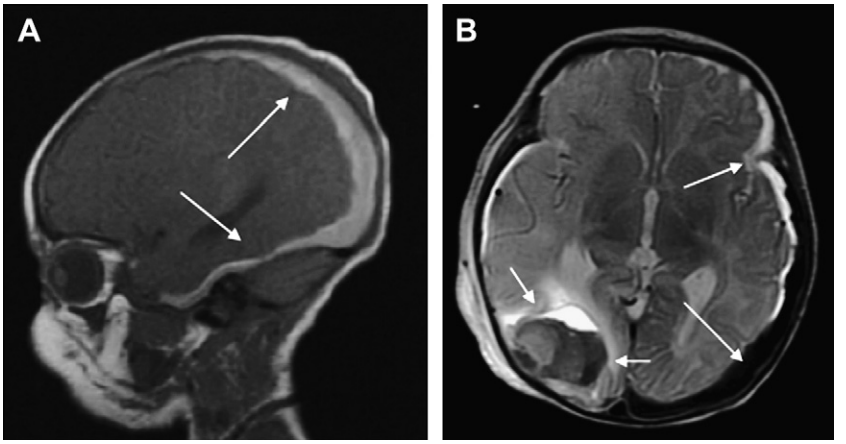

Fig. 13. Home-delivered newborn with seizures at 1 week of age; also, no vitamin K given at birth. T1 (*A*) and T2 (*B*) MR imaging shows acute-subacute left SDH (*long arrows*) plus right cerebral hemorrhage (*short arrows*); vitamin K deficiency confirmed and treated.

researchers, that IDH and SDH are commonly associated with HIE, particularly when associated with increases in central venous pressure. This also explains the frequency of RH associated with perinatal events.[42]

From the current forensic pathology evidence base, it may be concluded that (1) shaking may not cause direct brain injury but may cause indirect brain injury (ie, HIE) if associated with cervical spinal cord injury; (2) impact may produce direct or indirect brain injury (eg, SCIWORA); (3) the pattern of brain edema with thin SDH (dural vascular plexus origin) may reflect HIE whether or not due to AI or NAI; and (4) the same pattern of injury may result from nontraumatic or medical causes (eg, HIE from any cause of ALTE). Furthermore, because the observed edema does not represent TAI (which results in immediate neurologic dysfunction), a lucid interval is possible, particularly in infants whose sutured skull and

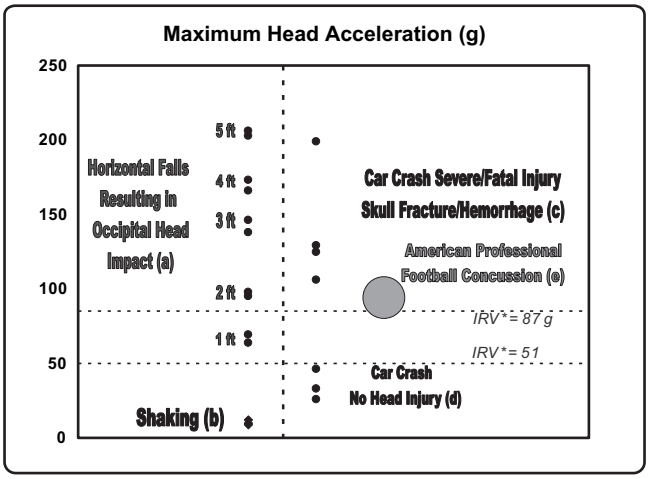

Fig. 14. Maximum head accelerations versus trauma mechanisms as correlated with injury thresholds. CRABI, child restraint air bag interaction; IRV, injury reference values. (*Data from* Van Ee C, PhD. Design research engineering. Available at: www.dreng.com. Accessed September 12, 2010; Leestma J. Forensic neuropathology. 2nd edition. Boca Raton [FL]: CRC Press; 2009; Mertz H. Anthropomorphic test devices. In: Melvin J, Nahum A, editors. Accidental injury: biomechanics and prevention. 2nd edition. New York: Springer; 2002. p. 84; Klinich JD, Hulbert G, Schneider LW. Estimating infant head injury criteria and impact response using crash reconstruction and finite element modeling. Society of Automotive Engineers Paper # 2002–22–0009, 2002; CRABI 12 [a, b]; CRABI 6 [c, d]; and [e] Pellman EJ, Viano DC, Tucker AM, et al. Concussion in professional football: reconstruction of game impacts and injuries. Neurosurgery 2003;53[4]:799–812.)

dural vascular plexus have the distensibility to tolerate early increases in intracranial pressure. Also, the lucid interval invalidates the premise that the last caretaker is always responsible in alleged NAI.

CLINICAL CHALLENGES

In the prosecution of NAI, it is often stipulated that short-distance falls cannot be associated with the triad, serious (eg, fatal) head injury, or a lucid interval. Traditionally, it has also been stipulated that nonintentional new bleeding in an existing SDH is always minor, that SDH does not occur in benign extracerebral collections (BECCs), and that symptomatic or fatal new bleeding in SDH requires newly inflicted trauma.[1–4,7,8,11] Several past and current reports refute the significance of low level falls in children, including in-hospital and outpatient clinic series.[43–51] There are other reports, however, including emergency medicine, trauma center, neurosurgical, and medical examiner series, that indicate a heightened need for concern regarding the potential for serious intracranial injury associated with minor or trivial trauma scenarios, particularly in infants.[52–74] This includes reports of skull fracture or acute SDH from accidental simple falls in infants, SDH in infants with predisposing wide extracerebral spaces (eg, BECCs of infancy, chronic subdural hygromas, arachnoid cyst, and so forth) (see **Fig. 4; Figs. 15** and **16**), and fatal pediatric head injuries due to witnessed, accidental short-distance falls, including those with a lucid interval, SDH, RH, and malignant cerebral edema (see **Fig. 6**). Also included are infants with chronic SDH from prior trauma (eg, at birth) who then develop rehemorrhage (see **Figs. 1, 4,** and **15**).

Short-Distance Falls, Lucid Intervals, and Malignant Edema

Hall and colleagues[44] reported that 41% of childhood deaths (mean age 2.4 years) from head injuries associated with AI were from low level falls (3 feet or less) while running or down stairs. Chadwick and colleagues[45] reported fatal falls of less than 4 feet in seven infants but considered the histories unreliable. Plunkett[56] reported witnessed fatal falls of 2 to 10 feet in 18 infants and children, including those with SDH, RH, and lucid intervals. Greenes and Schutzman[57] reported intracranial injuries, including SDH, in 18 asymptomatic infants with falls of 2 feet to 9 stairs. Christian and colleagues[63] reported three infants with unilateral RH and SDH/SAH due to witnessed accidental household trauma. Denton and Mileusnic[59] reported a witnessed, accidental 30-inch fall in a 9-month-old infant with a 3-day lucid interval before death. Murray and colleagues[60] reported more intracranial injuries in young children (49% <age 4 y; 21% <age 1 y) with reported low level falls (<15 ft), both AI and NAI. Kim and colleagues[61] reported a high incidence of intracranial injury in children (ages 3 mo to 15 y; 52% <age 2 y) accidentally falling from low heights (3 to 15 ft; 80% <6 ft; including 4 deaths). Because of the lucid intervals in some patients, including initially favorable Glascow Coma Scale scores (GCS) with subsequent deterioration, Murray and colleagues[60] and others expressed concern regarding caretaker delays and medical transfer delays contributing to the morbidity and mortality in these patients.[53–56,58–61] Bruce and colleagues[54,55] reported one of the largest pediatric series of head trauma (63 patients, ages 6 months to 18 years), both AI and NAI, associated with malignant brain edema and SAH/SDH (see **Fig. 6**). In the higher GCS (>8) subgroup,

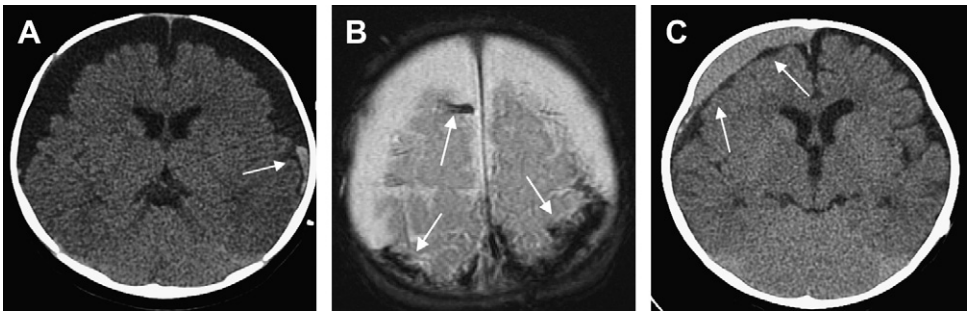

Fig. 15. Five-month-old infant with the triad and alleged NAI; also, macrocephaly from birth, recent seizure but no trauma. CT (*A*) and T2* MR imaging (*B*) shows large extracerebral collections with smaller recent hemorrhages (*arrows*). CT 3 months postdrainage (*C*) shows rehemorrhage (*arrows*). Diagnosis: BECC or chronic SDHG with rehemorrhage?

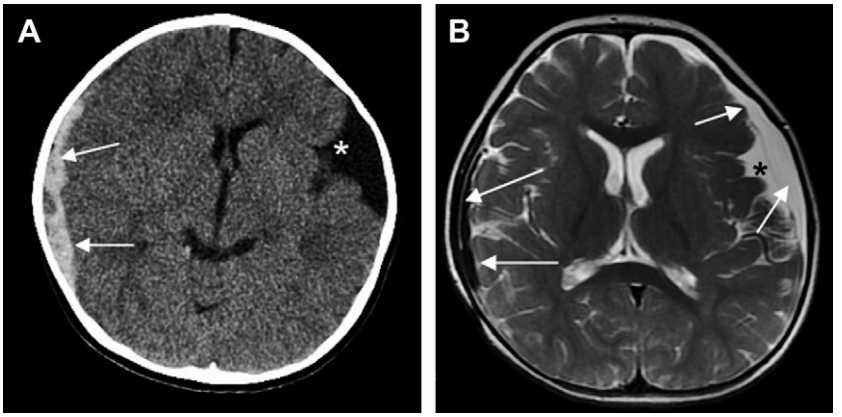

Fig. 16. Sixteen-month-old with triad (right RH) and alleged NAI; also, short-distance fall with right scalp impact. CT (*A*) shows left sylvian arachnoid cyst (*) and right hyperacute SDH (*arrows*). T2 MR imaging (*B*) 2 days later shows acute right SDH (*long arrows*) and smaller left sylvian arachnoid cyst (*) with subdural hygroma (*short arrows*).

there were 8 with a lucid interval and all 14 had complete recovery. In the lower GCS ($\leq$ 8) subgroup, there were 34 with immediate and continuous coma, 15 with a lucid interval, 6 deaths, and 11 with moderate to severe disability. More recently, Steinbok and colleagues[62] reported 5 children (4 <age 2 y; 3 falls) with witnessed AI, including SDH and cerebral edema detected by CT 1 to 5 hours post event. All experienced immediate coma with rapid progression to death (see **Fig. 6**).

Benign Extracerebral Collections

BECCs of infancy (also known as benign external hydrocephalus or benign extracerebral subarachnoid spaces) is a common and well-known condition characterized by diffuse enlargement of the subarachnoid spaces.[65–74] A transient disorder of cerebrospinal fluid (CSF) circulation, probably due to delayed development of the arachnoid granulations, is widely accepted as the cause and develops from birth. BECC is typically associated with macrocephaly but may also occur in infants with normal or small head circumferences, including premature infants. As with any cause of craniocerebral disproportion (eg, BECC, hydrocephalus, chronic SDH or hygroma, arachnoid cyst, or underdevelopment or atrophy), there is a susceptibility to SDH that may be spontaneous or associated with trivial trauma (see **Figs. 4** and **15**). A recent large series report and review by Hellbusch[73] emphasizes the importance of this predisposition and cites other confirmatory series and case reports (30 references). Papasian and Frim[68] designed a theoretic model that predicts the predisposition of benign external hydrocephalus to SDH with minor head trauma. Piatt's[66] case report of BECC with SDH (27 references), including RH, along with McNeely and colleagues'[72] case series are further warnings that this combination is far from specific for SBS/NAI.

Birth Issues

In addition to the examples discussed previously (eg, short-distance falls and BECCs), another important but often overlooked factor is birth-related trauma.[1,4,75–89] This includes normal as well as complicated labor and delivery events (pitocin augmentation, prolonged labor, vaginal delivery, instrumented delivery, cesarean section, and so forth). It is well known that acute SDH often occurs even with the normal birth process and that this predisposes to chronic SDH, including in the presence of BECC (see **Figs. 1, 4,** and **15**). Intracranial hemorrhages, including SDH and RH, have been reported in several CT and MR imaging series of normal neonates including a frequency of 50% by Holden and colleagues,[81] 8% by Whitby and colleagues,[76] 26% by Looney and colleagues,[82] and 46% by Rooks and colleagues.[78] Chamnanvanakij and colleagues[75] reported 26 symptomatic term neonates with SDH over a 3-year period after uncomplicated deliveries. Long-term follow-up imaging has not been provided in many of these series, although Rooks and colleagues[78] reported one child in their series who developed SDH with rehemorrhage superimposed on BECC (**Fig. 17**).

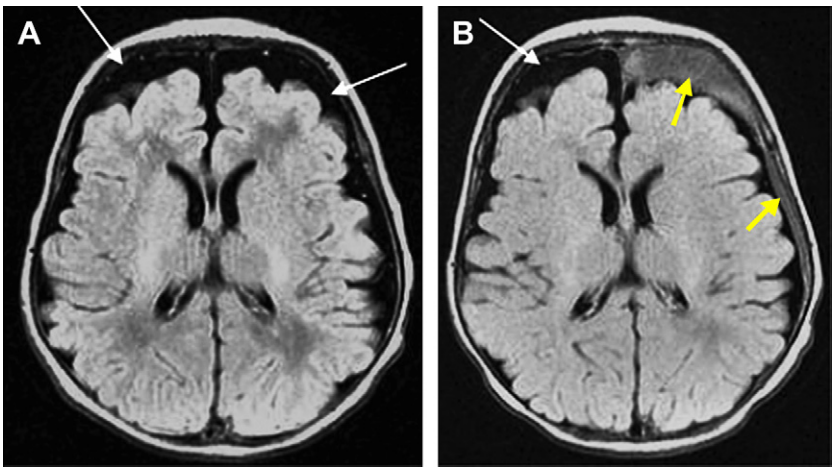

Fig. 17. BECC versus SDHG at birth (*A*) (*long arrows*) with SDH versus rehemorrhage 1 month later (*B*) (*yellow arrows*) on axial FLAIR MR images. (*Courtesy of* Veronica J. Rooks, MD, Tripler Army Medical Center, Honolulu, HI.)

Chronic SDH and Rehemorrhage

Chronic SDH is one of the most controversial topics in the NAI versus AI debate.[1–4,12,21,22,36–41] Unexplained SDH is often ascribed to NAI. By definition, a newly discovered chronic SDH started as an acute SDH that, for whatever reason, may have been subclinical. There is likely more than one mechanism for SDH that has prompted a revisiting of the concept of the subdural compartment.[12,40,41,90,91] Mack and colleagues[90] have provided an updated review on this important topic. In some cases of infant trauma, dissection at the relatively weak dura-arachnoid border zone (ie, dural border cell layer) may allow CSF to collect and enlarge over time as a dural interstitial (ie, intradural) hygroma. In other cases, there is bridging vein rupture within the dural interstitium that results in an acute subdural or intradural hematoma that extends along the dural border cell layer. Furthermore, traumatic disruption of the dural vascular plexus (ie, venous, capillary, or lymphatic), which is particularly prominent in young infants, may also produce an acute intradural hematoma. Some of these collections undergo resorption whereas others progress to become chronic SDH. Some progressive collections may represent mixed CSF-blood collections (see **Figs 1, 4,** and **15**).

The pathology and pathophysiology of neomembrane formation in chronic SDH, including rebleeding, is well established in adults and seems similar, if not identical, to that in infants.[83,92–112] Although acute SDH is most often due to impact or deformational trauma, whether or not AI or NAI, it must be differentiated from chronic SDH with rehemorrhage. Progression of chronic SDH and rehemorrhage is likely related to capillary leakage and intrinsic thrombolysis.[92,93] Other factors include dural vascular plexus hemorrhage associated with increases in intracranial or central venous pressures (eg, birth trauma, congenital heart disease, venous thrombosis, or dysphagic choking) or with increased meningeal arterial pressure (eg, reperfusion after hypoxia-ischemia) with resultant acute hemorrhage (or rehemorrhage) in normal infants or superimposed on predisposing chronic BECC, hygromas, hematomas, or arachnoid cysts (see **Figs. 1, 2, 4**, and **15–17**).[12,38,40,65–74,90,91] The phenomenon of acute infantile SDH, whether or not AI or NAI, evolving to chronic SDH and rehemorrhage, including RH, is well documented in several neurosurgical series reports, including those by Aoki and colleagues,[97,98] Ikeda and colleagues,[99] Parent,[94] Howard and colleagues,[102] Hwang and Kim,[95] Vinchon,[103,104] and others.

Conclusions

From the clinical evidence base, in addition to the biomechanical and neuropathology evidence bases, it may be concluded that (1) significant head injury, including SDH and RH, may result from low fall levels; (2) such injury may be associated with a lucid interval; (3) in some, the injury may result in immediate deterioration with progression to death; (4) BECC predisposes to SDH; (5) SDH may date back to birth; and (6) rehemorrhage into an existing SDH occurs in childhood and may be serious.

RH CHALLENGES

Many guidelines for diagnosing NAI depend on the presence of RH, including those of a particular pattern (eg, retinal schisis, and perimacular folds) and based on the theory of vitreous traction due to inflicted acceleration/deceleration forces (eg, SBS).[1–4,113–132] The specificity of RH for NAI has been repeatedly challenged, however. Plunkett[56] reported RH in two-thirds of eye examinations in children with fatal AI. Goldsmith and Plunkett[132] reported a child with extensive bilateral RH in a videotaped fatal accidental short-distance fall. Lantz and colleagues[122] reported RH with perimacular folds in an infant crush injury. Gilles and colleagues[120] reported the appearance and progression of RH with increasing intracranial pressure after head injury in children. Obi and Watts[125] reported RH with schisis and folds in two children, one with AI and the other with NAI. Forbes and colleagues[126] reported RH with epidural hematoma in five infant AI cases. From a research perspective, Brown and colleagues[128] found no eye pathology in their fatal shaken animal observations. Binenbaum and colleagues[127] observed no eye abnormalities in piglets subjected to acceleration/deceleration levels greater than 20 times what Prange and colleagues[30] predicted possible in inflicted injury. Emerson and colleagues[129] found no support for the vitreous traction hypothesis as unique to NAI. The eye and optic nerve are an extension of, and therefore a window to, the CNS, including their shared vascularization, meningeal coverings, innervation, and CSF spaces. RH has been reported with a variety of conditions, including AI, resuscitation, increased intracranial pressure, increased venous pressure, subarachnoid hemorrhage, sepsis, coagulopathy, certain metabolic dis,orders, systemic hypertension, and other conditions.[121,123,131] The common pathophysiology seems to be increased intracranial pressure or increased intravascular pressure. Furthermore, many cases of RH (and SDH) are confounded by the sequence or cascade of multiple conditions (eg, the unified hypothesis of Geddes) that often has a synergistic influence on the type and extent of RH. For example, consider the common situation of a child who has had trauma (factual or assumed) followed by seizures, apnea, or respiratory arrest and resuscitation with resultant HIE or coagulopathy. In much of the traditional NAI/SBS literature, little if any consideration has been given to any predisposing or complicating factors, and often there is no indication of the timing of the eye examinations relative to the clinical course or the brain imaging.[113,114,119,130]

From the research and clinical evidence base, it may be concluded that (1) RH is not specific for NAI, (2) RH may occur in AI and medical conditions, and (3) predisposing factors and complicating cascade effects must be considered in the pathophysiology of RH.

MEDICAL CONDITIONS MIMICKING NAI

A significant part of the controversy is the medical conditions that may mimic the clinical presentations (ie, the triad) and imaging findings of NAI.[1,2,4,25,26,89,101] Furthermore, such conditions may predispose to or complicate AI or NAI, as part of a cascade that results in or exaggerates the triad. In some situations, it may be difficult or impossible to tell which of these elements are causative and which are the effects. These include HIE, seizures, dysphagic choking ALTE, cardiopulmonary resuscitation, infectious or postinfectious conditions (eg, sepsis, meningoencephalitis, or postvaccinial), vascular diseases, coagulopathies, venous thrombosis, metabolic disorders, neoplastic processes, certain therapies, extracorporeal membrane oxygenation, and other conditions.[4,25,89,101] Regarding pathogenesis of the triad (with or without other organ system involement [eg, skeletal]) and whether or not due to NAI, AI, or medical etiologies, the pathophysiology seems to be a combination or sequence of factors, including increased intracranial pressure, increased venous pressure, systemic hypotension or hypertension, vascular fragility, hematologic derangement, and/or a collagenopathy imposed on the immature CNS, including the vulnerable dural vascular plexus as well as other organ systems.[4,12,25,38,90] Although the initial medical evaluation, including history, laboratory tests, and imaging studies, may suggest an alternative condition, the diagnosis may not be made because of a rush to judgment regarding NAI.[4–11] Such bias may have devastating effects on an injured child and family. It is important to be aware of these mimics, because a more extensive work-up may be needed beyond routine screening tests. Also, lack of confirmation of a specific condition does not automatically indicate the default diagnosis of NAI. In all cases, it is critical to review all past records dating back to the pregnancy and birth as well as the postnatal pediatric records, family history, more recent history preceding the acute presentation, details of the acute event itself, resuscitation, and the subsequent management, all of which may contribute to the clinical and imaging findings. An incomplete medical evaluation may result in unnecessary cost shifting to

child protection and criminal justice systems and have further adverse effects regarding transplantation organ donation in brain death cases and custody/adoptive dispositions for the surviving child and siblings.

Sirotnak's[89] recent review, along with others', extensively catalogs the many conditions that may mimic NAI[4,25,101]:

Birth Trauma and Neonatal Conditions

Manifestations of birth trauma, including fracture, SDH, and RH, may persist beyond the neonatal period. Other examples are the sequelae of extracorporeal membrane oxygenation therapy, at-risk prematurity, and congenital heart disease. When evaluating a young infant with apparent NAI, it is important to consider that the clinical and imaging findings may actually stem from parturitional and neonatal issues.[75–112] These include hemorrhage or rehemorrhage into extracerebral collections existing from birth (see **Figs. 1, 4, 13**, and **15**). There may be associated skeletal findings of birth trauma (eg, new or healing clavicle, rib, or long bone fractures), particularly in the presence of a bone fragility disorder (see **Figs. 1, 2** and **5**).[133–137]

Developmental Anomalies and Congenital Conditions

Vascular malformations are rarely reported causes for the triad but may be underdiagnosed (see **Fig. 9**). BECCs and arachnoid cysts are also known to be associated with SDH and RH, spontaneously and with trauma (see **Figs. 4, 15–17**).[65–74]

Genetic and Metabolic Disorders

Several conditions in the genetic and metabolic disorders category may present with intracranial hemorrhage (eg, SDH) or RH. These include osteogenesis imperfecta, glutaric aciduria type I (see **Fig. 12**), Menkes' kinky hair disease, Ehlers-Danlos and Marfan syndromes, homocystinuria, and others.[4,89,101,138–142]

Hematologic Disease and Coagulopathy

Conditions in the hematologic disease and coagulopathy category predispose to intracranial hemorrhage and RH (see **Figs. 11** and **13**). The bleeding or clotting disorder may be primary or secondary. A more extensive work-up beyond the usual screening tests is needed, including a hematology consultation. Conditions in the category include the anemias, hemorrhagic disease of the newborn (vitamin K deficiency), the hemophilias, thrombophilias, disseminated intravascular coagulation and consumption coagulopathy, liver or kidney disease, hemophagocytic lymphohistiocytosis, and anticoagulant therapy.[4,89,101,143–145] Venous thrombosis includes dural venous sinus thrombosis (DVST) and cerebral venous thrombosis (CVT). DVST or CVT may be associated with primary or secondary hematologic or coagulopathic states.[4,89,101,146–152] Risk factors include acute systemic illness, dehydration, fluid-electrolyte imbalance, sepsis, perinatal complications, chronic systemic disease, cardiac disease, connective tissue disorder, hematologic disorder, oncologic disease and therapy, head and neck infection, hypercoagulable, and trauma states. Infarction, SAH, SDH, or RH may be seen, especially in infants. High densities on CT may be present along the dural venous sinuses, tentorium, falx, or the cortical, subependymal, or medullary veins and be associated with SAH, SDH, or intracerebral hemorrhage (see **Fig. 8**). There may be focal infarctions, hemorrhagic or nonhemorrhagic, intraventricular hemorrhage, and massive, focal, or diffuse edema. Orbit, paranasal sinus, or otomastoid disease may be present. The thromboses and associated hemorrhages have variable MR imaging appearances depending on their age. CT venography (CTV) or magnetic resonance venography (MRV) may readily detect DVST but not CVT. The latter may be better detected as abnormal hypointensities on susceptibility-weighted T2* sequences but difficult to distinguish from hemorrhage (SDH or SAH), hemorrhagic infarction, contusion, or hemorrhagic shear injury.

Infectious and Postinfectious Conditions

Meningitis, encephalitis, or sepsis may involve the vasculature resulting in vasculitis, arterial or venous thrombosis, mycotic aneurysm, infarction, and hemorrhage.[4,89,101] SDH and RH may also be seen (see **Fig. 10**). Postinfectious illnesses may also be associated with these findings. Included in this category are the encephalopathies of infancy and childhood, hemorrhagic shock and encephalopathy syndrome, and postvaccinial encephalopathy.[4,89,101,153–158]

Toxins, Poisons, and Nutritional Deficiencies

The category of toxins poisons, and nutritional deficiencies includes lead poisoning, cocaine, anticoagulants, over-the-counter cold medications, prescription drugs, and vitamin deficiencies or depletions (eg, K, C, or D).[4,89,101,136,143,155–159] Preterm neonates, and other chronically ill infants, are particularly vulnerable to nutritional deficiencies and complications of prolonged immobilization that often primarily effect bone development. Furthermore, the national and

international epidemic of vitamin D deficiency and insufficiency in pregnant mothers, their term fetuses, and their undersupplemented breastfed term neonates predisposes them to rickets (ie, congenital). Such infants, who have also been subjected to the trauma of birth, may have skeletal imaging findings (eg, multiple healing fractures or pseudofractures) that are misinterpreted as NAI, especially in the presence of the triad (see **Figs. 2** and **5**).[136,137]

Dysphagic Choking ALTE as a Mimic of NAI

Apnea is an important and common form of ALTE in infancy whose origin may be central, obstructive, or combined.[25] The obstructive and mixed forms may present with choking, gasping, coughing, or gagging due to mechanical obstruction. When paroxysmal or sustained, the result may be severe brain injury or death due to a combination of central venous hypertension and hypoxia-ischemia. It is this synergism that produces cerebral edema and dural vascular plexus hemorrhage with SDH, SAH, and RH (see **Fig. 2**; **Fig. 18**). Examples include dysphagic choking (eg, aspiration of a feed or gastroesophageal reflux), viral airway infection (eg, RSV), and pertussis, particularly when occurring in a predisposed child (eg, prematurity, Pierre Robin syndrome, or sudden infant death syndrome).[25,160–167]

IMAGING CHALLENGES AND THE IMPORTANCE OF A DIFFERENTIAL DIAGNOSIS

CT

Because of the evidence-based challenges to NAI, imaging protocols should be designed to evaluate not only NAI versus AI but also the medical mimics. Noncontrast CT has been the primary modality for brain imaging because of its access, speed, and ability to show lesions (eg, hemorrhage and edema) requiring immediate neurosurgical or medical intervention.[4,77,83–99,102–112,168–181] Cervical spinal CT may also be needed. CT angiography (CTA) or CTV may be helpful to evaluate the cause of hemorrhage (eg, vascular malformation or aneurysm) or infarction (eg, dissection or venous thrombosis). A radiographic or scintigraphic skeletal survery should also be obtained according to established guidelines.[179,180]

MR Imaging

Brain and cervical spinal MR imaging should be done as soon as possible because of its sensitivity and specificity regarding pattern of injury and timing parameters.[4,104,181–190] Brain MR imaging should include T1, T2, T2*, fluid-attenuated inversion recovery (FLAIR), and diffusion-weighted imaging/apparent diffusion coefficient (DWI/ADC). Gadolinium-enhanced T1 images should probably be used along with MRA and MRV. T1 and T2 are necessary for estimating the timing of hemorrhage, thrombosis, and other collections using published criteria.[4,104,181] T2* techniques are most sensitive for detecting hemorrhage or thromboses but may not distinguish new (eg, deoxyhemoglobin) from old (eg, hemosiderin). DWI plus ADC can be quickly obtained to show hypoxia-ischemia or vascular occlusive ischemia.[4,154,189,190] Restricted or reduced diffusion, however, may be seen with other processes, including encephalitis, seizures, or metabolic disorders, and with suppurative collections and some tumors.[4,154,189,190] Gadolinium-enhanced sequences and MRS can be used to evaluate for these other processes. Additionally, MRA and

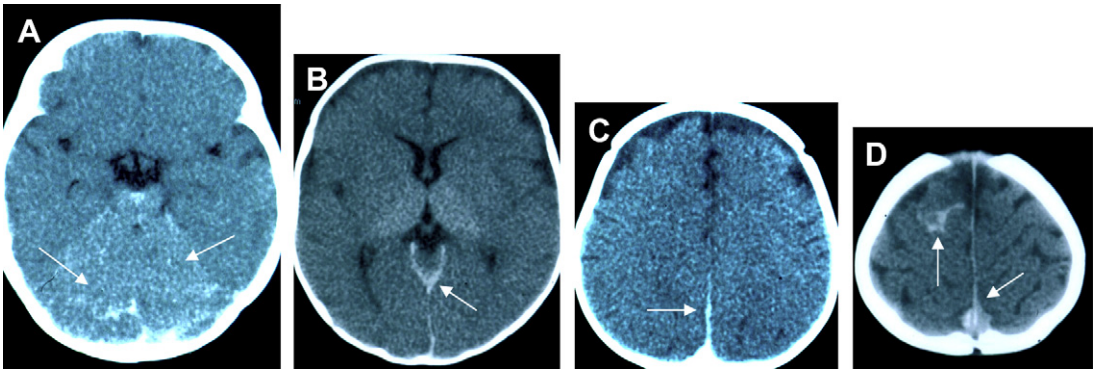

Fig. 18. Six-month-old infant with triad and alleged NAI; acute choking event while feeding. CT (*A–D*) shows bilateral cerebral edema with acute SAH and SDH (*arrows*), including along the falx, and tentorium. Autopsy confirmed the hemorrhages, a subdural membrane, and hypoxic-ischemic brain injury. (*Courtesy of* The Wisconsin Innocence Project.)

MRV are important to evaluate for arterial occlusive disease (eg, dissection) or venous thrombosis, although they cannot rule out small vessel disease. The STIR technique is particularly important for cervical spine imaging.

Scalp and Skull Abnormalities

Scalp injuries (eg, edema, hemorrhage, and laceration) are difficult to precisely time on imaging studies and depend on the nature and number of traumatic events or other factors (circulatory compromise, coagulopathy, medical interventions, and so forth).[1,4] Skull abnormalities may include fracture and suture splitting. Fracture may not be readily distinguished from sutures, synchondroses, their normal variants, or from wormian bones (eg, osteogenesis imperfecta) on CT or skull films. 3-D–CT surface reconstructions may be needed. In general, the morphology of a fracture cannot differentiate NAI from AI and must be correlated with the trauma scenario (eg, biomechanically) (see **Fig. 1**). Skull fractures are also difficult to time because of the lack of periosteal reaction.[1,4] Suture diastasis may be traumatic or a reflection of increased intracranial pressure but must be distinguished from pseudodiastasis due to a metabolic or dysplastic bone disorder (eg, congenital rickets) (see **Fig. 2**).[1,4,136,137] The growing fracture (eg, leptomeningeal cyst) is not specific for NAI and may follow any diastatic fracture in a young infant, including birth related (see **Fig. 1**).[1,2,4] Nondetection of scalp or skull abnormalities on imaging should not be interpreted as the absence of impact injury.

Intracranial Collections

It should not be assumed that such collections are always traumatic in origin. A differential diagnosis is always necessary and includes NAI, AI, coagulopathy (hemophilic and thrombophilic conditions), infectious and postinfectious conditions, metabolic disorders, and so forth.[2,4,22,89,90,101,106–110] It may not be possible to specify with any precision the components or age of an extracerebral collection because of meningeal disruptions (eg, acute or subacute subdural hygroma [SDHG] versus chronic SDH, or subarachnoid versus thin SDH).[1,4,103,104,173–176,181] Vezina[181] has recently summarized the literature regarding the complexity of timing of intracranial collections. Subarachnoid and subdural collections, hemorrhagic or nonhemorrhagic, may be localized or extensive and may occur about the convexities, interhemispheric (along the falx), and along the tentorium. With time and gravity, these collections may redistribute to other areas, including into or out of the spinal canal, and cause confusion.[4,177,181,191] For example, a convexity SDH may migrate to the peritentorial and posterior interhemispheric regions or into the intraspinal spaces. SDH migration may lead to a misinterpretation that there are hemorrhages of different timing. The distribution or migration of the sediment portion of a hemorrhage with blood levels (ie, hematocrit effect) may cause further confusion because density/intensity differences between the sediment and supernatant may be misinterpreted as hemorrhages (and trauma) of differing age and location.[4,104,178,181] Prominent subarachnoid CSF spaces are commonly present in infants (ie, BECCs). This entity predisposes infants to SDH, which may be spontaneous or associated with trauma of any type (eg, dysphagic choking ALTE) (see **Figs. 4, 15,** and **17**).[4,65–73] A hemorrhagic collection may continually change or evolve with regard to size, extent, location, and density/intensity characteristics. Rapid spontaneous resolution and redistribution of acute SDH over a few hours to 1 to 2 days has been reported.[4,177,191] A tear in the arachnoid may allow SDH washout into the subarachnoid space or CSF dilution of the subdural space.

For apparent CT high densities, it may be difficult to differentiate cerebral hemorrhage from subarachnoid hemorrhage or from venous thrombosis (see **Figs. 2, 3, 6–11, 15, 16**, and **18**).[4] According to the literature, hemorrhage or thromboses that are high density (ie, clotted) on CT (ie, acute to subacute) have a wide timing range of 0 to 3 hours up to 7 to 10 days.[4,104,178,181] Hemorrhage that is isohypodense on CT (ie, nonclotted) may be hyperacute (<3 h) or chronic (>10 d) (see **Figs. 3** and **11**). The low density may also represent pre-existing, wide, CSF-containing subarachnoid spaces (eg, BECC) or SDHG (ie, CSF-containing) that may be acute or chronic (see **Figs. 3, 12** and **15**).[4,103,104,175,181] Blood levels are unusual in the acute stage unless there is coagulopathy.[4,104,181,188] CT cannot distinguish acute hemorrhage from rehemorrhage on existing chronic collections (BECC or chronic SDHG) (see **Figs. 3** and **15**).[4,66,72,92–104,173,178,181] Traditionally, the interhemispheric SDH as well as mixed-density SDH were considered characteristic, if not pathognomonic, of SBS/NAI.[1,2,4,168,171–173] This has been proved unreliable. Interhemispheric SDH may be seen with AI or with nontraumatic conditions (eg, HIE, venous thrombosis, venous hypertension, or dysphagic choking ALTE) (see **Figs. 2, 6–10**).[178] Mixed-density SDH also occurs in AI as well as in other conditions (see **Figs. 3, 9,** and **11**).[178] Furthermore, SDH may occur in BECC

spontaneously or result from minor trauma (ie, AI), and rehemorrhage within SDH may occur spontaneously or with minor AI (see **Figs. 1, 4, 15,** and **17**).[4,12,38,40,72,90,104,178,181]

Only MR imaging may provide more precise information than CT regarding pattern of injury and timing, particularly with regard to (1) hemorrhage versus thromboses (**Table 1**) and (2) brain injury.[104,181–190] As a result, MR imaging has become the standard and should be done as soon as possible. Mixed-intensity collections, however, are problematic regarding timing.[181] Matching the MR imaging findings with the CT findings may help along with follow-up MR imaging. Blood levels may indicate subacute hemorrhage versus coagulopathy. The timing guidelines are better applied to the sediment than to the supernatant. With mixed-intensity collections, MR imaging cannot reliably differentiate BECC with acute SDH from acute SDHG/SDH, from hyperacute SDH, or from chronic SDH or chronic SDHG with rehemorrhage (see **Figs. 1, 4,** and **13–17**).[4,104,181] T2* hypointensities are iron sensitive but may not differentiate hemorrhages from venous thromboses that are not detected by MRV (eg, cortical, medullary, or subependymal).

BRAIN INJURY

Edema or swelling in pediatric head trauma may represent primary injury or secondary injury and be acute-hyperacute (eg, minutes to a few hours) or delayed (eg, several hours to a few days), including association with short-distance falls and lucid intervals.[4,53–62] The edema or swelling may be further subtyped as traumatic, malignant, hypoxic-ischemic, or related to (or combined with) other factors. Traumatic edema is related to areas of primary brain trauma (ie, contusion or shear) or to traumatic vascular injury with infarction (eg, dissection, herniation, or spasm) (see **Figs. 3, 6, 9,** and **11**). Traumatic edema is usually focal or multifocal, whether or not hemorrhagic. CT, however, may not distinguish focal or multifocal cerebral high densities as hemorrhagic contusion, hemorrhagic shear, or hemorrhagic infarction.[4] Focal or multifocal low density edema may also be seen with infarction (eg, arterial or venous occlusive), encephalitis, demyelination (eg, ADEM), or seizure edema.[4,89,146–154] Also, MR imaging often shows shear and contusional injury as focal/multifocal restricted diffusion, GRE hypointensities, and/or T2/FLAIR high intensities.[4] Focal/multifocal ischemic findings may also be due to traumatic arterial injury (eg, dissection) or venous injury (eg, tear or thrombosis), arterial spasm (as with any cause of hemorrhage), herniation, or edema with secondary perfusion deficit or seizures (eg, status epilepticus) (see **Figs. 2, 6,** and **11**).[4,64,154,189,192] These may not be reliably differentiated, however, from focal/multifocal ischemic or hemorrhagic infarction from nontraumatic causation (eg, dissection, vasculitis, venous, or embolic) even without supportive MRA, CTA, MRV, or angiography. Also, similar cortical or subcortical intensity abnormalities (including restricted diffusion) may also be observed with

Table 1
MR imaging of intracranial hemorrhage and thrombosis[a]

Stage	Biochemical Form	Site	T1–MR Imaging	T2–MR Imaging
Hyperacute (+ edema) (<12 hours)	Fe II oxyHb	Intact RBCs	Iso-low I	High I
Acute (+ edema) (1–3 days)	Fe II deoxy Hb	Intact RBCs	Iso-low I	Low I
Early subacute (+ edema) (3–7 days)	Fe III metHb	Intact RBCs	High I	Low I
Late subacute (–edema) (1–2 weeks)	Fe III metHb	Lysed RBCs (extracellular)	High I	High I
Early chronic (–edema) (>2 weeks)	Fe III transferrin	Extracellular	High I	High I
Chronic (cavity)	Fe III ferritin and hemosiderin	Phagocytosis	Iso-low I	Low I

[a] Fe II, ferrous; Fe III, ferric; Hb, hemoglobin; I, signal intensity; Iso, isointense; RBCs, red blood cells; +, present; –, absent. *Data from* Refs. [4,188,189]

encephalitis, seizures, and metabolic disorders. Therefore, a differential diagnosis is always required.[4,154,189,192]

Malignant brain edema, a term used for severe cerebral swelling after head trauma, may lead to rapid deterioration.[1,4,54,55,62] The edema is usually bilateral and may be related to cerebrovascular congestion (ie, hyperemia) as a vasoreactive rather than an autoregulatory phenomenon and associated with global ischemia. A unilateral form may also occur in association with an ipsilateral SDH that progresses to bilateral edema (see **Figs. 3** and **6**).[64] There may be rapid or delayed onset (ie, lucid interval). Predisposing factors are not well established but likely include a genetic basis. Hyperemic edema may appear early as accentuated gray-white matter differentiation on CT, then progresses to loss of differentiation.

Global hypoxia (eg, apnea or respiratory failure) or ischemia (eg, cardiovascular failure or hypoperfusion) is likely a major cause of or contributor to brain edema in a child with head trauma (eg, malignant edema).[4,38,40,54,55,62] HIE, depending on its severity and duration, may have a diffuse appearance acutely (ie, diffuse or vascular axonal injury) with decreased gray-white differentiation throughout the cerebrum on CT (eg, white cerebellum sign) and then evolve to a more specific pattern on CT or MR imaging (eg, border zone or watershed, basal ganglia/thalamic, cerebral white matter necrosis, reversal sign) (see **Figs. 2, 6, 7, 10,** and **18**).[4,189] It is typically bilateral but may not be symmetric. This more diffuse pattern may distinguish HIE from the multifocal pattern of primary traumatic injury, although they may coexist. Hypoxia-ischemic brain injury due to apnea/respiratory arrest may occur with head trauma or with neck/cervical spine/cord injuries (eg, SCIWORA) whether or not AI or NAI (see **Fig. 7**).[4,35,54,55,62] It may also occur with any nontraumatic cause (choking, paroxysmal coughing, aspiration, and so forth) (see **Figs. 2** and **18**).[4,25,160–166] In addition to the diffuse brain injury, there may be associated subarachnoid and SDH without mass effect (see **Figs. 2, 7, 10,** and **18**).[4,38,40,54,55,62] MR imaging shows hypoxic-ischemic injury, depending on timing, as diffuse-restricted diffusion on DWI/ADC plus matching T1/T2 abnormalities as the injury evolves (see **Figs. 2, 6** and **11**).[4,189] Other important contributors to edema or swelling include such complicating factors as seizures (eg, status epilepticus [see **Fig. 2**], fluid-electrolyte imbalance, other systemic or metabolic derangements (eg, hypoglycemia, hyperglycemia, hyperthermia), or hydrocephalus.[4] It is well known that many of these may also be associated with restricted diffusion along with other nontraumatic processes (encephalitis, seizures, and metabolic disorders).[4,154,186,187,189] Again, a differential diagnosis is required.

SUMMARY

An extensive review of the literature to date fails to establish an evidence base for reliably distinguishing NAI from AI or from the medical mimics. The medical and imaging findings alone cannot diagnose intentional injury. Only a child protection investigation may provide the basis for inflicted injury in the context of supportive medical, imaging, or pathologic data. The duty of a radiologist is to give a detailed description of the imaging findings, provide a differential diagnosis, and communicate the concern for NAI, directly to the primary care team in a timely manner. Radiologists should be prepared to consult with child protection services; other medical and surgical consultants, including a pathologist or biomechanical specialist; law enforcement investigators; and attorneys for all parties as appropriate. Radiologists must also be aware of certain conditions that are known to have clinical and imaging features that may mimic abuse. These should be properly evaluated, and the possibility of combined or multifactorial mechanisms with synergistic effects should also be considered. Furthermore, a negative medical evaluation does not make NAI the default diagnosis. A timely and thorough multidisciplinary evaluation may be the difference between appropriate child protection versus an improper breakup of a family or a wrongful indictment and conviction.

REFERENCES

1. Kleinman P. Diagnostic imaging of child abuse. New York: Mosby Year Book; 1998.
2. Frasier L. Abusive head trauma in infants and children. St Louis (MO): GW Medical Publishing; 2006.
3. Kellogg N. Committee on child abuse and neglect. Evaluation of suspected child physical abuse. Pediatrics 2007;119:1232–41.
4. Barnes P, Krasnokutsky M. Imaging of the CNS in Suspected or Alleged NAI. Top Magn Reson Imaging 2007;18:53–74.
5. Donohoe M. Evidence-based medicine and shaken baby syndrome part I: literature review, 1966–1998. Am J Forensic Med Pathol 2003;24: 239–42.
6. Leestma J. Case analysis of brain injured admittedly shaken infants, 54 cases 1969–2001. Am J Forensic Med Pathol 2005;26:199–212.

7. Lyons G. Shaken baby syndrome: a questionable scientific syndrome and a dangerous legal concept. Utah Law Rev 2003;1109:1–22.
8. Gena M. Shaken baby syndrome: medical uncertaintly casts doubt on convictions. Wis L Rev 2007;701:1–26.
9. Goudge Hon ST. Report of the inquiry into pediatric forensic pathology in Ontario. Ontario Ministry of the Attorney General. Queen's Printer for Ontario September 30, 2008. Available at: www.goudgeinquiry.ca. Accessed September 12, 2010.
10. Mackey M. After the court of appeal: R v Harris and others [2005] EWCA crim 1980. Arch Dis Child 2006;91:873–5.
11. Tuerkheimer D. The next innocence project: shaken baby syndrome and the criminal courts. Wash U L Rev 2009;87(1):1–58.
12. Squier W. Shaken baby syndrome: the quest for evidence. Dev Med Child Neurol 2008;50:10–4.
13. David TJ. Non-accidental head injury—the evidence. Pediatr Radiol 2008;38(Suppl 3):S370–7.
14. Jaspan T. Current controversies in the interpretation of non-accidental head injury. Pediatr Radiol 2008;38(Suppl 3):S378–87.
15. Guyatt, Haynes RB, Jaeschke RZ, et al. Users' guides to the medical literature. XXV. Evidence-based medicine. JAMA 2000;284:1290–6.
16. Collins J. Evidence-based medicine. J Am Coll Radiol 2007;4(8):551–4.
17. Blackmore C, Medina LS. Evidence-based radiology and the ACR appropriateness criteria. J Am Coll Radiol 2006;3(7):505–9.
18. Crosskerry P. The importance of cognitive errors in diagnosis and strategies to minimize them. Acad Med 2003;78:775–80.
19. Newman, DH. Physician says medical ideology "gets in the way" of evidence-based medicine. New York Times 4-2-2009; AMA News 4-3-09 [online].
20. Groopman J, Hartzband P. Why 'quality care' is dangerous. Wall St J. Available at: WSJ.com. Accessed April 8, 2009.
21. Feldman K, Bethel R, Shugerman P, et al. The cause of infant and toddler subdural hemorrhage: a prospective study. Pediatrics 2001;108:636–46.
22. Hobbs C, Childs A, Wynne J, et al. Subdural haematoma and effusion in infancy: an epidemiological study. Arch Dis Child 2005;90:952–5.
23. Keierleber J, Bohan T. Ten years after Daubert: the status of the states. J Forensic Sci 2005;50:1–10.
24. Udashen G, Sperling C. Texas v. Hurtado (Daubert), 2006.
25. DeWolfe CC. Apparent life-threatening event: a review. Pediatr Clin North Am 2005;52:1127–46.
26. Bonkowsky J, Guenther E, Filoux F, et al. Death, child abuse, and adverse neurological outcome of infants after an apparent life-threatening event. Pediatrics 2008;122:125–31.
27. Uscinski R. Shaken baby syndrome: fundamental questions. Br J Neurosurg 2002;16:217–9.
28. Ommaya A. Whiplash injury and brain damage. JAMA 1968;204:75–9.
29. Duhaime A, Gennerelli T, Thibault L, et al. The shaken baby syndrome. A clinical, pathological, and biomechanical study. J Neurosurg 1987;66:409–15.
30. Prange M, Coats B, Duhaime A, et al. Anthropomorhic simulations of falls, shakes, and inflicted impacts in infants. J Neurosurg 2003;99:143–50.
31. Leestma J, editor. Forensic neuropathology. 2nd edition. Boca Raton (FL): CRC Press; 2009. p. 603.
32. Pierce MC, Bertocci G. Injury biomechanics and child abuse. Annu Rev Biomed Eng 2008;10:85–106.
33. Ommaya A, Goldsmith W, Thibault L. Biomechanics and neuropathology of adult and paediatric head injury. Br J Neurosurg 2002;16:220–42.
34. Bandak FA. Shaken baby syndrome: a biomechanics analysis of injury mechanisms. Forensic Sci Int 2005;151:71–9.
35. Barnes P, Krasnokutsky M, Monson K, et al. Traumatic spinal cord injury: accidental vs. nonaccidental injury. Semin Pediatr Neurol 2008;15:178–84.
36. Geddes J, Hackshaw A, Vowles G, et al. Neuropathology of inflicted head injury in children. I. Pattern of brain injury. Brain 2001;124:1290–8.
37. Geddes J, Vowles G, Hackshaw A, et al. Neuropathology of inflicted head injury in children. II. Microscopic brain injury in infants. Brain 2001;124:1299–306.
38. Geddes J, Tasker R, Hackshaw A, et al. Dural haemorrhage in non-traumatic infant deaths: does it explain the bleeding in 'shaken baby syndrome'? Neuropathol Appl Neurobiol 2003;29:14–22.
39. Byard R, Blumbergs P, Rutty G, et al. Lack of evidence for a causal relationship between hypoxic-ischemic encephalopathy and subdural hemorrhage in fetal life, infancy, and early childhood. Pediatr Dev Pathol 2007;10:348–50.
40. Cohen M, Scheimberg I. Evidence of occurrence of intradural and subdural hemorrhage in the perinatal and neonatal period in the context of hypoxic ischemic encephalopathy. Pediatr Dev Pathol 2009;12:169–76.
41. Croft P, Reichard R. Microscopic examination of grossly unremarkable pediatric dura mater. Am J Forensic Med Pathol 2009;30:10–3.
42. Emerson M, Pieramici D, Stoessel K, et al. Incidence and rate of disappearance of retinal hemorrhage in newborns. Ophthalmology 2001;108:36–9.

43. Chadwick D, Bertocci G, Castillo E, et al. Annual risk of death resulting from short falls among children. Pediatrics 2008;121:1213–24.
44. Hall J, Reyes H, Horvat M, et al. The mortality of childhood falls. J Trauma 1989;29:1273–5.
45. Chadwick D, Chin S, Salerno C, et al. Deaths from falls in children: how far is fatal. J Trauma 1991;31: 1335.
46. Helfer R, Slovis T, Black M. Injuries resulting when small children fall out of bed. Pediatrics 1977;60: 533–5.
47. Reiber G. Fatal falls in childhood. Am J Forensic Med Pathol 1993;14:201–7.
48. Williams R. Injuries in infants and small children resulting from witnessed and corroborated free falls. J Trauma 1991;31:1350–2.
49. Lyons T, Oates R. Falling out of bed: a relatively benign occurrence. Pediatrics 1993;92:125–7.
50. Oehmichen M, Meissner C, Saternus K. Fall or shaken: traumatic brain injury in children caused by falls or abuse at home—a review on biomechanics and diagnosis. Neuropediatrics 2005;36:240–5.
51. Duhaime A, Alario A, Lewander W, et al. Head injury in very young children: mechanisms, injury types, and ophthalmologic findings in 100 hospitalized patients younger than 2 years of age. Pediatrics 1992;90:179–85.
52. Schutzman SA, Barnes PD, Duhaime A-C, et al. Evaluation and management of children younger than two years old with apparently minor head trauma: proposed guidelines. Pediatrics 2001; 107:983–93.
53. Stein S, Spettell C. Delayed and progressive brain injury in children and adolescents with head trauma. Pediatr Neurosurg 1995;23:299–304.
54. Bruce D. Delayed deterioration of consciousness after trivial head injury in childhood. Br Med J (Clin Res Ed) 1984;289:715–6.
55. Bruce D, Alavi A, Bilaniuk L, et al. Diffuse cerebral swelling following head injuries in children: the syndrome of malignant brain edema. J Neurosurg 1981;54:170–8.
56. Plunkett J. Fatal pediatric head injuries caused by short-distance falls. Am J Forensic Med Pathol 2001;22:1–12.
57. Greenes D, Schutzman S. Occult intracranial trauma in infants. Ann Emerg Med 1998;32:680–6.
58. Arbogast K, Margulis S, Christian C. Initial neurologic presentation in young children sustaining inflicted and unintentional fatal head injuries. Pediatrics 2005;116:180–4.
59. Denton S, Mileusnic D. Delayed sudden death in an infant following an accidental fall. Am J Forensic Med Pathol 2003;24:371–6.
60. Murray J, Chen D, Velmahos G, et al. Pediatric falls: is height a predictor of injury and outcome? Am Surg 2000;66:863–5.
61. Kim K, Wang M, Griffith P, et al. Analysis of pediatric head injury falls. Neurosurg Focus 2000;8: 1–9.
62. Steinbok P, Singhal A, Poskitt K, et al. Early hypodensity on CT scan of the brain in accidental pediatric head injury. Neurosurgery 2007;60: 689–95.
63. Christian CW, Taylor AA, Hertle RW, et al. Retinal hemorrhages caused by accidental household trauma. J Pediatr 1999;135:125–7.
64. Durham SR, Duhaime A- C. Maturation-dependent response of the immature brain to experimental subdural hematoma. J Neurotrauma 2007;24: 5–14.
65. Azais M, Echenne B. Idiopathic pericerebral effusions of infancy (external hydrocephalus). Annales Pediatr (Paris) 1992;39:550–8.
66. Piatt J. A pitfall in the diagnosis of child abuse: external hydrocephalus, subdural hematoma, and retinal hemorrhages. Neurosurg Focus 1999;7(4): 1–8.
67. Pittman T. Significance of subdural hematoma in a child with external hydrocephalus. Pediatr Neurosurg 2003;39:57–9.
68. Papasian N, Frim D. A theoretical model of benign external hydrocephalus that predicts a predisposition towards extra-axial hemorrhage after minor head trauma. Pediatr Neurosurg 2000;33:188–93.
69. Hangique S, Das R, Barua N, et al. External hydrocephalus in children. Ind J Radiol Imag 2002;12: 197–200.
70. Mori K, Sakamoto T, Mishimura K, et al. Subarachnoid fluid collection in infants complicated by subdural hematoma. Childs Nerv Syst 1993;9: 282–4.
71. Ravid S, Maytal J. External hydrocephalus: a probable cause for subdural hematoma of infancy. Pediatr Neurol 2003;28:139–41.
72. McNeely PD, Atkinson JD, Saigal G, et al. Subdural hematomas in infants with benign enlargement of the subarachnoid spaces are not pathognomonic for child abuse. Am J Neuroradiol 2006;27:1725–8.
73. Hellbusch L. Benign extracerebral fluid collections in infancy: clinical presentation and long-term follow-up. J Neurosurg 2007;107:119–25.
74. Mori K, Yamamoto T, Horinaka N, et al. Arachnoid cyst is a risk factor for chronic subdural hematoma in juveniles. J Neurotrauma 2002;19:1017–27.
75. Chamnanvanakij S, Rollins N, Perlman J. Subdural hematoma in term infants. Pediatr Neurol 2002;26: 301–4.
76. Whitby E, Griffiths P, Rutter S, et al. Frequency and natural history of subdural hemorrhages in babies and relation to obstetric factors. Lancet 2004;363: 846–51.

77. Hayashi T, Hashimoto T, Fukuda S, et al. Neonatal subdural hematoma secondary to birth injury. Childs Nerv Syst 1987;3:23–9.
78. Rooks V, Eaton J, Ruess L, et al. Prevalence and evolution of intracranial hemorrhage in asymptomatic term infants. AJNR Am J Neuroradiol 2008; 29:1082–9.
79. Volpe JJ. Neurology of the newborn. 4th edition. Philadelphia: WB Saunders; 2000.
80. Ney J, Joseph K, Mitchell M. Late subdural hygromas from birth trauma. Neurology 2005;65:517.
81. Holden K, et al. Cranial MRI of normal term neonates: a pilot study. J Child Neurol 1999;14: 708–10.
82. Looney C, Smith J, Merck L, et al. Intracranial hemorrhage in asymptomatic neonates: prevalence on MRI and relationship to obstetric and neonatal risk factors. Radiology 2007;242: 535–41.
83. Powers C, Fuchs H, George T. Chronic subdural hematoma of the neonate. Pediatr Neurosurg 2007;43:25–8.
84. Ross M, Fresquez M, El-Hacklad M. Impact of FDA advisory on reported vacuum-assisted delivery and morbidity. J Matern Fetal Med 2000;9:321–6.
85. Towner D, Castro M, Eby-Wilkens E, et al. Effect of mode of delivery in nulliparous women on neonatal intracranial injury. N Engl J Med 1999;341: 1709–14.
86. Polina J, Dias M, Kachurek D, et al. Cranial birth injuries in term newborn infants. Pediatr Neurosurg 2001;35:113–9
87. Alexander J, Leveno K, Hauth J, et al. Fetal injury associated with cesarean delivery. Obstet Gynecol 2006;108:885–90.
88. Doumouchtsis S, Arulkumaran S. Head trauma after instrumental births. Clin Perinatol 2008;35: 69–83.
89. Sirotnak A. Medical disorders that mimic abusive head trauma. In: Frasier L, editor. Abusive head trauma in infants and children. St Louis (MO): GW Medical Publishing; 2006. p. 191–226.
90. Mack J, Squier W, Eastman J. Anatomy and development of the meninges: implications for subdural collections and cerebrospinal fluid circulation. Pediatr Radiol 2009;39:200–10.
91. Haines D, Harkey H, Al-Mefty O. The subdural space: a new look at an outdated concept. Neurosurgery 1993;32:111–20.
92. Kawakami Y, Chikama M, Tamiya T, et al. Coagulation and fibrinolysis in chronic subdural hematoma. Neurosurgery 1998;25:25–9.
93. Murakami H, Hirose Y, Sagoh M, et al. Why do chronic subdura hematomas continue to grow slowly and not coagulate? J Neurosurg 2002;96: 877–84.
94. Parent AD. Pediatric chronic subdural hematoma. A retrospective comparative analysis. Pediatr Neurosurg 1992;18:266–71.
95. Hwang S, Kim S. Infantile head injury, with special reference to the development of chronic subdural hematoma. Child's Nerv Syst 2000;16:590–4.
96. Fung E, Sung RY, Nelson EA, et al. Unexplained subdural hematoma in young children: is it always child abuse. Pediatr Int 2002;44:37–42.
97. Aoki N, Masuzawa H. Infantile acute subdural hematoma. J Neurosurg 1984;61:273–80.
98. Aoki N. Chronic subdural hematoma in infancy. J Neurosurg 1990;73:201–5.
99. Ikeda A, Sato O, Tsugane R, et al. Infantile acute subdural hematoma. Child's Nerv Syst 1987;3:19–22.
100. Dyer O. Brain haemorrhage in babies may not indicate violent abuse. BMJ 2003;326:616.
101. Hymel K, Jenny C, Block R. Intracranial hemorrhage and rebleeding in suspected victims of abusive head trauma: addressing the forensic controversies. Child Maltreat 2002;7:329–48.
102. Howard M, Bell B, Uttley D. The pathophysiology of infant subdural haematomas. Br J Neurosurg 1993; 7:355–65.
103. Vinchon M, Noizet O, Defoort-Dhellemmes S, et al. Infantile subdural hematomas due to traffic accidents. Pediatr Neurosurg 2002;37: 245–53.
104. Vinchon M, Noule N, Tchofo P, et al. Imaging of head injuries in infants: temporal correlates and forensic implications for the diagnosis of child abuse. J Neurosurg 2004;101:44–52.
105. Maxeiner H. Demonstration and interpretation of bridging vein ruptures in cases of infantile subdural bleeding. J Forensic Sci 2001;46:85–93.
106. Minns R. Subdural haemorrhages, haematomas, and effusions of infancy. Arch Dis Child 2005;90: 883–4.
107. Hobbs C, Childs A, Wynne J, et al. Subdural haematoma and effusion in infancy. Arch Dis Child 2005;90:952–5.
108. Datta S, Stoodley N, Jayawant S, et al. Neuroradiological aspects of subdural haemorrhages. Arch Dis Child 2005;90:947–51.
109. Kemp A. Investigating subdural haemorrhage in infants. Arch Dis Child 2002;86:98–102.
110. Jayawant S, Rawlinson A, Gibbon F, et al. Subdural haemorrhages in infants: population based study. BMJ 1998;317:1558–61.
111. Jayawant S, Parr J. Outcome following subdural haemorrhages in infancy. Arch Dis Child 2007;92: 343–7.
112. Trenchs V, Curcoy A, Navarro R, et al. Subdural haematomas and physical abuse in the first two years of life. Pediatr Neurosurg 2007;43: 352–7.

113. Galaznik J. Eye findings and allegations of shaking and non-accidental injury: post-publication peer review (8 August 2007). Pediatrics 2007;19: 1232–41.
114. Galaznik J. Shaken baby syndrome: letter to the editor. Dev Med Child Neurol 2008;50:317–9.
115. Levin A, Wygnanski-Jaffe T, Shafiq A, et al. Post-mortem orbital findings in shaken baby syndrome. Am J Ophthalmol 2006;142:233–40.
116. Morad Y, Kim Y, Armstrong D, et al. Correlation between retinal abnormalities and intracranial abnormalities in the shaken baby syndrome. Am J Ophthalmol 2002;134:354–9.
117. Kirshner R, Stein R. The mistaken diagnosis of child abuse. A form of medical abuse? Am J Dis Child 1985;139:873–5.
118. Tongue A. The ophthalmologists role in diagnosing child abuse. Ophthalmology 1991;98:1009–10.
119. Gardner H. Correlation between retinal abnormalities and intracranial abnormalities in the shaken baby syndrome. Am J Ophthalmol 2003;135: 745–6.
120. Gilles E, McGregor M, Levy-Clarke G. Retinal hemorrhage asymmetry in inflicted head injury: a clue to pathogenesis? J Pediatr 2003;143:494–9.
121. Gilliland M, Luthert P. Why do histology on retinal hemorrhages in suspected nonaccidental injury. Histopathology 2003;43:592–602.
122. Lantz P, Sinal S, Staton C, et al. Perimacular retinal folds from childhood head trauma: evidence-based case report. BMJ 2004;328: 754–6.
123. Aryan H, Ghosheh F, Jandial R, et al. Retinal hemorrhage and pediatric brain injury: etiology and review of the literature. J Clin Neurosci 2005; 12:624–31.
124. Lueder GT, Turner JW, Paschall R. Perimacular retinal folds simulating nonaccidental injury in an infant. Arch Ophthalmol 2006;124:1782–3.
125. Obi E, Watts P. Are there any pathognomonic signs in shaken baby syndrome. J AAPOS 2007;11: 99–100.
126. Forbes B, Cox M, Christian C. Retinal hemorrhages in patients with epidural hematomas. J AAPOS 2008;12:177–80.
127. Binenaum G, Forbes B, Raghupathi R, et al. An animal model to study retinal hemorrhages in nonimpact brain injury. J AAPOS 2007;11:84–5.
128. Brown S, Levin A, Ramsey D, et al. Natural animal shaking: a model for inflicted neurotrauma in children? J AAPOS 2007;11:85–6.
129. Emerson MV, Jakobs E, Green WR. Ocular autopsy and histopathologic features of child abuse. Ophthalmology 2007;114:1384–94.
130. Gardner H. Retinal folds. Arch Ophthalmol 2007; 125:1142.
131. Lantz PE. Postmortem detection and evaluation of retinal hemorrhages. Abstract, presented at the AAFS Annual meeting. Seattle, Washington, February, 2006. Am Acad Forens Sci 2006.
132. Goldsmith W, Plunkett J. Biomechanical analysis of the causes of traumatic brain injury in infants and children. Am J Forensic Med Pathol 2004;25: 89–100.
133. Jenny C. Committee on Child Abuse and Neglect. Evaluating infants and young children with multiple fractures. Pediatrics 2006;118:1299–303.
134. Bishop N, et al. Unexplained fractures in infancy: looking for fragile bones. Arch Dis Child 2007;92: 251–6.
135. Kleinman P. Problems in the diagnosis of metaphyseal fractures. Pediatr Radiol 2008;38(Suppl 3): S388–94.
136. Keller K, Barnes P. Rickets vs. abuse: a national and international epidemic. Pediatr Radiol 2008; 38:1210–6.
137. Keller KA, Barnes PD. Rickets vs. abuse – the evidence: reply to editorial commentaries. Pediatr Radiol 2009;39:1130.
138. Ganesh A, Jenny C, Geyer J, et al. Retinal hemorrhages in type I osteogenesis imperfecta after minor trauma. Ophthalmology 2004;111:1428–31.
139. Groninger A, Schaper J, Messing-Juenger M, et al. Subdural hematoma as clinical presentation of osteogenesis imperfecta. Pediatr Neurol 2005;32: 140–2.
140. Strauss K, Puffenberger E, Robinson D, et al. Type I glutaric aciduria, part 1: natural history of 77 patients. Semin Med Genet 2003;121C:38–52.
141. Nassogne MC, Sharrad M, Hertz-Pannier L, et al. Massive subdural haematomas in Menkes disease mimicking shaken baby syndrome. Childs Nerv Syst 2002;18:729–31.
142. Ernst L, Sondheimer N, Deardorff M, et al. The value of the metabolic autopsy in the pediatric hospital setting. J Pediatr 2006;148:779–83.
143. Brousseau T, Kissoon N, McIntosh B. Vitamin K deficiency mimicking child abuse. J Emerg Med 2005;29:283–8.
144. Rooms L, Fitzgerald N, McClain KL. Hemophagocytic lymphohistiocytosis masquerading as child abuse. Pediatrics 2003;111:636–40.
145. Liesner R, Hann I, Khair K. Non-accidental injury and the haematologist: the causes and investigation of easy bruising. Blood Coagul Fibrinolysis 2004;15(Suppl 1):S41–8.
146. Roach E, Golomb M, Adams R, et al. Management of stroke in infants and children. Stroke 2008;39: 2644–91.
147. Carvalho KS, Bodensteiner JB, Connolly PJ, et al. Cerebral venous thrombosis in children. J Child Neurol 2001;16:574–85.

148. Fitzgerald KC, Williams LS, Garg BP, et al. Cerebral sinovenous thrombosis in the neonate. Arch Neurol 2006;63:405–9.
149. DeVeber G, Andrew M, Group CPISS. Cerebral sinovenous thrombosis in children. N Engl J Med 2001;345:417–23.
150. Barnes C, deVeber G. Prothrombotic abnormalities in childhood ischaemic stroke. Thromb Res 2006; 118:67–74.
151. Sebire G, Tabarki B, Saunders D, et al. Cerebral venous sinus thrombosis in children: risk factors, presentation, diagnosis, and outcome. Brain 2005;128:477–89.
152. Krasnokutsky M, Barnes P. Cerebral venous thrombosis: a mimic of nonaccidental injury. Scientific Paper Session. Miami (FL): Society for Pediatric Radiology; 2007.
153. Menge T, Hemmer B, Nessler S, et al. Acute disseminated encephalomyelitis. An update. Arch Neurol 2005;62:1673–80.
154. Moritani T, Smoker W, Sato Y, et al. Diffusion-weighted imaging of acute excitotoxic brain injury. AJNR Am J Neuroradiol 2005;26:216–28.
155. Yazbak F. Multiple vaccinations and the shaken baby syndrome. National Vaccine Information Center. The Vaccine Adverse Event Reporting System (VAERS) of the Center for Disease Control and Prevention (CDC) and the Food and Drug Administration (FDA). Available at: www.nvic.org/doctors_corner/ed_yazbak_shaken-baby_syndrome.htm. Accessed September 12, 2010.
156. Innis M. Vaccines, apparent life-threatening events, Barlow's disease, and questions about Shaken baby syndrome. J Am Phys Surg 2006; 11:17–9.
157. Clemetson CA. Is it "shaken baby," or Barlow's disease variant? J Am Phys Surg 2004;9:78–80.
158. Clemetson CA. Caffey revisited: a commentary on the origin of "shaken baby syndrome". J Am Phys Surg 2006;11:20–1.
159. Marinetti L, Lehman L, Casto B, et al. Over-the-counter cold medications—postmortem findings in infants and the relationship to cause of death. J Anal Toxicol 2005;29:738–43.
160. Geddes J, Talbert D. Paroxysmal coughing, subdural, and retinal bleeding: a computer modeling approach. Neuropathol Appl Neurobiol 2006;32:625–34.
161. Talbert D. The sutured skull and intracranial bleeding in infants. Med Hypotheses 2006;66:691–4.
162. American Academy of Pediatrics red book online. Pertussis. 2003:472. Available at: http://www.aapredbook.aappublications.org/cgi/content/full/2003/1/3.9. Accessed September 12, 2010.
163. Surridge J, Segedin E, Grant C. Pertussis requiring intensive care. Arch Dis Child 2007;92:970–5.
164. CDC National Immunization Program. General pertussis information. 2000:2. Available at: http://www.cdc.gov/doc.do/id/0900f3ec80228696. Accessed September 12, 2010.
165. Page M, Jeffery H. The role of gastro-oesophageal reflux in the aetiology of SIDS. Early Hum Dev 2000;59:127–49.
166. Mohan P. Aspiration in infants and children. Pediatr Rev 2002;23:330–1.
167. Barnes P, Galaznik J, Krasnokutsky M, et al. CT in infant dysphagic choking acute life threatening event (ALTE – a mimic of child abuse). Scientific Session. Scottsdale (AZ): Society for Pediatric Radiology; 2008.
168. Zimmerman RA, Bilaniuk LT, Bruce D, et al. Interhemispheric acute subdural hematoma. A computed tomographic manifestation of child abuse by shaking. Neuroradiology 1979;16: 39–40.
169. Cohen RA, Kaufman RA, Myers PA, et al. Cranial computed tomography in the abused child with head injury. AJNR Am J Neuroradiol 1985;6:883–8.
170. Bird CR, McMahan JR, Gilles RH, et al. Strangulation in child abuse: CT diagnosis. Radiology 1987; 163:373–5.
171. Hymal KP, Rumack CM, Hay TC, et al. Comparison of intracranial CT findings in pediatric abusive and accidental head trauma. Pediatr Radiol 1997;27: 743–7.
172. Ewings-Cobbs L, Prasad M, Kramer L, et al. Acute neuroradiologic findings in young children with inflicted or noninflicted traumatic brain injury. Childs Nerv Syst 2000;16:25–33.
173. Barnes PD, Robson CD. CT findings in hyperacute nonaccidental brain injury. Pediatr Radiol 2000;30: 74–81.
174. Wells R, Vetter C, Laud P. Intracranial hemorrhage in children younger than 3 years. Arch Pediatr Adolesc Med 2002;156:252–7.
175. Wells R, Sty J. Traumatic low attenuation subdural fluid collections in children younger than 3 years. Arch Pediatr Adolesc Med 2003;157:1005–10.
176. Stoodley N. Neuroimaging in non-accidental head injury: if, when, why and how. Clin Radiol 2005; 60:22–30.
177. Duhaime AC, Christian C, Armonda R, et al. Disappearing subdural hematomas in children. Pediatr Neurosurg 1996;25(3):116–22.
178. Tung GA, Kumar M, Richardson RC, et al. Comparison of accidental and nonaccidental traumatic head injury in children on noncontrast computed tomography. Pediatrics 2006;118(2):626–33.
179. Slovis TL, Smith WL, Strain JD, et al. Expert panel on pediatric imaging. Suspected physical abuse—child. Reston (VA): American College of Radiology (ACR); 2005 [online].

180. Di Pietro MA, Brody AS, Cassady CI, et al for Section on Radiology; American Academy of Pediatrics. Diagnostic imaging of child abuse. Pediatrics 2009;123:1430–5.
181. Vezina G. Assessment of the nature and age of subdural collections in nonaccidental head injury with CT and MRI. Pediatr Radiol 2009;39: 586–90.
182. Ewing-Cobbs L, Kramer L, Prasad M, et al. Neuroimaging, physical, and developmental findings after inflicted and non-inflicted traumatic brain injury in young children. Pediatrics 1998;102: 300–7.
183. Rooks VJ, Sisler C, Burton B. Cervical spine injury in child abuse: report of two cases. Pediatr Radiol 1998;28:193–5.
184. Chabrol B, Decarie JC, Fortin G. The role of cranial MRI in identifying patients suffering from child abuse and presenting with unexplained neurological findings. Child abuse Negl 1999;23:217–28.
185. Barlow KM, Gibson RJ, PcPhillips M, et al. Magnetic resonance imaging in acute nonaccidental head injury. Acta Pediatr 1999;88:734–40.
186. Suh D, Davis P, Hopkins K, et al. Non-accidental pediatric head injury: diffusion-weighted imaging findings. Neurosurgery 2001;49:309–20.
187. Ichord R, Naim M, Pollack A, et al. Hypoxic-ischemic injury complicates inflicted and accidental traumatic brain injury in young children: the role of diffusion-weighted imaging. J Neurotrauma 2007;24:106–18.
188. Zuerrer M, Martin E, Boltshauser E. MRI of intracranial hemorrhage in neonates and infants at 2.35 Tesla. Neuroradiology 1991;33:223–9.
189. Barkovich A. Pediatric neuroimaging. Philadelphia: Lippincott-Raven; 2005. p. 190–290.
190. Barnes P. Pediatric brain imaging. In: Blickman J, Parker B, Barnes P, editors. Pediatric radiology: the requisites. 3rd edition. Philadelphia: Elsevier; 2009. p. 221–7.
191. Zouros A, Bhargava R, Hoskinson M, et al. Further characterization of traumatic collections of infancy. J Neurosurg 2004;100:512–8.
192. Fullerton HJ, Johnston SC, Smith WS. Arterial dissection and stroke in children. Neurology 2001;57:1155–60.