Evidence-Based Update
Imaging in Nonaccidental Injury and the Mimics

Blood, Brain, & Bones

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CNS Injury (i.e. Triad) – Suspected NAI

**Imaging can not distinguish nonaccidental injury from accidental injury, or from predisposing or complicating medical conditions.**


Skeletal Injury – Suspected NAI

**Imaging can not distinguish nonaccidental injury from accidental injury, or from predisposing or complicating medical conditions, including the Bone Fragility Disorders.**


Shaken Baby Syndrome
The Triad

“Shaken Baby Syndrome (SBS) is a form of physical non-accidental injury (NAI) to infants, characterized by

acute encephalopathy with subdural and retinal hemorrhages (i.e. the Triad),

occurring in the context of inappropriate or inconsistent history and

commonly accompanied by other apparently inflicted injuries (e.g. fractures).”

Shaken Baby Syndrome (SBS)

Battered Child Syndrome / Shaken Baby Syndrome
The “Traditional” Literature

- **Caffey J.** Multiple fractures in the long bones of infants suffering from chronic subdural hematoma. Am J Roentgenol 1946; 56: 163-173.


Child Abuse – NAI

“Traditional” Medical Diagnosis

- Injury out of proportion to history (unwitnessed)
- Tearing or shearing of brain, blood vessels, bone.
- Injuries of varying ages
- RH+ SDH + Encephalopathy (the Triad)
- +/- Skeletal Lesions (the Tetrad)

- Clinical & Imaging Criteria?
- Quality of Evidence?
- Mimics including accidental trauma?
- Forensic Pediatrician?
- Multidisciplinary CPS?
Evidence-Based Medicine
Quality of Evidence (QOE)
Diagnosis or Prognosis

- **Class I**: Prospective study; broad case spectrum v. controls; gold standard; blinded evaluation; tests for diagnostic accuracy.
- **Class II**: Prospective study, narrow case spectrum; Retrospective study broad case spectrum; gold standard; controls; blinded evaluation; tests for diagnostic accuracy.
- **Class III**: Retrospective study; narrow case / control spectrum; blinded evaluation;
- **Class IV**: Non-blinded evaluation; expert opinion alone; descriptive case series; no controls.

Guyatt et al. Users’ guides to the medical literature. XXV. Evidence-based medicine. JAMA 2000;284:1290-1296;
Crosskerry P. The importance of cognitive errors in diagnosis and strategies to minimize them. Acad Med 2003;78:775-780.
Evidence-Based Medicine
Quality of Evidence (QOE)
Recommendations

- **Level A**: Established useful / predictive (or not) for given condition / population; [1 Class I or 2 Class II studies] = Standard.
- **Level B**: Probably useful / predictive; 1 Class II or 3 Class III studies] = Guideline.
- **Level C**: Possibly useful / predictive [2 Class III studies] = Optional.
- **Level D**: Data inadequate or conflicting; Unproven; = Contraindicated.

INFORMED CONSENT

Crosskerry P. The importance of cognitive errors in diagnosis and strategies to minimize them. Acad Med 2003;78:775-780.
Evidence-Based Medicine
Shaken Baby Syndrome
Quality of Evidence (QOE)

• Few published reports merit a rating above class IV.

• Class IV: test not applied in blinded fashion; expert opinion alone; descriptive case series without controls.

• Not basis for standards or guidelines.

• Inconsistent Diagnostic Criteria; Faulty Inclusion Criteria; Circular Logic; Conviction and Confession based.


Rules of Evidence – Standards for Admissibility of Expert Testimony

- **Frye Standard** - testimony generally accepted in the relevant scientific community.

- **Daubert Standard (Kumho)** - assessment of scientific reliability of testimony.

- **Civil Action** - money at risk; “preponderance of evidence”.

- **Criminal Action** - life or liberty; due process; innocent until proven guilty “beyond a reasonable doubt” vs. “clear and convincing evidence”; constitutional right to confront accusers; “burden of proof” on the prosecution.

- **SBS cases** – Expert defines SBS / NAI as presence of injury (e.g. the Triad) without sufficient historical explanation; unconstitutionally shifts burden to defendant to prove the expert theory wrong.

Texas v. Hurtado (Daubert), Udashen, Sperling 2006.
Lyons. SBS. Utah Law Rev 2003;1109.
Head Injury in NAI Prosecution

- SDH + RH + Encephalopathy = SBS / NAI  [the “Triad”]
- Shaking alone in otherwise healthy child can cause SDH leading to death.
- Never due to short fall.
- Immediately symptomatic, i.e. “no lucid interval.”
- Symptoms in child with prior head injury = newly afflicted injury and not spontaneous rebleed (e.g. benign extracerebral collections).
- Last caretaker always guilty (“Unwitnessed”).
3mM Acute Life Threatening Event (ALTE)
Interhemispheric SDH +/- Edema
= SBS / NAI (Myth?)
Tearing of Veins & Brain

8mF Macrocephaly
SDHs of Varying Age = Multiple Inflicted Injuries (Myth?)
Tearing of Veins & Brain

8mM ALTE
Hyperacute right SDH = NAI (Myth?)
Tearing of Brain & Veins

Head Injury in NAI Prosecution

- Shaking alone in otherwise healthy child may cause SDH leading to death.
- Otherwise, such injury requires:

  “Force equivalent to motor vehicle accident or a 2-story fall.”

  \[ F = MA \]

  Force = Mass x Acceleration
Evidence Base
Shaken Baby Syndrome
Biomechanics - The Brain

- Ommaya A. Whiplash injury and brain damage. JAMA 1968; 204: 75-79.
- Luck JF, Prange M, Nightingale RW, Loyd A, Dibb A, Ottaviano D, Tran L, Myers BS. Tensile mechanical properties of the pediatric human osteoligamentous cervical spine. Injury & Orthopaedic Biomechanics Research Laboratory, Department of Biomedical Engineering. Duke University, Durham, NC, USA. (9979 5.3 Spine Kinematics and Injury Biomechanics S151).
“Our evidence, both direct and circumstantial, indicates that manual whiplash shaking of infants is a common primary type of trauma in the so called Battered Infant Syndrome”.

“Current evidence, though manifestly incomplete and largely circumstantial, warrants a nationwide educational campaign on . . the potential pathogenicity of habitual . . shaking of infants . .”

[Caffey cites Guthkelch]

Child Abuse Centers Established (e.g. Kempe, Chadwick)

Caffey. The whiplash shaken infant syndrome: manual shaking by the extremities with whiplash-induced intracranial and intraocular bleedings, linked with residual permanent brain damage and mental retardation. Pediatrics 1974;54; 1.
“This suggests that in some cases repeated acceleration-deceleration rather than direct violence is the cause of the haemorrhage, the infant having been shaken rather than struck by his parents”.

[Guthkelch cites Ommaya]

Rear-end auto collision simulation.
Adult rhesus monkeys on a sled.
Measure angular accelerations head on neck, without head impact.
Results: Brain injury -19; Neck injury-11.
40g Threshold for intracranial injury (concussion, subdural hemorrhage, shear injury).
Caffey & Guthkelch not realize such injury thresholds may not be attained in SBS.

Ommaya, Whiplash injury and brain damage. JAMA 1968;204:75.
Duhaime et al 1987 SBS

1 month old infant ATD model subjected to adult shakes and impacts.

“All shakes (11g) fell below injury thresholds . . , while impacts (52g) spanned concussio, subdural hematoma, and diffuse axonal injury ranges.”

“Severe head injuries commonly diagnosed as shaking injuries require impact to occur and that shaking alone in an otherwise normal baby is unlikely to cause the shaken baby syndrome.”

Autopsy series: all fatal cases (13) had signs of blunt head impact (more than half noted only at autopsy); all with uncontrollable increased intracranial pressure.

“Shaken-Impact Syndrome”


1.5 month old infant model subjected to minor falls, shakes, inflicted impacts.

“In general, peak angular acceleration and maximum change in angular velocity increased with increasing fall height and surface hardness”.

“These findings suggest that inflicted impacts against hard surfaces may be more frequently associated with clinically significant inertial brain injuries than vigorous shaking or falls from less than 1.5-m”.

“In addition, there are no data showing that the peak angular acceleration and maximum change in angular velocity of the head experienced during shaking and inflicted impact against unencased foam is sufficient to cause SDHs or TAIIs in an infant”.

Chris Van Ee Ph.D. Design Research Engineering (www.dreng.com);


Thus, while it is possible to produce trauma in an infant by shaking, e.g. SDH, particularly when shaking is prolonged and repeated at intervals, the injuries would include the cervical cord and spine, but not the brain case, nor contusions in the cerebrum or cerebellum if no impact was also imposed.


“Head acceleration and velocity levels commonly reported for SBS generate forces that are far too great for the infant neck to withstand without injury . . and can potentially cause severe, if not lethal, spinal cord or brain stem injury . . at levels well below those reported for the SBS.”

Biomechanical Evidence Base
Conclusions & Recommendations

- **Shaking** may *theoretically* cause brain injury if associated with *cervical spinal cord injury*.

- **Short-distance falls** (or any impact, accidental or NAI) can produce brain injury.

- In addition to *fall height, impact surface and type of landing* are important factors.

- **Head-first impacts** in young infants are the most dangerous.

- Should always do both **Brain and Cervical Spine CT, as well as MRI (STIR)**.

- Imaging may not distinguish accidental from nonaccidental injury?
2 yM SCIWORA: Partial High Cervical Cord Transection, SAH, SDH, RH, and Hypoxic-Ischemic Brain Injury (CT, Path, Biomech).

Evidence Base
Head Injury in NAI
Neuropathology

Geddes et al – Neuropathology of Inflicted Head Injury
“Young Infant”

- 37 / 53: infants < 9 mo. age
- Shaken only 8 (1 admission).
- Apnea, respiratory distress (ALTE)
- **Old injury (15)**
- Retinal Hem’s, fracture, **thin SDH**
- Increased ICP / swelling
- **Hypoxic-ischemic axonal brain injury (no TAI !!)**
- **Traumatic axonal brainstem / cord injury (impact in all)**

Geddes J et al. Dural Haemorrhage (DH) in Non-traumatic Infant Deaths

- Autopsy series
- 50 cases without trauma
- < 5 months of age
- Infection - 6
- Hypoxia-ischemia – 26
- Infection + HI – 8
- SIDS – 4
- C/W SBS – 3

Geddes et al. Dural Vascular Plexus Haemorrhage (DH) in Non-traumatic Infant Deaths

- **Common Features:** severe hypoxia-ischemia, cerebral venous hypertension, arterial hypertension, brain swelling, venous immaturity / fragility and increased permeability.

- **Result:** intracranial venous dural hemorrhage [+ retinal hemorrhage].

- **Also,** hypoperfusion followed by reperfusion, especially via dural arterial supply with dural hemorrhage (no blood-brain-barrier).

- **Coagulopathy.**

- **“The Unified Hypothesis”** (i.e. the **Cascade**).


Dural Border Cell Layer/Subdural Compartment & Dural Vascular Plexus

- The **DBC** is approximately 8 microns thick
- The **DBC** is the "weakest link" in dural/arachnoid interface due to scarcity of tight junctions and prominent interstitial spaces.

Haines, 1993
Dural Vascular Plexus Haemorrhage vs. Bridging Vein Rupture in Non-traumatic Infant Deaths

Dural Vascular Plexus Hemorrhage in Hypoxia-ischemia. Courtesy Julie Mack, Hershey Medical Center, Penn State Univ.

Hypoxia-ischemia (Reperfusion), Venous Hypertension, Vascular Fragility, Coagulopathy = Edema + Thin SDH.


Dural Vascular Plexus Hemorrhage / Hygroma (DH)
Julie Mack, Hershey Medical Center

Fetal

Neonatal

BECC

Spontaneous Intracranial Hypotension

Osteogenesis Imperfecta

GA1
Acute Life Threatening Event
ALTE


• Apnea – Central, Obstructive, Mixed.
Differential Diagnosis of ALTE


Gastrointestinal (33%)

- Gastroesophageal reflux
- Gastroenteritis
- Esophageal dysfunction
- Colic
- Surgical abdomen
- Dysphagia
Differential Diagnosis of ALTE

Neurologic (15%)

- Seizure
- Central apnea/hypoventilation syndromes (apnea of prematurity, Ondine’s curse)
- Head injury (intraventricular hemorrhage, subarachnoid hemorrhage)
- Meningitis/encephalitis
- Hydrocephalus
- Brain Tumor
- Neuromuscular disorders
- Vasovagal reaction
- Congenital malformation of the brainstem
Differential Diagnosis of ALTE

Respiratory (11%)
- Respiratory syncytial virus
- Pertussis
- Aspiration pneumonia
- Other lower or upper respiratory tract infection
- Reactive airway disease
- Foreign body

Otolaryngologic (4%)
- Laryngomalacia
- Subglottal and/or laryngeal stenosis
- Obstructive sleep apnea
Differential Diagnosis of ALTE

Cardiovascular (1%)
- Congenital heart disease
- Cardiomyopathy
- Cardiac arrhythmia/prolonged QTc
- Myocarditis

Metabolic/endocrine
- Electrolyte disturbance
- Hypoglycemia
- Inborn error of metabolism

Other infections
- Sepsis
- Urinary tract infections
Differential Diagnosis of ALTE

Child maltreatment syndrome
- Shaken Impact syndrome
- Intentional suffocation
- Munchausen-by-proxy syndrome

Other diagnoses
- Physiologic event (periodic breathing, acrocyanosis)
- Breath-holding spell
- Choking
- Drug or toxin reaction
- Unintentional smothering
- Anemia
- Hypothermia
- Idiopathic ALTE/apnea of infancy (23%)
Acute Life Threatening Event (ALTE)
Dysphagic Apnea - Choking


DH + RH in Pertussis

American Academy of Pediatrics Red Book Online: Pertussis. 2003 (see also The Centres for Disease Control & Prevention website)
6 w/o male Macrocephaly, Dysphagic Choking ALTE, HIE, & Coagulopathy

5w infant with Triad and alleged NAI; also, “cold” symptoms, vitamin D undersupplemented, acute choking episode during feeding, & status epilepticus. Chest film (a) shows bilateral lung opacities. CT (b-c) plus T2* MRI (d) shows bilateral cerebral edema with bilateral thin, acute-subacute hemorrhages (or thromboses) about the falx, tentorium, and convexities. Vertex CT (e) shows suture diastasis vs. pseudodiastasis (arrows; craniotabes?). DWI (f) shows global hypoxic-ischemic injury. Later CT (g) shows atrophy and chronic SDH.
6m 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, The Wisconsin Innocence Project
Dural Hemorrhage & Hypoxia-Ischemia

Mimics
Alleged NAI with Triad: Infection with Dural & Cortical Venous Sinus Thrombosis with Dural Hemorrhage & Retinal Hemorrhage

Mimics
Alleged NAI: Dural Venous Sinus Thrombosis with infarction, DH, & RH.

- The Canadian Pediatric Ischemic Stroke Registry.
- Incidence of the disorder 0.67 case per 100,000 children per year.
- Neonates were most commonly affected.
- Fifty-eight percent of the children had seizures, 76 percent had diffuse neurologic signs, and 42 percent had focal neurologic signs.
- Risk factors included head and neck disorders (in 29 percent), acute systemic illnesses (in 54 percent), chronic systemic diseases (in 36 percent), and prothrombotic states (in 41 percent).
- Venous infarcts occurred in 41 percent of the children.
- Neurologic deficits were present in 38 percent of the children, and 8 percent died; half the deaths were due to sinovenous thrombosis.
- Predictors of adverse neurologic outcomes were seizures at presentation and venous infarcts.
- Sinovenous thrombosis in children affects primarily neonates and results in neurologic impairment or death in approximately half the cases. The occurrence of venous infarcts or seizures portends a poor outcome.
20 m infant with Triad & Alleged NAI. Left SDH with cerebral cortical and pial AVM at autopsy. CT (a,b) shows left mixed-density SDH & SAH (long arrows) plus interhemispheric hemorrhage (short arrows) with marked left cerebral swelling and shift.
9mF with Triad & alleged NAI; also, recent fall & coagulopathy (later confirmed platelet disorder). Initial CT (a) shows mixed-density right SDH with right cerebral edema. Postoperative CT 5 days later shows other cerebral & intraventricular hemorrhages. T1 MRI (c) 11 days postoperatively shows evolving right cerebral high-intensity cortical injury & hemorrhages.
Home-delivered newborn with seizures at 1 week of age; also, no vitamin K given at birth. T1 (a) & T2 (b) MRI show acute-subacute left SDH (long arrows) plus right cerebral hemorrhage (short arrows); vitamin K deficiency confirmed & treated.
Hematology


In cases of suspected non-accidental injury in children, it is vital that a haematologist confirms the presence or absence of a haemostatic disorder so that the child welfare and legal systems can make accurate judgements regarding the cause of isolated injuries.

The present paper will discuss commonly used methods for the diagnosis of coagulation disorders in children, and will describe how the investigation of easy bruising and bleeding can be highly problematic.

For instance, some frequently used tests for the assessment of haemostasis in children are insensitive, inappropriate, or based on values derived from adult populations.

Furthermore, artefact is a frequent problem, and many cases present with a negative family history of bleeding.

Crucial to eliminate coagulation factor deficiencies (including factor XI and factor XIII), vWD, platelet disorders (including thrombocytopenia and leukaemia), and rare conditions such as £E2-antiplasmin deficiency. The tests to eliminate these conditions alone incur considerable expense and do not include measures to eliminate other potentially relevant disorders, such as plasminogen activator inhibitor-1 or vitamin C deficiencies.
Presumed NAI with Triad: Pneumococcal Meningitis with DH+RH
Delayed Edema + Herniation
Presumed NAI with Triad
1yM Glutaric Acidopathy
Type 1
12m infant with triad & alleged NAI. Glutaric Acidopathy Type 1. CT (a) & T2 MRI (b) show bilateral SDH of varying age (long arrows), wide sylvian fissures, plus basal ganglia and cerebral white matter abnormalities (short arrows).
16m with Triad (right RH) and alleged NAI; also, short fall with right scalp impact. CT (a) shows left sylvian arachnoid cyst (c) and right hyperacute SDH (arrows). T2 MRI (b) 2 days later shows acute right SDH (long arrows) and smaller left sylvian arachnoid cyst (c) with subdural hygroma (short arrows).
Neuropathology + Biomechanical Evidence Base
Conclusions

• **Shaking** may *theoretically cause* brain injury if associated with *cervical spinal cord injury*.

• **Impact** may produce direct or indirect brain injury (accidental or NAI).

• **Brain edema with thin SDH** (dural vascular plexus origin) may reflect *Hypoxia-Ischemia + Cascade* (accidental or NAI).

• **Brain edema with thin SDH** may result from medical causes (e.g. *Hypoxia-Ischemia + Cascade*) from any cause of ALTE).

• Should always do both **Brain and Cervical Spine CT, as well as MRI**.

• Imaging may not distinguish accidental from nonaccidental injury, or from predisposing or complicating medical conditions.
Evidence Base for Short Falls & Lucid Intervals - “Sutures!”
Malignant Edema with Fatal Progression

Malignant Edema
Lucid Intervals vs. Fatal Progression

Bruce et al (1981) Accidental & NAI:
• Higher GCS (>8) subgroup, 8 / 14 lucid interval; all complete recovery.
• Lower GCS (<8) subgroup, 34 with immediate / continuous coma, 15 lucid interval, 6 deaths, and 11 with permanent disability.


Steinbok et al (2006) witnessed accidental injury:
• 5 children (4 < age 2yr.; 3 falls)
• SDH and cerebral edema by CT 1-5 hours post-event.
• Immediate coma and rapid progression to death.

Significant head injury, including death, may result from low fall levels (or any Impact, accidental or NAI).

Such injury may be associated with a lucid interval (i.e. caretaker blamed for delay).

The lucid interval invalidates the premise that the last caretaker is always responsible in alleged NAI.

In other cases, the injury may result in immediate deterioration with malignant edema & progression to death.

Predispositions including Genetic?

Imaging may not distinguish nonaccidental from accidental injury.
Short Fall - Malignant Edema with SDH

Short Fall with SDH, Malignant Edema
7mM Macrocephaly
CT & MRI
Benign Extracerebral Collections of Infancy
[ BECC, BESS, BEH ]
Evidence Base
Benign Extracerebral Collections (BECC)
SDH - Rebleed

8mf Macrocephaly & Seizure
BECC vs. Chronic SDH with Re-hemorrhage vs. Acute Subdural Hygroma + Hematoma
5m infant with the Triad and alleged NAI; also, macrocephaly from birth, recent seizure but “no” trauma. CT (a) and T2* MRI (b) show large extracerebral collections with smaller recent hemorrhages (arrows). CT 3 months post-drainage (c) shows rehemorrhage (arrows). Diagnosis: BECC or chronic SDHG with rehemorrhage?
Evidence Base for SDH with Rebleed

- Dyer O. Brain Haemorrhage in Babies may not Indicate Violent Abuse. BMJ 2003; 326; 616.
Evidence Base
SDH - Rebleed
Birth Factors

BECC vs. SD Hygroma at birth (a) with SDH vs. rehemorrhage one month later (b).

BECC vs. SDHG at birth (a - long arrows) with SDH vs. rehemorrhage one month later (b- short arrows) on axial FLAIR MRIs (a,b). Courtesy Veronica J. Rooks, MD, Tripler Army Medical Center, Honolulu HI.
Alleged NAI with Triad: 9 w/o Birth Trauma
Alleged NAI with Triad: 9 w/o Birth Trauma
9w infant with Triad and alleged NAI; also, history of traumatic labor and delivery. Skull film (a), CT (b) plus FLAIR (c), T2 (d), T1 (e) MRI show bilateral skull fractures with left growing fracture (long white arrows), chronic bifrontal cerebral white matter clefts (short white arrows-c), plus acute, subacute, & chronic subdural hemorrhages / rehemorrhages (black arrows).
Evidence Base
Conclusions and Recommendations

• **Re-hemorrhage** may occur in an old SDH without recent trauma and be associated with a **lucid interval** (Sutures !!).

• **SDH** occurs in **benign extracerebral collections**.

• **Old SDH** may date back to **Birth**.

• **Serial head circumference** measurements, caregiver education, preventive measures, attention to nonspecific symptoms, early imaging “before the crash”.

• **Imaging** may not distinguish nonaccidental injury from accidental injury.
Head Injury in NAI - Prosecution
Retinal Hemorrhage (RH) only occurs in SBS / NAI.

Evidence-Based Challenges

- **Tongue A.** The ophthalmologists role in diagnosing child abuse. Ophthalmology 1991;98;1009-1010.
Head Injury in NAI - Prosecution
Retinal Hemorrhage only occurs in SBS / NAI.
Evidence-Based Challenges

Evidence-Based Challenges to SBS / NAI as the only cause for the “Triad”

- Plunkett 2001. RHs in 2/3 of the fatal accidental head injuries.
- Gilles 2003. RHs reflect increased intracranial pressure after head injury.
- Lantz 2004. RH with perimacular folds in a case of crush injury to an infant’s head.
- Forbes. AAPOS 2007. RHs in 60% of accidental infant EDH.
- Obi. AAPOS 2007. RHs, schisis, folds in both AI & NAI.
- Binenbaum. AAPOS 2007. No RHs in 3-5 day old piglets subjected to rotational acceleration / deceleration 40x inflicted ‘shaking’ reported by Prange 2003.
- Emerson. 2007. Finds no support for the vitreous traction hypothesis for RH.
- Gilliland. MGF 2006. Use of the triad of scant SDH, brain swelling, and retinal hemorrhages to diagnose non-accidental injury is not scientifically valid. NAME 2006.


Reactions to the Evidence-Based Challenges

- **AAO** removes SBS Resource Website for revision on RHs June 2007.
- **AAP** revised position statement on RHs to be released Spring 2009.
- **AAP** 2001 position paper expires without renewal.
- **NAME** 2001 position paper expires without renewal.

Am Acad Ophthalmology (AAO). Shaken Baby Syndrome Resources Website.


Evidence Base
Conclusions

- The Triad: RH + SDH + Edema not specific for NAI.
- May occur with accidental trauma.
- May occur with medical conditions.
- Must consider Predisposing Risk Factors.
- Must consider Multifactorial, Synergistic, & Cascade Effects.

Dural +/- Retinal Hemorrhage Differential Diagnosis

- Trauma (AI vs. NAI)
- Hypoxia-Ischemia / Reperfusion
- Parturitional Injury
- Venous thrombosis
- Apnea / Choking /Respiratory Arrest
- Infection & Post-infectious (e.g. vaccinial)
- Status Epilepticus
- Hematologic (Coagulopathies)
- Vitamin Deficiency (C, D, K).
- Metabolic Disorders (e.g. GA1, Menkes)
- Vascular / Connective Tissue Diseases (e.g. OI).
- ECMO
- Congenital Heart Disease
- Cervical Spinal Cord Injury
- Multifactorial / Synergistic (including CPR)
- No Body Knows
Over-the-Counter Cold Medications—Postmortem Findings in Infants and the Relationship to Cause of Death

- 10 deaths in infants under 12 months old.
- Toxicology findings include over-the-counter (OTC) cold medications.
  - Ephedrine, pseudoephedrine, dextromethorphan, diphenhydramine, chlorpheniramine, brompheniramine, ethanol, carbinoxamine, levorphanol, acetaminophen, and the anti-emetic metoclopramide.
- Majority of these deaths were either toxicity from the OTC cold medications directly or as a contributory factor in the cause of death.
- Only two of the cases were the result of possible child abuse.
- Caregiver mistaken notion that OTC cold medications formulated for children are also safe for use in infants.
- OTC cold medications in infants can result in toxicity that can lead to death.
- CDC / FDA Ban.
Child abuse is a terrible crime and the failure to recognize it is unforgivable. An erroneous diagnosis of inflicted head trauma is just as tragic and the resulting destruction of a family is one of the gravest injustices of modern times. Many have recently questioned the existence of the so-called “Shaken Baby Syndrome” and the concept that the last caretaker must have been guilty. Careful reviews often uncover relevant findings that were missed or ignored. Recent pediatric vaccinations have been suspected as precipitating factors. A recent combination of seven antigens is the focus of this investigation.

• VAERS  - Vaccine Adverse Event Reporting System CDC & FDA.
Child Abuse – Nonaccidental Injury (NAI) Recommendations
“A Compassionate Approach”

- Thorough Medical & Forensic Work-Up.
- **Child & Family Protective Evaluation & Management.**
- Timely Imaging – CT, MRI, Skeletal Survey.
- **Differential Diagnosis.**
- Multidisciplinary Approach.
- Research - Genetic / Molecular Predisposition.
- Reframing Neglect / Abuse - Opportunities for Prevention.
- Prenatal Care 84%, Medical Setting Births 99%.
- Early Risk Detection, Management.
- Parent training (e.g. during pregnancy).
- Home visitation.
- Social Support.
- Stop Cost-Shifting from Medical / Social System to Criminal Justice System.
Mandatory Reporting - Suspected NAI
Recommendations

• Should do Brain and Cervical Spine CT.
• Strongly recommend Brain and Cervical Spine MRI (ASAP).
• Provide detailed description of imaging findings.
• **Differential Diagnosis.**
• May not distinguish fractures from suture variants (3DCT?).
• Caution regarding timing estimates, especially CT.
• Direct reporting to the primary healthcare professional.
• Recommendations for further imaging (e.g. skeletal survey).

• Impression: “The imaging findings can not distinguish nonaccidental from accidental injury or from predisposing and complicating medical conditions.”
CT & MRI in Alleged NAI
Limitations

• Barnes P. Imaging of the CNS in Suspected or Alleged NAI. ASPNR Gyrations Newsletter 2007; 2: 5-7 <www.aspnr.org>
Timing of Hemorrhage (Thrombosis)
CT Limitations

- High density (i.e. clotted, acute to subacute) 3 hr. to 7-10 days;
- Not differentiate hemorrhage v. thrombosis (e.g. venous);
- Iso-Hypodense (i.e. nonclotted):
  - Hyperacute (<3 hrs.)
  - Chronic (> 7-10 days)
  - BECC or subdural hygroma (acute or chronic);
- Blood levels unusual in acute stage except coagulopathy.
- Not distinguish acute hemorrhage from re-hemorrhage upon BECC or chronic SDH;
- Interhemispheric SDH not characteristic for NAI;
- Mixed-density SDH may occur in AI.

Hyperacute right SDH vs. chronic SDH with re-hemorrhage?
+/- Unilateral Edema = NAI
(Myth?)

Timing of Hemorrhage (Thrombosis)
MRI Limitations

- **Hyperacute (< 12 hr.):** T1 iso-hypointense, T2 hyperintense;
- **Acute (1-3 days):** T1 iso-hypointense, T2 hypointense;
- **Early Subacute (3-7 days):** T1 hyperintense, T2 hypointense;
- **Late Subacute (7-14 days):** T1 hyperintense, T2 hyperintense;
- **Early Chronic (> 14 days):** T1 hyperintense, T2 hyperintense;
- **Late Chronic (> 1 – 3 months):** T1 iso/hypointense, T2 hypointense.

Timing of Hemorrhage (Thrombosis)  
MRI Limitations

- **Mixed intensity** - problematic regarding timing.
- **Blood Levels** - subacute hemorrhage vs. coagulopathy.
- **Timing guidelines** - sediment not supernatant.
- **CSF Component** - BECC v. acute SDHG v. hyperacute SDH v. chronic SDH, SDHG.
- **GRE** - iron-sensitive but not assist with timing unless matched with T1, T2, and CT.
- **GRE / SWI** - sensitive to venous thromboses (e.g. cortical, medullary, subependymal) that may not detected by MRV.
- **FLAIR** sensitive, but not specific for hemorrhage unless matched with T1, T2, GRE, CT.

BECC vs. Chronic SDH with Re-hemorrhage vs. Acute Subdural Hygroma + Hematoma
Timing of Hemorrhage

Vezina G. Assessment of the nature and age of subdural collections in nonaccidental head injury with CT and MRI. Pediatr Radiol Online 21 March 2009

Density evolution of hemorrhage on CT images.

• Hyperacute: Isodense <3 hours
• Acute: Hyperdense Few hours → 7–10 days
• Subacute: Isodense 2–3 weeks
• Chronic: Hypodense >3 weeks
Timing of Hemorrhage
Vezina G. Assessment of the nature and age of subdural collections in nonaccidental head injury with CT and MRI. Pediatr Radiol Online 21 March 2009

Signal evolution of SDH on MRI.

- **Hyperacute**: <12–24 h \(T1\downarrow\text{ or } T2\uparrow\)

- **Acute**: 1–3 days \(T1\downarrow\text{ or } T2\downarrow\downarrow\)

- **Early subacute**: 2–3 days → 1–2 weeks \(T1\uparrow\ T2\downarrow\downarrow\)

- **Late subacute**: 1–2 weeks → 1–2 months \(T1\uparrow\ T2\uparrow\uparrow\)

- **Chronic**: Few weeks → months/years \(T1\leftrightarrow\ T2\downarrow\downarrow\)

- **Chronic**: Few weeks → months/years \(T1\downarrow\text{ (>CSF) } T2\uparrow\)
CNS Injury (i.e. Triad) – Suspected NAI

**Imaging can not distinguish nonaccidental injury from accidental injury, or from predisposing or complicating medical conditions.**


NAI – Bones
Skeletal Fragility Disorders

• Fractures, often multiple, unexplained
• Ribs, CMLs, etc.
• Varying ages
• Skeletal Survey
• DXA scan
• SOS Ultrasound
• QCT

3m 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) & after (c) vitamin D supplementation show “healing” CML (arrows)?


Radiographic Findings
Congenital Rickets
Skull

Radiographic Findings
Congenital Rickets
Facial Bones - Pre & Post Vitamin D Therapy

Radiographic Findings of Congenital Rickets
Looser’s Zones + Metaphyseal Changes (2m, 4m, 15m)

Radiographic Findings of Congenital Rickets
Wrist Changes Pre & Post Vitamin D Therapy

Radiographic Findings of Congenital Rickets
Lower Extremity (distal tibial tilt)

Skeletal Injury – Suspected NAI

Imaging cannot distinguish nonaccidental injury from accidental injury, or from predisposing or complicating medical conditions, including the Bone Fragility Disorders.


**Vitamin D Deficiency – Congenital Rickets**


Evidence-Based Update
Brain Imaging in Nonaccidental Injury and the Mimics

Blood, Brain, & Bones

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Case # 1

- 19 y au pair phones 911: 8 m/o boy in resp. distress 3y/o sibling boy also at home.
- Mother and father physicians at work.; EMT responds, CPR, intubation unsuccessful.
- ER: Child unresponsive, posturing, bag ventilation. Pupils fixed, dilated. Extensive RH / macular folds. No signs of trauma; No spontan. movements, increased tone.
- IV access, intubation; CBC, clotting studies, blood gases normal; Negative prior medical history; No history of trauma; Child irritable that day; A little “rough” handling by au pair.
- CT: mixed density right convexity and interhemispheric SDH, edema, herniation, right occipital skull fracture, scalp swelling?; No MRI; Skeletal Survey - healing right distal radius fracture 2-4 wks. old.
- Emergency craniotomy for hyperacute SDH; Ventilator dependant infant dies 5 days later; Postmortem confirms fracture, SDH, infarctions.
- Au pair charged with first degree murder, in custody >1 year. Battle of experts at trial - new injury vs. old injury with rebleed (not admissible: child fell out of shopping cart on to head 1 mo. PTA; sibling with # arm fractures in past).
- Au pair convicted of manslaughter, sentenced to time served, and released.

[Baby Eappen - Nanny Case]
Case # 1
CT: Hyperacute / Acute SDH (white arrows).

Case # 2

- 41 y/o physician father phones 911 (social worker mother at dentist); 5 m/o girl (29 wk, premi, IVF; DPT-2, HIB-2, 1 wk PTA, fussy); respiratory distress, shaken, CPR, EMT response;
- ER: status Sz, intubated; High WBC, low HCT; abnormal PTT, fibrinogen; Bilateral RH with fibrosis (ROP);
- CT: asymm. cerebral low Ds, loss of GWD, and enhancement; high Ds + enhancement IHF / falx / tent. / DVS; high and low density SA and SD collections;
- CTV: nonopacified segments of SSS, nonopacified adjacent cortical veins (hemorrhages, thromboses, infarctions, subdural collections, communicating hydrocephalus, meningoencephalitis / venous thrombosis); no MRI; skeletal survey negative;
- Brain death at 3 days; death at 3 wk; autopsy: normal neck, cx spine; no skeletal injuries; neuropath – widespread postmortem necrosis & small hemorrhages, perivascular inflammatory cellular infiltration, venous thrombosis, meningeal fibrosis;
- Appeal, conviction, appeal.

[Baby Scoon Case]
Case # 2

**Dural Venous Sinus Thrombosis.** Hemorrhages / thromboses (a), enhancing cortical infarctions (b), & thrombosis superior sagittal sinus (c,d).

Cerebral Venous Thrombosis as a Mimic of NAI (Pediatr Radiol; Rejected 1999)