

PART 4 -- "TIME OF INJURY -- HEAD" through "HEADBANGING AND SELF-MUTILATION"

TIME OF INJURY -- HEAD

(ALSO CALLED "LUCID INTERVAL")

See also "Bruises aging." See also under "Shaken." For a summary, see Reichert & Schmidt in Lazoritz & Palusci's SBS book (2001) at 81-82. (below).

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. Caffey J, Pediatrics 1974 Oct; 54(4): 396-403. See also 1972a. Caffey states that these shaking incidents are usually repetitive: "repeated during long periods," "in paroxysms...repeated...over periods of days or weeks." 401 Caffey takes account of the obvious difference in force between manual shaking and an automobile accident by saying this: "It is obvious that although the single manual shake of an infant may be less forceful and pathogenic than the single whiplash in an automobile accident, the summation of the injurious effects of the many repeated but less forceful manual shakings may be much more harmful..." 401

Diffuse cerebral swelling following head injuries in children: the syndrome of "malignant brain edema." Bruce DA, Alavi A, Bilaniuk L, Dolinskas C, Obrist W, Uzzell B. J Neurosurg 1981 Feb; 54(2): 170-178. Abstract: The commonest CT finding in head-injured children is bilateral diffuse cerebral swelling. Cerebral blood flow and CT density studies suggest that this swelling is due to cerebral hyperemia and increased blood volume, not to edema. The clinical history, course, and outcome of 63 children with this CT pattern are reviewed. 14 children had a GCS of greater than 8; all of these made a complete recovery and followup CT scans were normal. 49 children had GCS of 8 or less. 15 had a history of a lucid interval following the initial unconsciousness. One of these children died of delayed brain swelling; the others recovered well with minimal neurological deficit. 34 children were rendered immediately and continuously unconscious. There was a high incidence of second lesions on the CT scan, 50% of this group developed intracranial htn and five died. All of the others were in coma for period ranging from weeks to months. Followup CT scans showed an extracerebral collection with a density of CSF in 27% of the patients, and ventriculomegaly with large sulci in 35%, whereas this pattern was seen only once in those with a lucid interval. The difference between those with and without a lucid interval is related to the degree of primary diffuse impact injury to the white matter.

Biomechanics of acute subdural hematoma. Gennarelli TA and Thibault LE. J Trauma 1982; 22: 680-686

Diffuse axonal injury due to non-missile head injury in humans: an analysis of 45 cases. Adams et al. Ann Neurol 1982; 12: 557-563. No lucid intervals seen.

Head injury in man and experimental animals: clinical aspects. Gennarelli TA. Acta Neurochir Suppl (Wien) 1983; 32: 1-13. Cited by Reichert & Schmidt in Lazoritz & Palusci's SBS book (2001) for the proposition that "Acute subdural hematoma associated with severe neurological decompensation, cerebral edema, or death occurs in the setting of an injury involving a major mechanical force followed by immediate or rapid onset of neurological symptoms." p. 81.

Delayed deterioration following mild head injury in children. Snoek JW, Minderhoud JM, Wilmink JT. Brain 1984 Mar; 107(Pt 1): 15-36. A series of 42 children is described who, following a seemingly minor

or trivial head injury, developed neurological signs after a lucid or symptom-free period. This group constitutes 4.34 percent of 967 consecutive patients aged 2 months to 17 years who were seen by members of the neurological staff during the years 1978-1981. Only one patient had an intracranial hematoma. The majority of patients showed a benign transient syndrome consisting of either convulsive or nonconvulsive signs with a spontaneous and full recovery. There were, however, 3 deaths in this series, apparently due to severe and uncontrollable unilateral or diffuse brain swelling, demonstrating the malignant counterpart of this benign syndrome. The theories seeking to explain these phenomena are reviewed. Special reference is made to the hypothesis of Bruce and his associates regarding brain swelling as a causative factor. It is considered that an adequate theory to explain the pathogenesis is still lacking. It is concluded that the juvenile brain responds to cranial trauma in a manner different from the adult brain. This implies a different approach in policy on hospital admission. See editorial by David Bruce next below. Reichert & Schmidt (2001) state that this article primarily deals with delayed onset of seizures, followed by recovery.

Delayed deterioration of consciousness after trivial head injury in childhood. (editorial). Bruce DA. Br Med J (Clin Res Ed) 1984 Sep 22; 289(6447): 715-716.

The head-injured child who "talks and dies." Humphreys RP, Hendrick EB and Hoffman HJ. Child's Nervous Syst 1990 May; 6(3): 139-142. The phenomenon of "talking and deteriorating" after closed head injury exists in children. A variety of causes have been identified, few of which are operatively remedial. Four Cases of children with head trauma are reported, in each of whom there was an interval during which the child verbalized to some degree. Rapid neurological decline then occurred approximately 30-50 h postinjury in each child, who subsequently died from their trauma. In all instances the children were injured in motor vehicle accidents or falls, had initial GCS of 9 or better, and demonstrated irritability and restlessness just prior to their deterioration. In no circumstance was a space-occupying intracranial hematoma present. Post-mortem brain examinations in two of the children showed in common multiple cerebral contusions, brain edema with herniation, and HIE.

Head-injured patients who talk and deteriorate into coma: analysis of 211 patients studied with computed tomography. J Neurosurg 1991 Aug; 75(2): 256-261. See editorial, J Neurosurg 1992 Jul; 77(1): 161-162.

Post-traumatic seizures: a critical review. Dalmady-Israel C and Zasler ND. Brain Injury 1993 May-Jun; 7(3): 263-273. Post-traumatic seizures are a well-recognized complication of head injury. However, the issue of seizure risk assessment remains controversial. The authors present a critical review of the literature pertaining to post-traumatic seizures, with particular emphasis on current concepts of definitions, incidence and risk factors. Different methods of risk assessment are reviewed and the possibility of utilizing functional imaging techniques for seizure risk assessment is also explored.

Risk factors in the outcome of children with minor head injury. Hahn YS, McLone DG. Pediatr Neurosurg 1993; 19: 135-142

Patients who talk and deteriorate. Rockswold GL and Pheley PJ. Ann Emerg Med 1993 Jun; 22(6): 1004-1007. Patients who "talk and deteriorate" are defined as those who utter recognizable words at some time after head injury and then deteriorate to a severe head-injured condition (GCS of 8 or less) within 48 hours of injury. They represent a very small but important subgroup of patients with brain injury. In approx 75% of these pts, the cause of this deterioration is intracranial hematoma. Despite the fact that talking indicates nonlethal impact brain injury, deterioration is marker of poor prognosis. Outcome depends on early recognition of deterioration and rapid removal of mass lesions. The challenge for emergency physicians is to distinguish patients at risk for deterioration from the many pts evaluated after head injury.

The time interval between lethal infant shaking and onset of symptoms: a review of the shaken baby syndrome literature. Nashelsky MB, Dix JD. Am J Forens Med P 1995 Jun; 16(2): 154-157. They reviewed the literature up to 1995 and found only three published SBS cases where the time interval from shaking to onset could be determined. Two were immediate, and one took four days. In that case, the child had vomiting during the four days before onset of frank seizures. In that case the authors suspected

reshaking. Conclusion: the medical literature provides few data that would confirm or deny that the onset of symptoms is immediate.

Ken Feldman comments that post-traumatic seizures are much more variable in onset than are apnea, arrest, and LOC. He also suggests reshaking when onset of apnea, arrest, and LOC is delayed. (Personal communication, May 30, 1999)

Abusive head trauma: the relationship of perpetrators to their victims. Starling SP, Holden J, Jenny C. Pediatrics 1995; 95: 259-262. This was a retrospective chart review of 151 Colorado AHT patients aged 3 months to 24 months "to determine the perpetrator of abuse." The perpetrator was identified in 127 cases, with 87 men and 40 women. Caretakers were stratified by level of certainty of being the abuser:

1. confession
2. conviction
3. criminal charges
4. discrepant history

Male perpetrators outnumbered female by more than 2:1. 37% fathers, 20% boyfriends, 17% female babysitters ("a large, previously unrecognized group), mothers 12%. All but one of the *confessed* abusers was with the child at the time of onset of symptoms. [note the qualification: *confessed* abusers. -JKR] The authors signal the point that no prevention efforts have ever been directed at babysitters.

See the same author's 2004 article pursuing the same line of inquiry with confessions. Dr Reece cites this article among others in his review in the Quarterly for winter 2005 of Leestma's 2005 article on shaking confession cases, for the proposition that there have been more published shaking confession cases since 2001 than in all the years up to 2001.

Outcome and prognosis of whiplash shaken infant syndrome: late consequences after a symptom-free interval. Bonnier C, Nassogne MC, Evrard P. Dev Med Child Neurol 1995; 37: 943-956 Increased ICP was present in 55%-85%. Less than 8% had brainstem lesions. (Compare Oehmichen et al., 1998 and Geddes et al., 1998.)

Moderate head injuries in children as compared to other age groups, including the cases who had talked and deteriorated. Ceviker N et al. Acta Neurochir (Wien) 1995; 133(3-4): 116-121. Turkish ER study of 231 pts studied for risk factors for deterioration or death. Found that SAH was the only risk factor that independently predicted deterioration or death. See also ER study by Greenes & Schutzman, 1997.

Abusive head trauma? A biomechanics-based approach Kent P Hymel et al. (above under "Shaken." DAI= immediate LOC.

Infants with isolated skull fracture: what are their clinical characteristics, and do they require hospitalization? Greenes DS, Schutzman SA. Ann Emerg Med 1997 Sep; 30(3): 253-259. See these authors' later studies 1999 and 2001.

Restricting the time of injury in fatal inflicted head injuries Willman K, Bank DE, Senac M, Chadwick DL. Child Abuse & Neglect 1997; 21(10): 929-940. Retrospective chart review of 95 hospital cases of documented fatal head injury, mostly MVA's and bicycle injuries, in which there was a documented pre-hospital course, in ch under 16yrs, avg age 8 yrs age range 3 mos to 16 years. Only 4 cases were under 2. There were no lucid intervals except for one anomalous case of a bicycle EDH who was killed by surgical drill. Authors' conclusion: children with fatal head injury do not have return of consciousness. Fatal HI is severe force usually causing SDH SAH, and precludes consciousness after the event. "Unless an epidural hematoma is present, children who die from blunt head injuries probably do not experience lucid intervals." "The results of this study suggest that a fatal head injury that does not involve an epidural hematoma must have occurred after the last known time that the child exhibited normal behavior." "Excepting cases involving epidural hematomas, the time of injury in a fatal head injury case can be restricted to after the last confirmed period of normal consciousness for the child." "If a history purports a lucid interval, ...that history is likely false and the injury is likely inflicted."

Cerebral edema was evident by CT within 1h and 17 min. Other moral: cerebral edema develops much faster in trauma cases than in anoxia cases, where it has traditionally been said at least 24 h?? See also Dias et al., below (1998) and Oehmichen on axonal injury (1998), below.

There is a letter to the editor from Dr Plunkett and an authors' reply. Dr Plunkett's letter, vol 22, no. 10, pp. 943-944 (1998) says that as to lucid intervals in fatally head-injured infants, as opposed to older children "There is little objective data to resolve this issue. Only one published study (Nashelsky & Dix, 1995) specifically addresses a lucid interval in infantile fatal head trauma. Two additional articles (Howard, Bell & Utley, 1993; Aoki & Masuzawa, 1984) document the lucid interval (or lack thereof) in each of their cases (N=54)..." Dr Plunkett presents the following criticisms of the paper:

1. Head injury in older children is irrelevant to the abusive head trauma area, because the [skull and] brain are different.
2. Lack of an age-matched control group for the under-2 age group.
3. Motor vehicle and bicycle accidents are not biomechanically comparable to abusive head trauma and rotational falls in infants.
4. There has to be return of consciousness in some subdurals. Otherwise one would never see chronic subdurals in ambulatory patients.

Authors' reply:

1. No specific reply
2. We didn't have enough infants because we accessed only one trauma system.
3. Whether MVA's are biomechanically comparable to AHT is unknown, because the exact events in AHT cases are unwitnessed ("are not occurring in a lab.") There are significant similarities in the pathology (subdural hematomas and brain swelling.)
4. Unquestionably there is return of consciousness in some subdurals, but these are nonfatal ones. The subdurals are successfully treated. In fatal subdurals death results from brain swelling, not the subdural. In such severe brain swelling cases, there will be no return of consciousness. The subdural is a marker for brain injury, but the cerebral edema is the brain injury itself. [my paraphrase -JKR]

Head injury -- abuse or accident? Wilkins B. Arch Dis Child (Br.) 1997 May; 76: 393-397. (have)
This is a literature review by an Australian pediatric intensivist. He considers the lucid-interval question in passing, from an ER perspective, stating, under the category of "cerebral injury," "[C]hildren can be deeply unconscious after a minor head injury ... or are not unconscious initially, but develop coma later in the first day with cerebral oedema and intracranial haemorrhage. For example, four children in Toronto had a lucid interval after a minor or moderate head injury but lapsed into coma suddenly and died with cerebral contusions, cerebral oedema, and hypoci-ischaemic encephalopathy. (citing Humphreys, 1990).

Lucid interval in infantile fatal head trauma. (letter) Plunkett J. Child Abuse Negl 1997 Oct; 21(10): 943-946

Chapter: Head Trauma. In: Kleinman PK, *Diagnostic Imaging of Child Abuse, 2d ed.* (1998), p. 319.
"The time required to develop sufficient swelling or edema for detection by CT is not precisely known. Authorities have estimated the time interval between the insult and CT documentation of edema as 12 to 48 hours. (citing Barkovitch, 1995; Zimmerman & Bilaniuk, 1994). In a study of severe accidental head injuries with documented time interval between the injury and CT, Willman and colleagues noted cerebral swelling as early as 1 hour and 17 minutes after injury. In the authors' experience, the CT appearance of cerebral swelling or edema after inflicted head injury is highly variable and depends on the number, timing, severity, and duration of the assault(s); the types of primary and secondary injuries; and the time delay in presentation or arrival at the hospital. Swelling or edema may become apparent on CT within a few hours of the acute event, or may not become visible until 1 or 2 days after the injury... Early CT findings of edema related to primary focal or diffuse brain injury (e.g. contusion, DAI, vascular injury) may consist of focal or multifocal low density and loss of gray-white matter differentiation. This [focal/multifocal] pattern may gradually evolve to a diffuse pattern involving one or both cerebral hemispheres with reductions in the size of the sulci, fissures, ventricles, or cisterns... In a matter of hours, and depending on the

superimposition of edema due to other causes, this pattern evolves or progresses to that of diffuse low density and loss of gray-white matter differentiation...

“There is a distinct tendency for profoundly injured infants to manifest brain edema by CT that primarily involves the cerebral cortex and subcortical white matter but apparently spares the basal ganglia, thalamus, brainstem, and cerebellum. Cohen and colleagues applied the term reversal sign to describe this phenomenon.”

Cerebral complications of nonaccidental head injury in childhood. Gilles EE and Nelson MD Jr. *Pediatr Neurol* 1998; 19: 119-128. Retrospective analysis of 14 ch with severe AHT. None had a lucid interval. See under “Fall vs inflicted.”

Serial radiography in the infant: shaken impact syndrome. Dias MS, Backstrom J, Falk M, Li V. *Pediatr Neurol* 1998; 29: 77-85. A retrospective study of hospital radiographs in 33 shaken babies with a view toward establishing the time-sequence of evolution of the intracranial changes. In 15 cases the time of injury was known. These had SDH in 13, usually scanned avg 2 ½ hrs after injury. 5 of these had a chronic or mixed SDH. Dr. Reece comments that the retrospective nature and the gaps in timing make it hard to get an organized picture of timing from this type of study.

Interval duration between injury and severe symptoms in nonaccidental head trauma in infants and young children. Gilliland MGF. *JFS* 1998 May; 43(3): 723-725. A prospective postmortem study examined the interval between injury and onset of symptoms in 76 head injury deaths. The mechanisms were shake, shaken impact, and impact. The interval was less than 24h in 80% of shakes, 72% of shaken impacts, and 69% of blunt traumas. The interval was greater than 24 hours in more than 25% of each of these latter groups and was more than 72 hours in four children. The variable intervals between injury and severe symptoms warrant circumspection in describing the interval for investigators or triers of fact. It should be noted that in all of the cases in which information was supplied by someone other than the perpetrator, the child was NOT NORMAL during the interval. But lucid interval did occur confirmed by independent observers in some cases.

Non-accidental head injury infants -- the “shaken-baby syndrome.” Duhaime A-C et al., *NE J Med* 1998; 338: 1822-1829. Lucid intervals do not occur in this setting.

Cerebral complications of nonaccidental head injury in childhood. Gilles EE and Nelson MD JR. (see above under “Shaken”). *Pediatr Neurol* 1998 Aug; 19(2): 119-128. Shows the radiographic evolution of infarctions in the brain. 14 pts. None had a lucid interval.

Axonal injury -- a diagnostic tool in forensic neuropathology? A review. Oehmichen M, Meissner C, Schmidt V, Pedal I, Konig HG, Saternus K-S. *Forensic Science International* 1998; 95: 67-83. (have) Did BAPP on 252 brains from closed head trauma, gunshot, hypoxia, exsanguination. Found BAPP + in 65 to 100 % of all groups if they survived more than 3 hours. Never found it if survived less than 3 h. No difference was found between the staining patterns of traumatic and nontraumatic cases. Also no difference between acceleration-deceleration injuries and impact injures.

Minimum post-injury intervals for positivity:

BAPP	3 h
Ubiquitin	6 h
Silver stain	18 h
H & E	24 h
Microglial reaction	4 d

Mark Hansen, Why are Iowa’s babies dying? *ABA Journal*, August 1998, 74-78. See followup article by Barry Siegel in the *Los Angeles Times*, July 11, 1999. State Medical Examiner who erroneously dx’d SBS in several SIDS cases.

Shaken infant syndrome: selected controversies. Krous HF and Byard RW. *Pediatric Developmental Pathology* 1999; 2: 497-498. Brief review of the literature on three controversies:

Lucid interval	no
Rebleeds	nonpathologic
Impact	not necessary

Clinical indicators of intracranial injury in head-injured infants. Greenes DS and Schutzman SA. Pediatrics 1999; 104: 861-867. 608 children under two seen in ER for head trauma. 431 were asymptomatic. 14 of these asymptomatics had an intracranial injury (3%). None of these 14 asymptomatic children with intracranial injury had any subsequent deterioration. (Some of the 14 asymptomatics with intracranial injury had subdurals, but none of them deteriorated or required any specific treatment.) None of the 431 asymptomatic children with history of head injury had any subsequent deterioration. Betty Spivack comments (Child Abuse Quarterly 2000 Mar; vii(2): 11) that this article demonstrates that asymptomatic children are highly unlikely (0/431) to have a subsequent neurological deterioration; or in other words, there is no lucid interval. See also the same authors' 1997 and 2001 papers.

Analysis of missed cases of abusive head trauma. Jenny C, Hymel K, Ritzen A, Reinert S, Hay T. JAMA 1999; 281: 621-626. According to Dr Reece's presentation of the top ten papers of the past ten years at the 2006 National Shaken Baby Conference, defined AHT cases by

- confession
- discrepant history
- associated unexplained injuries
- delayed rescue

Defined "missed" as: child had been seen before with vom, irrit, facial or scalp swel, AMS, respir distress, or sz, AND the cranial imaging showed age of injury consistent with the time of the earlier visit. Had 173 cases. Of these, 54 (31%) were classified as "missed." The total 173 pts had:

SDH	87%
Parenchymal hem	45%
Cerebral contusion or DAI	37%
EDH	2%
RH	66%
Extracranial frac	35%

The average number of prior visits was 2.8, and the average delay of dx was 7 days. The following factors increased the likelihood of missing the diagnosis on the initial visit: young age, white race, less severe symptoms, and an intact family. Dr Reece lists four independent variables that predicted that the diagnosis would be made correctly at the first visit: abnormal respiratory status, sz, face or scalp injuries, and parents not living together. The most frequent erroneous diagnoses were viral gastroe, accidental head injury, rule out sepsis, increasing head size, otitis media, and epilepsy. Of the five deaths in the missed group, four could have been prevented by early diagnosis. 28% of the missed pts were reinjured in the interval.

CT findings in hyperacute nonaccidental brain injury. Barnes PD and Robson CD. Pediatr Radiol 2000; 30: 74-81. Scholarly discussion of the low-density and high-density CT components in infant SDH and contrasts them with the findings of rebleed in adults with chronic SDH. Rebleed has not been shown to occur in infants. Discusses the 1997 au pair case in detail. Wilbur Smith (Child Abuse Quarterly, July 2000) says that this is must reading for AHT.

Shaking-impact syndrome and lucidity. (letter) Byard RW, Donald T, Hilton JN, Krous HF. Lancet 2000 Feb 26; 355(9205): 758. Initially these authors cite the conclusions of Duhaime, Rorke & Zimmerman's 1998 review in the NEJM and Wilman, Bank et al. (1997) that lucid interval does not follow *fatal* head injury involving SDH and cerebral swelling. Also their own observation (SBS: selected controversies, 1999) that "Reliable witnesses of significant infant shakings have also noted an immediate change in conscious state." They state that "[O]ur own observations are that severe had trauma in infants is invariably associated with an immediate alteration in conscious state..." HOWEVER, "there are still unanswered questions." (1) Assessment of consciousness in infants is "extremely difficult." It requires a

trained pediatric expert. This is rarely available in shaking situations either at the event or for some time afterwards. Lay observations are completely unreliable, even apart from the possibility of deception. Furthermore, (2) "Ascertainment of when an infant with a severe inflicted head injury was last neurologically normal may not be possible when the history relies heavily on the veracity of a person who may be, or may be associated with, the perpetrator." Therefore, conclusions based on clinical experience, such as the above-cited scholarship, should not be negated by lay observations in a particular case. "Unfortunately, the uncertainties that persist surrounding the issue of lucidity in infants with inflicted injury mean that we are still left with considerable difficulties when we attempt to plot a time course for events in these serious, complex, and highly emotive cases."

Fatal pediatric head injuries caused by short-distance falls. Plunkett J. Am J Forens Med Path 2001 Mar; 22(1): 1-12. See under "Fall versus inflicted" and "Shaken." Purpose: to refute two commonly-held doctrines: (a) that short falls in children are never fatal, and (b) that there is never a lucid interval after a fatal head injury. Presents 18 cases of toddlers and preschool and school-age children (not infants) who had witnessed short falls, mostly on playground equipment, and many had some delay (15 min to 3 h) in the appearance or recognition of symptoms. Four had considerable delay, one with an EDH and three with SDH's including one large expanding SDH with a 48 hour lucid interval clinically documented. Commentators have pointed out that some of these accidents, including all of those in the youngest age group, were unwitnessed. The only long lucid intervals were in EDH cases or older children with large space-taking SDH's. The purpose of this article was to refute the contention of Williams that short falls never cause fatal head injury and the contention of Wilman that there is never a lucid interval in fatal head injury in peds. (Wilman says "probably" never.) See the comment of Mary Case 2002 Mar limiting the "never a lucid interval" doctrine to *severe or fatal* inflicted head injury.

NAME Position Paper on AHT in infants and young children, 2001. See above under "Shaken." It asserts that ALOC is immediate in DAI/ rotational injury, although apnea may be delayed. "Studies in children dying of *accidental* head injuries indicate that children with diffuse injury show an immediate decrease in the LOC (citing Duhaime, SBS NEJM 1998; Willman, 1997). Studies in children with *nonaccidental* head injuries also indicate that they show an immediate decrease in their LOC at injury. (citing Gilles & Nelson, 1998) Individuals sustaining diffuse brain injury of mod to severe degree become symptomatic immediately." (citing Ommaya & Gennarelli, 1974; Adams, DAI, 1982; Adams, DAI, 1989). (See also SPR roundtable 2001, below). Children with rebleeds into a chronic SDH will always (a) be in a susceptible group with an enlarged subdural space, *and* (b) have been symptomatic before the rebleed. (See SPR roundtable 2001 under "Battered Child Syndrome, above.) Apnea is not due to brain swelling nor to ischemia, but to primary injury of the brain stem respiratory center. (This latter statement seems debatable.) Unknown how long a shaken infant can live without treatment, since many survive repeated attacks. Discounts any possibility that an expanding hematoma is responsible for deterioration in AHT victims.

This paper is commented on later by members of the journal's editorial board, who say that debate occurred as to whether it should be designated a "position paper" or just a submission of the authors.

The time-of-injury aspect of this paper is also commented on by Huntington (see 2002 Mar below), who offers a case report of a 13 month old AHT victim who was irritable, sleepy and vomiting for 24 hours before presenting to ER.

Mary Case replies that, "The claim that a young child has been fine for hours after a fatal head injury was inflicted and then suddenly developed symptoms is a claim that has no support from legitimate or mainstream medicine. When a child has suffered a serious acceleration injury to the brain that will result in long-term neurologic impairment or cause death, the so-called lucid interval is a fiction. The change from "fine" to "not fine" may be lethargy or it may be unresponsiveness, but it is a neurologic change, and it occurs at the time of injury." Am J Forens Med Path 2002 Mar; 23(1): 105-106.

Shaken baby syndrome: rotational cranial injuries -- technical report (T0039). American Academy of Pediatrics, Committee on Child Abuse & Neglect. Pediatrics 2001 Jul; 108(1): 206-210. In the most severe cases, the child "usually becomes immediately unconscious and suffers rapidly escalating, life-threatening CNS dysfunction." But: often the infants are put to bed, and later brought in convulsing or [otherwise neurologically compromised.] With comatose infants, "respiratory difficulty progressing to apnea or bradycardia...results..." See under "Shaken."

Pediatric minor head trauma. Schutzman SA, Greenes DS. *Ann Emerg Med* 2001 Jan; 37(1): 65-74. Minor head trauma causes a large number of intracranial injuries. Most of the death and disability (7,000 deaths and 29,000 permanent dsbs) are caused by deterioration and secondary brain injury. The clinical problem becomes to identify those at risk for (a) intracranial injury, and (b) deterioration and secondary brain injury. While limiting unnecessary imaging procedures. See the same authors' earlier ER studies of skull fractures and falls, 1999 and 1997.

Controversial aspects of child abuse: a roundtable discussion. *Society for Pediatric Radiology.* *Pediatric Radiology* 2001; 31: 760-774. (see above under "Battered Child Syndrome") Dr. Boal discusses lucid interval and timing of injury for the case of devastating injury to the brain. "With the exception of epidural hematomas in older children, which area a rare event with abuse, well-documented research has shown that a lucid interval does not occur. Whether or not the injury is accidental or non-accidental, an infant that is a victim of severe closed head injury does not act normally, take a bottle, interact with the caregiver, and then become moribund."

Reichert KW and Schmidt M, Neurologic Sequelae of Shaken Baby Syndrome. *In:* Lazoritz S and Palusci VJ, *The Shaken Baby Syndrome: A Multidisciplinary Approach.* New York: The Haworth Maltreatment & Trauma Press, 2001, p. 81-82. "...major mechanical force followed by immediate or rapid onset of neurological symptoms." (citing Gennarelli, 1983). "[I]t can be discerned that there is no evidence of a prolonged interval of lucency between the injury and onset of symptoms in children with acute subdural hematoma and brain swelling. This injury is seen in all *severe* cases of child abuse associated with coma or death. Thus, an alert, well-appearing child has not already sustained a devastating acute injury that will become clinically obvious hours to days later. Timing of the traumatic event is more difficult to establish in patients with *mild* neurological injuries..." [emphasis added --JKR]

Symptoms following head injury (letter). Huntington RW III. *Am J Forens Med P* 2002 Mar; 23(1): 105. Case report of a 13 month female AHT victim with presenting complaint of 24 hours of vomiting irritable and sleepy. Fussy and clingy after admission. Head CT negative. Found with decreased respirations several hours after admission. Mother fled. Autopsy showed thin widespread SDH and DAI by BAPP. +RH, ONSH. This letter is in criticism of the NAME Position Paper on infant head injury and its position that there is never any significant lucid interval in fatal injury. Mary Case replies that the vomiting irritable and sleepy in this case counts as "lethargy" and therefore this case only confirms the argument that symptoms are immediate -- "symptoms" in the sense of lethargy or unconsciousness.

Does second impact syndrome exist? McCrory P. *Clin J Sports Med* 2001; 11: 144-149. Second impact syndrome is sudden neurological deterioration immediately after a second impact occurring some hours or days after a previous head impact. This guy argues that it doesn't exist.

Biomechanics and neuropathology of adult and paediatric head injury. (review) Ommaya AK, Goldsmith W, Thibault L. *Br J Neuros* 2002; 16: 220-242. This article (unfavorably reviewed by Duhaime and Christian in the Quarterly for January 2003, see under Fall versus Inflicted) includes comments on lucid intervals, taken from the study by Berney (1994). See under Fall versus Inflicted, below.

Infantile subdural hematomas due to traffic accidents. Vinchon M, Noizet O, Defoort-Dhellemmes S, Soto-Ares G, Dhellemmes P (Lille). *Pediatric Neurosurgery* 2002 Nov; 37(5): 245-253. Retrospective review of 18 cases of infantile SDH due to TA (under 24 mos). At least some of the blood was already hypodense by CT on the first hospital day. Blood hyperdensity was always found during the first week, and turned hypodense on about day 9. Three pts had RH "of a type distinct from that found in SBS. "The fact that a **single and recent trauma can result in mixed-density ISDH** can be of great importance in forensic medicine."

Delayed sudden death in an infant following an accidental fall: a case report with review of the literature. Scott Denton and Darinka Mileusnic. *Am J Forens Med P* 2003 Dec; 24(4): 371-376. A nine month old asthmatic black male was sitting on the edge of the bed while his grandmother dressed a sibling. He fell off and hit the back of his head on a vinyl-covered concrete floor, 30 inch fall distance. He cried and had a

knot on the back of his head. Was taken to baby sitter, did well all day, acted normal for the next two days. At 72 hours after the fall, he was found dead by his mother. Last seen alive 8 hours earlier, at 64 hours post-injury. Autopsy showed a 9 cm linear parietal skull fracture with diastasis of the lambdoid suture, a subgaleal hematoma with evidence of aging (centrally red with yellow margins), a 2 cm posterior SDH, and a tear of the corpus callosum with FAI. Sections of the fracture showed an acute fracture with early periosteal reaction. Swollen brain (weight not given but a photograph is given). "Analysis of the fall revealed a rotational component... which could account for the described injuries." Discussion: addresses several issues with this case: (1) whether it really was accidental, (2) whether fatal brain injury from a low-height fall is possible, (3) the lucid interval, (4) DAI, apparently because they anticipate an argument that the child could not have become comatose or died without DAI or with only the very localized FAI found in this case. (In other words, that there must have been some other cause of death, such as suffocation or asthma).

(1) Whether it really was accidental. The authors detail a thorough forensic and police investigation which corroborated the family's story.

(2) Whether fatal brain injury could result from a low-height fall. The authors accept the view of Duhaime Alario et al. (1992) that low-height falls rarely cause significant brain injury in infants. "However, every fall is different." They ascribe the death to "secondary brain injury," by which they mean swelling and delayed cell death, which they say is age-dependent. "It has been shown that infants and young toddlers lose consciousness less frequently, and a smaller proportion of [their] head injuries lead to immediate coma in comparison to other children with the same grades of traumatic energy." (citing Barney et al., 1994). Argue that this age group should not be lumped statistically or pathologically with young infants or older children.

(3) Lucid interval. The above argument, to which is added the authors' own experience plus citation of Nashelsky and Dix, 1995 and Huntington, 2002, to argue that the commonly held tenet that children who eventually die of head injury were unconscious from the first moment, is not necessarily true. (Nashelsky & Dix, "The time interval between lethal infant shaking... Huntington, Symptoms following head injury, AJFMP 2002), and also the periods of lethargy, drowsiness, irritability, temperature irregularities, poor feeding, and GI symptoms referred to in other articles (Duhaime et al., SBS, NEJM 1998; Ward, 1995; Haviland, 1997), which the authors find to allow for "a certain progression" in symptoms. Also the report of delayed deterioration by Snoek et al., 1984. Also pointing out that one of the most frequently cited articles on time of injury -- head (Wilman, Chadwick, 1997) used a study group with an average age of 8 ½ years, mostly traffics. Also citing authorities for the proposition that clinical signs and radiologic studies in the early post-injury phase are insensitive as indicators of brain injury. Also saying that autopsy determination of the age of brain injury in hospitalized, ventilated or operated children is difficult because of the hospital- and anoxia-induced artifacts.

(4) The authors discuss DAI. Say that DAI is rare in nonaccidental head trauma, citing Geddes I and II; Smith, Nanoka, 2000). Coma is more a reflection of focal axonal damage in the brain stem. Also, the plane of rotation makes a difference as to both the location and the results of axonal injury; the distribution caused by cervical hyperextension may be more likely to affect consciousness and survival, because it is in the lower brainstem and rostral cord. (This finding also explains the presence of apnea at presentation, as noted by Geddes II.) Also saying that localized gray-white shearing does not necessarily imply DAI or diffuse injury, but may be localized (citing Geddes). Goes on to discuss hypoperfusion/hypoxia/swelling as the key features of poor outcome in children under 24 months (citing Pearl, 1998; Adelson et al., 1997; Ewing-Cobbs, 1999; Tabori, 2000). [But in the particular case being presented, the authors do not describe any hypoxic or other neuronal/axonal injury in the brain stem, and the spinal cord was not examined. Despite their attribution of "a significant rotational component" to the injury in their case, with the implication that the rotation was in a sagittal plane which would be likely to affect the brain stem or spinal cord according to their own argument. -JKR] The authors in their abstract describe the science of head trauma in small children as an "evolving field of study." Dr Reece comments in the *Quarterly* for Spring 04 that the case was thoroughly investigated and the authors' conclusion appears logical. Dr Reece questions whether the child's asthma might have played a role, since hypoxia was not excluded as a cause of the cerebral edema. The baby was unobserved for the last eight hours.

Traumatic low-attenuation subdural fluid collections in children younger than 3 years. Wells RG and Sty JR. Arch Ped Adol Med 2003; 157: 1005-1010. According to Wilbur Smith's review in the *Quarterly* for Jan 2004, this article by two established experts in pediatric radiology "shakes up" the conventional wisdom that the combination of low-attenuation and high-attenuation means acute and chronic. That conventional wisdom was based on studies of adults. Cf. Vinchon et al., 2002, above, showing early development of hypodense areas by CT in very acute SDH's in accident victims under 24 months.

Inflicted Childhood Neurotrauma. Proceedings of a conference sponsored by the Department of Health and Human Services, National Institutes of Health, National Institute of Child Health and Human Development, the Office of Rare Diseases, and the National Center for Medical Rehabilitation. Edited by Robert M. Reece, MD and Carol E. Nicholson, MD. (2003) A collection of literature reviews available from <http://www.aap.org/>. The timing of clinical presentation of inflicted childhood neurotrauma, by Kent P. Hymel, with responses from Steve Boos, David Chadwick, and Elizabeth Gilles is one of the literature reviews in the collection. **"The medical literature generally supports the conclusion that victims of moderate to severe, inflicted childhood neurotrauma become rapidly clearly, and persistently ill."** (p. 65) (emphasis in original) Describes the following papers: Willman et al., 1997: described the acute clinical presentation of 95 witnessed fatal accidentally head-injured children. The average age was 8.5 years, much older than a typical inflicted population. 90% of the victims had a GCS of 8 or below at the scene. On arrival in the emergency room, 97% had a GCS of 8 or lower. Really the only exceptions were a few EDH's. Concluded: "Except in cases involving epidural hematomas, the time of injury in a fatal head injury case can be restricted to *after* the last confirmed period of normal consciousness for the child." Levin et al., 1992: Prospective study accumulated 35 children 0-4 years old presenting to the ER with traumatic unconsciousness due to a head injury -- some abused, some not. Of these 35, 77% presented with GCS 3-5. Johnson et al., 1995: n=28 AHT very young patients (average age < 6 mos): presenting symptoms: 57% apnea or abnormal breathing, 82% requiring immediate intubation, 71% seizures in ER, 50% had first BP < 80, 54% initial pH < 7.3. Nashelsky & Dix, 1995: Searched the literature and found only 3 cases of SBS where there was enough information published to determine the time-course of symptoms after the injury. Found that in two of the cases, symptoms occurred immediately after shaking, while in the third, there was four days of vomiting followed by seizures. Starling et al., 1995: found that out of 37 perpetrators who confessed, 36 were with the child when it became visibly ill. Gilles & Nelson, 1998: Were able to track the time-course of symptoms in 9 SBS cases. Found that none had a lucid interval. Concluded "...infants and children severely injured by non-accidental mechanisms do not experience a lucid interval nor do they recover to their pre-morbid state..." Gilliland on interval duration, 1998: Retrospectively reviewing the Dallas Coroner's files on 76 AHT deaths aged < 1 year, found that a number of the decedents had delayed presentation 24 hours or more after the injury by history. But, "in all the cases where the children were seen by an independent observer after injury, they were described as not normal." Hymel adds his own previously published conclusions from an earlier literature review on TADD, to the effect that delayed onset of symptoms can occur due to cerebral edema, but only in cases of focal brain injury such as contusion or EDH. As to improving the state of knowledge of time of injury -- head, he says that only eyewitness accounts can suffice, and that will require more multicenter prospective series. The response from Steve Boos looks specifically at the literature on lucid interval and TADD. Snoek, Minderhoud, 1984, and Humphreys et al., 1990 found that lucid intervals definitely occur in adults and children in accidental circumstances, but do they occur in moderate to severe AHT circumstances? That is "currently disputed." Hendrick, Harwood-Dash et al., 1964 deemed it to be a rare occurrence outside the case of EDH. Hahn et al., 1984 found lucid intervals in a 2.2% of 318 patients under 3 years admitted with any type of head injury, and only one of these had an EDH, while 6 had SDH, cerebral edema, or cerebral contusion. Boos comments on this that "A small but measurable occurrence in children with SDH and cerebral swelling would suggest that lucid interval could be found in a limited number of children with moderate to severe AHT." (p. 70) Goes on to discuss fatal cases specifically. Says that in fatal cases, the possibility of a lucid interval is "even more highly contested." Reviewing an study by Hahn et al. in Pediatric Neurosurgery for 1993 of 790 children admitted in a lucid condition after head injuries (GCS 13-15), both of the two deaths presented with GCS 13, i.e. were lucid. (One died of an EDH, the other of delayed cerebral edema). Furthermore, some of the other patients who did not deteriorate and die would have done so but for early surgical intervention. Therefore, lucid intervals can occur in fatal head

injury cases. But, note that in order to even be included in this study, patients by definition had to have a history of either immediate LOC, neurological deficits, nausea, vomiting, a skull fracture, or questions about abuse & neglect. Therefore, all were “symptomatic” at presentation to the emergency room, albeit not unconscious. Thus, concludes Boos, “**Symptomatic lucid intervals may occur uncommonly in children with nonfatal injuries and rarely in children with fatal injuries resembling AHT.**” (p. 70) [But note, he says “symptomatic” lucid intervals, not asymptomatic lucid intervals, re this paper. –JKR] Then he notes Carole Jenny’s well-known paper (1999) showing that the symptoms of AHT can be missed even by a physician; of those that were missed, 9% had fatal AHT.

Another literature review in this collection is a literature summary by Robert A. Zimmerman, MD, entitled “Radiographic evaluation of inflicted childhood neurotrauma.” Under “areas of ambiguity,” Zimmerman discusses the confusion about interpreting hyperacute SDH versus acute/subacute SDH. He briefly summarizes this problem as of 2002, citing the work of Sargent et al. (1996) and Barnes & Robson (2000). In a hyperacute SDH, you see high density representing clotted blood and low density representing unclotted “fresh” blood. “It [the low-density component] may also represent extrusion of serum into the site of bleeding from early clot retraction or leakage of CSF from an arachnoidal tear.” (p. 88) Goes on to say that “This acute/hyperacute SDH can be mistaken for the chronic SDH with a spontaneous or new trauma leading to rebleeding.” [As to this ambiguity, see also Wells & Sty, Arch Ped Adol Med 2003, 157: 1005 (above); Vinchon et al., J Neurosurg (Pediatr I) 2004; 101: 44, summarized in the *Quarterly* for January 2004 and Spring 2005, respectively. –JKR]

Another contribution is the one on biochemical, metabolic, and molecular responses in the brain after inflicted childhood neurotrauma, by Patrick M. Kochanek MD. This includes the statement that “Severe TBI results in a **robust acute and subacute inflammatory response**. Clinical studies in iTBI have defined unique facets of the inflammatory response in the acute and subacute phases. Neutrophil accumulation in the injured brain may contribute to blood-brain barrier damage, edema, and oxidative stress. Neutrophil accumulation is mediated by a number of molecular factors, including the chemokine interleukin 8 (IL-8) and endothelial adhesion molecules ... such as ICAM-1. Levels of IL-8 and soluble ICAM-1 are elevated in CSF after severe TBI.” (p. 194) Kochanek gives in tabular form the results of a literature search on biochemical mechanisms in TBI (two pages long).

Other contributions in this volume are summarized above under “Shaken.”

Analysis of perpetrator admissions to inflicted traumatic brain injury in children. Starling SP, Patel S, Burke BL, Sirotnak AP, Stronks S, Rosquist P. Arch Ped Adol Med 2004 May; 158(5); 454-458. See above under “Shaken.” Studied 81 perpetrator confessions in 453 AHT cases. Found 57 confessions in which the time of infliction was specifically described. Of these 52 (91%) said the onset of symptoms was immediate. The others were vague, but said less than 24 hours. Authors’ conclusion: “**The symptoms of inflicted head injury in children are immediate.**” See same author’s 1995: 97% of perpetrators said they were present when the baby became symptomatic.

Imaging of head injuries in infants: temporal correlates and forensic implications for the diagnosis of child abuse. Vanchon M, Noule N, Tchofo PN et al. J Neuros (Pediatr I) 2004; 101: 44-52. From the review by Mark Dias in the Spring 2005 issue of the *Quarterly*: Describe concomitant CT and MRI scans of acute traumatic SDH in 20 infants out of 184 seen for trauma. These 20 infants had the following mechanisms of head injury: 10 shaking, 2 beatings, 4 traffics, 4 birth trauma. The time of injury was known in all 20. Now 16 of these had SDH’s. Found that all the CT scans done during the first seven days after injury showed hyperdense blood. All the ones done after 11 days did not show any hyperdense blood. The initial CT scan of all the SDH’s showed mixed density collections. When later CT scans were done, they all showed sedimentation of the collection toward the posterior areas along the falx and tentorium. They also performed MRI scans on the 20 patients and compared them with the CT scans. The MRI findings showed areas consistent with CSF in the “supernatant” corresponding to the areas of mixed density on the CT scans. In other words, the findings suggested that there was a layering out of the SDH, with blood clot layering below a mixture of blood, serum, and CSF, accounting for the mixed-density appearance on CT scan. The clot portion did undergo a radiographic evolution over time which, with further case studies, might lead to a timing scale, but not at present. Dr Dias comments that this work makes a valuable

contribution. He and they have already reported that mixed-density subdurals can be acute and do not necessarily indicate previous injury. (Dias et al., *Pediatric Neuros* 1998; 29: 77-85; Vinchon et al., *Pediatric Neuros* 2002; 37: 245-253). This article adds support to that idea and goes against the “prevailing dogma” that mixed density means old and recent. Dr Dias comments that this dogma comes from adult material which is inapplicable in pediatrics.

Traumatic intracranial injuries can be clinically silent. Hymel KP. *J Pediatr* 2004; 144: 701-702. According to Dr Starling’s summary in the Winter 05 Quarterly, these authors retrospectively identified 130 children under 2 years of age referred for suspected abuse who had **no neurological abnormalities on presentation**. 75% of them had a neuroimaging study done, of which **29% showed an intracranial injury**. This correlates with Carole Jenny’s well-known 1999 study on missed AHT. Dr Starling comments that it supports the early use of cranial imaging in suspected abuse even if there are no overt symptoms to support it.

Starling SP. Head Injury. In: Giardino AP and Alexander R, *Child Maltreatment: A Clinical Guide and Reference, Third Edition*. St. Louis: G. W. Medical Publishing, 2005. The use of imaging to date a head injury is limited. It can date intracranial hemorrhage as to acute (a few days) or chronic (days to weeks). Timing via imaging should be used with caution. Watch out: mixed density can have various meanings:

- old and new bleeding
- hyperacute (active bleeding)
- early clot retraction with serum separation
- arachnoid tears with leakage of CSF into the subdural space

The literature on time of injury: Wilman, Bank, 1997: Onset of symptoms was immediate in all but one of 95 accidental head injuries (often with injury types identical to those seen in AHT), and the only lucid interval was an EDH. Gilles & Nelson, 1998, had 11 patients, all with immediate onset of irreversible neurological symptoms. Gilliland, interval duration, 1998, found that severe or less severe symptoms such as lethargy were always present immediately in the cases that had an independent observer. And Ommaya’s primate studies (*JAMA*, 1968) found immediate LOC in his rhesus monkeys subjected to whiplash with DAI. And Starling et al., perpetrator study, 1995 found that 97% of the perpetrators were with the child when the child had onset of LOC, and most of these perpetrators were not the child’s usual caregiver, and therefore did not spend large amounts of time with the child ordinarily. **Conclusion as to time of injury:** “Both clinical and animal model studies support the immediate onset of symptoms in abusively head-injured children. The symptoms may be mild, such as vomiting or lethargy, or more severe, including respiratory compromise and seizures... A child who appears well, eats, or is playful did not sustain a devastating brain injury earlier in the day.” (p. 49)

Case analysis of brain-injured admittedly shaken infants: 54 cases, 1969-2001. Jan E. Leestma, MD. *Am J Forens Med P* 2005 Sep; 26(3): 199- 212. This literature review (reviewed by me in the *Quarterly* for Winter 2005) collects all the articles (23 articles) from Guthkelch (1971) on down that contain case reports where babies were documented to have been shaken, as opposed to some other history. As to the eleven (11) pure shakens that L was able to find in this literature, none of them had immediate onset of unconsciousness. As to the twelve (12) shaken-plus-impacts, 17% had immediate onset of symptoms. [But note that these are what L considered “valid” cases, that is, cases in which the published account contained specific data about time of onset. And of course there is the problem that the time of onset often is concealed or not reliably observed, so these data may mean nothing. –JKR] For this article, see above under “Shaken.”

Motor vehicle crash brain injury in infants and toddlers: a suitable model of inflicted head injury? Shah M, Vavilala MS, Feldman KW, Hallam DK. *Child Abuse & Neglect* 2005; 29(9): 953-967. This is a retrospective chart review, trying to answer the questions:

- 1 Can there be a lucid interval after a severe or fatal head injury?
- 2 Are they immediately symptomatic?

Reviewed the records of 51 children of 0 to 36 months who were in motor vehicle accidents. Found that LOC information was frequently missing, as was level-of-consciousness information. Found, according to Dr Duhaime's review in the Winter 2006 *Quarterly*, that the pts with focal contact injuries tended to be conscious and have good outcomes, while those with diffuse injuries tended to be unconscious and have poor outcomes. See above under "Shaken."

Nonaccidental head trauma as a cause of child death. Graupman P, Winston KR (DG). *J Neuros* 2006; 104: 245-250. From Dr Heilman's review in the Winter 2007 *Quarterly*, this study from DG retrospectively reviewed the charts of 36 fatal AHT chil admitted to DG. Found that all of them had depressed LOC on admission. "In many of the cases," Dr Heilman writes, "the timing of the injury could not be established. In the 18 children where the timing could be established, only 17% arrived at the hospital in less than three hours after the injury." In other words, there was delayed rescue in 83%. 75% had GCS 3 on admission; therefore, 25% had GCS better than 3, which is interesting, given the fact that 83% of all the cases had delayed rescue and all were fatal.

Mechanisms, clinical presentations, injuries, and outcomes from inflicted versus noninflicted head trauma during infancy: results of a prospective, multicentered, comparative study. Hymel KP, Makaroff KL, Laskey A, Conaway MR, Blackman JA. *Pediatrics* 2007 May; 119(5): 922-929. See above under "Shaken" and "Head injury -- fall vs. inflicted." The authors present the following case: a 19 month old black child who fell 6 to 10 feet onto concrete, cried immediately, and was consolable. But 30 minutes later he developed impaired consciousness lasting for over six hours including periods of apnea, flaccidity, unresponsiveness, and seizure activity. His CT scan revealed only a skull fracture without any intracranial or brain injury. He improved "dramatically" and was discharged on the second hospital day. Lost to followup. Of him the authors observe, "This case seems to demonstrate that delayed and significant clinical deterioration can occur after closed pediatric head trauma, even in the absence of visible intracranial injuries." Note by JKR: It was a severe concussion, hardly surprising in a fall of six to ten feet onto concrete. Why were the symptoms delayed? Probably a localized cortical contusion that didn't show up on the admission CT. It would have shown up on a followup imaging or MRI. Such a contusion would be expected to undergo localized swelling and neuron damage which takes time to develop. He recovered because young children are resilient.

BUNK BEDS

Did you know there is such a thing as a "bunk bed fracture?" It's a buckle fracture (an axial jamming fracture) of the base of the first metatarsal. See Subtle fractures in kids: how not to miss them. Leonard E. Swischuk, MD (Galveston). *Appl Radiol* 2002 Nov; 31(11): 15-19, under "Fractures -- in general," above.

Bunk bed injuries. Selbst SM, Baker MD, Sharnes M. *Am J Dis Chil* 1990; 144(6): 721-723. Says 12% had concussions, one case had SDH, 10% had fractures, 40% had lacerations, 28% had contusions.

Bunk beds: a still-underestimated risk for accidents in childhood? Mayr JM, Seebacher U, Lawrenz K et al. *Eur J Pediatr* 2000; 159: 440-443. Studied 218 bunk bed accidents. Found 91 major injuries, including 7 simple skull fractures, 44 concussions, 2 spleen lac, 33 long bone fractures. There were no cases of intracranial bleeding and no deaths.

Over a nine-year period Jan 1, 1999 to Aug 9, 1999, nine (9) children were killed by falls from bunk beds, according to the Consumer Product Safety Commission's Final Rule of Dec 22, 1999. As opposed to 84 who were killed by hanging and entrapment. Hanging has involved the siderails. Entrapment has involved the wall. Also having the upper bunk material fall through onto the child sleeping in the bottom bunk. Accordingly, the Commission's mandatory standards pertain to preventing hanging and entrapment, and say nothing about preventing falls, except as appears below. <http://www.cpsc.gov/> It does say that children under two should not be put in bunk beds, but in cribs. It does say, in the mandatory warning label

to be placed on all bunk beds, "WARNING: To help prevent serious or fatal injuries from entrapment or falls: never allow a child under 6 years on upper bunk."

Reece & Ludwig, *Child Abuse: Medical Diagnosis and Management, Second Edition (2001)*, Betty Spivack and Kent Hymel in ch. 1 ("Biomechanics of physical injury"), p. 18: "In specific circumstances, accidental pediatric falls may cause severe cranial injury (albeit rarely). These potentially dangerous circumstances include stairway falls in an infant walker, stairway falls in the arms of an adult, or falls from an elevated bunk bed." (citing Chiaviello, Walkers, 1994; Chiaviello, Stairways, 1994; Joffre & Ludwig, Stairways, 1988; Selbst, Bunk beds, 1990.)

Preventing Childhood Falls. CDC SafeUSA program, July 9th, 2001. (have) A short seven page summary on falls at <http://www.cdc.gov/safeusa/falls.htm>

Cause of infant and toddler subdural hemorrhage: a prospective study. Feldman KW et al. Pediatrics 2001; 108: 636-646. 66 cases, abuse in 59%.

Bunk versus conventional beds: a comparative assessment of fall injury risk. Belechri M, Petridou E, Trichopoulos D. (Athens) J Epid Comm H 2002 Jun; 56(6): 413-417. Retrospective study of ER records of 1881 ch w bed fall injuries: 197 bunkb, 1684 regular b. Ages 0 – 14 years. Found increased relative risk of injury from fall from bunkb, inj more serious -- included TBI, fracs, multiple inj, and inj req hosp. Apparently no deaths. Recommends bedrails.

Infant-furniture-related injuries among preschool children in New Zealand, 1987-1996. Morrison L, Chalmers DL, Parry ML, Wright CS. J Paediatr Child H 2002; 38: 587-592. From Sam Gulino's review in the *Quarterly* for July 03: National injury database search found 1,679 injuries requiring hosp with 43 fatalities, mostly mechanical asphyxia. Most of the hosps were falls. 62% of all the injuries were head injuries. Of the falls requiring hosp, 44% caused intracranial injury, 11% skull fxx

beds	40%
bunk beds	20% (bunk chil were older)
strollers	12%
walkers	7%
cots	5%
high chairs	4%
bouncers	3%
changing tables	2%

Also examined 130 walker falls, found that they had higher accident injury scores than nonwalker stair falls and other falls.

Knight's Forensic Pathology, Third Edition. Saukko P and Knight B. New York: Oxford University Press, 2004. Chapter 22, "Fatal Child Abuse." See above under "Classic Articles." Includes a discussion of falls causing skull fracture and brain injury.

Shaken baby syndrome: theoretical and evidential controversies. Minns RA. Journal of the Royal College of Physicians of Edinburgh 2005; 35: 5-15. See above under "Shaken" for a detailed summary of this review article. This article states that "Falls from top bunks may also rarely produce SDH's."

WALKERS

The NEISS data, variously reported:

- 1974 CPSC chart review of 176 ED's finding 3,700 walker victims in 1974 (1)
- 1975 CPSC Fact Sheet 66 apparently repeating the above data (2)

- 1980 CPSC Revised Fact Sheet 66 saying that in an unspecified year (?1980), 24,000 infants sustained injury resulting in ER visits, with 54% of these being from falling down stairs. (3)
- 1980 Text of Trinkoff & Parks, 1993, quoting an estimate from the CPSC based on the NEISS to the effect that in 1980 there were 10,016 injuries requiring ER visits. (4)
- 1981 Text of Trinkoff & Parks, 1993, quoting an estimate from the CPSC based on the NEISS to the effect that in 1981 there were 17,125 injuries requiring ER visits (5)
- 1991 Text of Trinkoff & Parks, 1993, quoting an estimate from the CPSC based on the NEISS to the effect that in 1991 more than 27,000 walker-related injuries requiring ER visits were estimated to have occurred, with 90 percent of the injuries to the head, face, or mouth. (6)
- 1993 Text of AAP Policy Statement, 1995, citing NEISS for the fact that “in 1993, 25,000 children were treated in hospital ED’s for injuries associated with the use of infant walkers; about ¼ of the injuries were described as ‘more serious.’ ” (7)
- 1992 Eleven deaths occurred for 1989 through 1993. Text of the AAP Policy Statement, 1995, citing the source given in note 7(b), below.
- 1995 AAP Policy Statement No. RE 9520 quotes NEISS data to the effect that in 1993 25,000 children were treated for walker injuries, with ¼ of the injuries being “more serious.” States that there were 11 deaths in 1989 through 1993 (7)
- 1997 Text of CPSC press release #98-142, 1998, stating that the CPSC estimates that in 1997 walkers were involved in 14,300 hospital ER-treated injuries to children younger than 15 months, and that “Walkers have been involved in 34 deaths since 1973.” (8)

Sources given for the above information:

- (1) Footnote 1 of the AMA Policy Statement, 1991, which cites “US CPSC. Baby Walker Injuries: Hazard Analysis. Washington, DC: US Bureau of Epidemiology, 1974.”
- (2) Footnote 2 of the AMA Policy Statement, 1991, which cites “US CPSC. Baby Walkers. Washington DC: US Bureau of Epidemiology, 1975. US CPSC Fact Sheet 66.”
- (3) Text of the AMA Policy Statement, 1991, and is cited to “US CPSC. Baby Walkers. Washington DC: US Bureau of Epidemiology, 1980. US CPSC Fact Sheet 66 Revised.”
- (4) Text of Trinkoff & Parks, citing “CPSC: NEISS. Washington DC, 1984.”
- (5) Same as reference (4)
- (6) Citing “CPSC: NEISS, injury estimates -- 1990, 1991. Washington DC, 1992”
- (7) Citing two sources:
 - (a) Karels TR, ed. Briefing package -- baby walker petition HP-92-2. Washington, DC: US CPSC, 1992
 - (b) CPSC. Baby walkers: advance notice of proposed rulemaking. Federal Register, 1994; 59: 39306-39311.
- (8) The press release does not give a source, but it appears to be NEISS data.

16 CFR 1500.86 [a] 4. (1971) The mandatory standards to prevent injuries to the fingers and toes by pinching or collapse of the frame. Also voluntary standards to prevent tipovers. See the voluntary standard revision of 1996.

Problems resulting from the excessive use of baby walkers and baby bouncers. Simpkins M and Raikes A. Lancet 1972; 1: 747

Burns to infants using walker aids. Miller R, Colville J, Hughes NC. Injury 1975; 7: 8-10

Infant walkers: developmental tool or inherent danger. Ridenour M. Percept Mot Skills 1982; 55: 1201-1202. Not useful in development; may be bad for motor development.

The infant walker: a previously unrecognized health hazard. Kavanagh CA and Banco L. Am J Dis Child 1982; 136: 205-206 These two pediatricians prospectively did a questionnaire survey on 195 patients aged 5 to 15 month in their practice over a three-month period. 150 of the patients used walkers. Of those, 47 (31%) suffered mishaps.

Injuries

Contusions/abrasions	38
Head injury or skull fracture	5
Lacerated lip	2
Perforated palate	1
Avulsed tooth	1

Mechanisms

Tipped over	38
Fell down stairs	15
Pushed by sibling	2

Case 1: a 7 month old male propelled himself down a flight of stairs. Had a large ecchymosis over the left frontoparietal area. No LOC. Normal neurological exam. Skull series disclosed two left frontal fractures, one of which was depressed, and a linear left parietal fracture. Infant underwent operative elevation of the depressed fracture, did well.

Case 2: an 8 month old male tipped over his walker, landed on his mouth and knocked out a front tooth.

Baby walker injuries. Fazen LE and Felizberto PI. Pediatrics 1982 Jul; 70: 106-107. This was a survey by two pediatricians of 49 families in their practice. 86% used walkers. 50% of the infants experienced at least one accident in the walker. Two required hospitalization -- both fell down stairs and suffered head injuries. The first, a 6 month male, fell down a flight of stairs, had a concussion and facial bruising. The second, a 7 month male, fell down a flight of stairs and had multiple head and neck abrasions and a transitory unilateral facial palsy. "baby walker accidents may represent a new unrecognized cause of pediatric morbidity..." "The majority of these accidents result in subclinical injuries that are not reported. When injuries do occur, head trauma is the most likely problem noted." This study was very similar in design to Kavanagh & Banco's simultaneous study, above.

Injuries related to baby walkers. Hobroyd HJ. Pediatrics 1982; 70: 147

Infant walkers and cerebral palsy. Holm VA, Harthun-Smith L, Tada WL. Am J Dis chil 1983; 137: 1189-1190. Bad for these children.

Preventing accidents in the home. Heather Lang-Runtz. Can Med Assoc J 1983 Sep; 129: 482-485. Cited by AAP policy statement 1995 for the proposition that the increased mass and the increased starting speed of 1 m/sec increase the energy of walker-stairs compared to other falls. The author is a freelance writer in Ottawa. The article discusses all types of household accidents to all age groups. The author attributes to Dr. Andre L'Archeveque, a pediatrician at Hopital Sainte-Justine in Montreal, an expert on children's accidents and chairman of the Canadian Pediatric Society's accident prevention committee, the statement that "A baby in a walker can cover 1 metre in 1 second, so parents should make sure that the door to the basement is always closed."

Infant walkers. Blasco PA, Baumgartner MC. Am J Dis Chil 1984; 138: 992. Ch w cerebral palsy who use walkers get more symptomatic, not less.

Baby walker-related injuries. Wellman S and Paulson JA. Clinical Pediatrics 1984 Feb; 23(2): 98-99 A retrospective review of the charts of 38 children seen in the emergency room of Rainbow Babies and Children's Hospital for walker-related injuries over three years from 1979 to 1981. Ages 5 to 24 months,

mean age 9 months. 37 had head or face injuries. One got burned by pulling a hot iron down on his hand. 25 of the injuries resulted from a fall down stairs. The head and face injuries included:

15	hematomas
9	abrasions
6	lacerations

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“Only one child was hospitalized, and this was for suspicion of child abuse.” 99

Head injuries related to the use of baby walkers. Stoffman JM, Bass MJ, Fox AM. Can Med Assoc J 1984 Sep; 131: 573-575. This paper presents two separate studies. In the first study, these ER doctors retrospectively reviewed the charts of 52 children under 24 months who came to the ER with head injuries. Walkers were involved in the head injuries of 42% (10 of 24) of the patients under 12 months of age, and in none of those over 12 months. All the walker head injuries involved stairs. There were three skull fractures. (See further on). In the second study here, the authors mailed a survey to all the families attending their clinic, asking about walker use and injuries. They got 152 responses. 82% used a walker. 36% reported that their child had had a fall while using the walker. 8% of the falls resulted in contact with a doctor. Note 1: in the chart study, the history of walker-stairs fall was accepted as fact; there was no attempt to go behind the history. Note 2: The survey showed that the walker-related falls were associated with the experience of an older sibling: if an older sibling was reported to have fallen, the study child was more likely to have had two or more (!) falls in a walker.

Head injuries and baby walkers. (letter) Stewart AR. Can Med Assoc J 1984; 131: 1327. Developmentally bad. Commenting favorably on the study by Stoffman & Bass.

Head injuries and baby walkers. (letter) Taylor F. Can Med Assoc J 1985; 132: 96. Developmentally bad. Commenting on the study by Holm.

Infant walkers and cerebral palsy. Bachman DS. Am J Dis Chil 1985; 139: 11. Bad for these children.

Patterns of walker use and walker injuries. Rieder MJ, Schwartz C, Newman J. Pediatrics 1986 Sep; 78(3): 488-493. From the Hospital for Sick Children, Toronto. The first-ever prospective study. The authors state that the stimulus for their study was the case of a 6-month-old infant who fell down 14 steps in a walker onto a concrete floor and died as a result of severe cerebral injury. Studied the injuries sustained by infants 4 to 15 months old adm to this hospital for injuries in walkers during a 1-year period in 1984. 139 injuries. The most severe injuries were caused by falls down stairs; these falls accounted for 123 of the injuries.

Fell down stairs	123	89%
Fell out of walker	10	7%
Burns	3	2%
Object pulled onto pt	3	2%

Injuries:

Skull fracture	19	(2 depressed fxx)
Closed head injury	93	
Forearm fracture	3	
Clavicular fracture	2	
Nasal fracture	1	
Burn	3	
Dental	7	
Laceration	6	
Abrasion	3	
Soft tissue inj	1	
Nasal inj	1	

All but one of the fractures were caused by falls down stairs. 85 of the 93 closed head injuries were caused by falls down stairs. There were 20 hospital admissions, of which 16 were for skull fracture. Two depressed skull fractures required elevation. The great preponderance of the skull fractures and CHI were in the age range 7 to 10 months. Two children were re-injured: one CHI victim suffered a fractured radius in a second fall. Another CHI victim had a second CHI resulting in long-term hemiparesis; "the cause of the hemiparesis was uncertain..."

The infant walker: an unappreciated household hazard. Connecticut Medicine 1990; 54: 127-129

Do retinal hemorrhages occur with accidental head trauma in young children? Alario A and Duhaim T (abstract) Am J Dis Child 1990; 144: 445 (1990) 50 chil under 2 who fell or suffered other accidental trauma such as MVA's, including 25 who fell down stairs, (many in walkers): 6 hd intracranial injuries, 14 had uncomplicated skull fxx, 2 EDH. None had RH.

Chronic subdural hematoma: another babywalker-stairs related injury. DiMario FJ Jr. Clinical Pediatrics 1990 Jul; 29(7): 405-408. Case report of a 23 month old male referred to pediatric neurology because of enlarging head size. The mother related that at age 6 months he had fallen down a flight of stairs into the basement in his walker, landing on a cement floor. He was dazed for about one minute, with no LOC, no seizures or vomiting. Was seen by ped, exam normal. Subsequent normal growth & development except for the head size becoming abnormally large. Cranial ultrasonography at 9 months of age disclosed a small right-sided subdural hematoma. At 23 months development was normal, but the right head had a prominent convexity. There was pupillary anisochoria. Fundi were normal. Leg clonus and + Babinski bilaterally. Left arm ataxia (spastic diplegia). MRI revealed a large right-sided chronic SDH causing midline shift and uncal herniation. VP shunt done. Improved. MRI scan done at 23 months shows a very large white collection with minimal interhemispheric involvement. In evaluating children with a history of symptomatic walker-stairs accidents, "Consideration of an abused "shaken baby child abuse is also warranted as the etiologic event."

Walker-related burns in infants and toddlers. Johnson CF, Ericson AK, Caniono D, Pediatric Emergency Care 1990 Mar; 6(1): 58-61.

Baby walker related injuries -- a continuing problem. Coats TJ, Allen M. Arch Emerg Med 1991 Mar; 8(1): 52-55. The authors retrospectively reviewed the records of 1049 baby visits to their accident and emergency unit at Leicester Royal Infirmary by patients aged 0 to 24 months. They found 22 injuries associated with baby walkers, including three skull fractures. The most common mechanism was a fall down stairs. The incidence of injury was comparable to that of car accidents -- a common hazard.

Head injury and the use of baby walkers: a continuing problem. Partington MD, Swanson JA, Meyer FB. Ann Emerg Med 1991 Jun; 20(6): 652-654. Retrospective chart review at the Mayo Clinic of 129 children under 24 months who were evaluated in the ER or clinic for head injuries, not including facial injuries. (Similar design to the Canadian study of Stoffman et al., above. But this study is much more elaborate, and includes extensive information about non-walker-related falls.) There were 19 walker-related head injuries. 18 of these came from falling down stairs. Nine walker patients had skull fractures: six linear, , one comminuted,, two with multiple linear fractures. One pt got traumatic meningitis from a fx that communicated with the nasal cavity. 9/19 was 47% of walker head injuries were skull fractures. (Compared to only 27% of nonwalker stairway falls). No extremity fractures in the walker cases. All patients did well without postadmission sequelae.

This article is cited by the AAP Committee on Injury and Poison Prevention (2001) for the proposition that "[W]alkers accounted for 45% of falls down stairways causing head injury in children younger than 24 months, and these walker-related stairway falls caused more severe injury."

Use of infant walkers. Board of Trustees, AMA. Am J Dis Chil 1991 Aug; 145: 933-934. (have) Cited in AAP policy statement, 1995, for the proposition that parents report that injuries occur to 12% to 40% of all infants who use walkers. This article cites a 1974 study by the CPSC involving review of visits to 176 emergency departments and personal interviews, finding that 3700 children in the sample had received

treatment for trauma resulting from walkers. [Footnote 1: CPSC, Baby walker injuries: hazard analysis. Washington DC: US Bureau of Epidemiology, 1974. Footnote 2: US CPSC. Baby walkers. Washington DC: SU Bureau of Epidemiology, 1975. CPSC Fact Sheet 66.] Citing CPSC revised fact sheet 66, 1980, for the proposition that in 1980, 24,000 infants sustained an injury related to the use of walkers resulting in 8600 ED visits; 54% of these involved falling down stairs. The Board goes on to say that the most common types of injuries involve falling down stairs, tipping over, and finger entrapment. Almost all *serious* trauma results from falling down stairs. Closed head injury most common, fol by fractures (skull, arm, clavicle), and other injuries such as burns, dental injuries, and lacerations.

Infant walker use in private practice populations. Coury DL, Kasten EF, Shepherd L, Mirvis B, Columbus PROBE group. Am J Dis Chil 1992 Apr; 146: 507. (have) Cited in AAP policy statement, 1995, for the proposition that walkers are used by 55-92% of all infants. This brief report from the Department of Pediatrics, Ohio State Univ School of Med, reports a questionnaire survey of parents of 234 one-year-old patients seen in private practice. 92% used walkers. 12% reported an injury in the walker, most (36%) beinf minor head injury.

Head injury in very young children: mechanism, injury types, and ophthalmologic findings in 100 hospitalized patients younger than 2 years of age. Duhaime A-C, et al. Pediatrics 1992; 90: 179-185. Reports one case of walker stairs as one of four cases of accidental injury with RH. No details given on the RH.

Prevention strategies for infant walker-related injuries. Trinkoff A and Parks PL. Public Health Reports 1993 Nov-Dec; 108(6): 784-788. (have) There were an estimated 27,804 walker-related injuries requiring emergency-room attention in 1991. In 1981 there were 17,125, as estimated by the CPSC from the NEISS (citing NEISS for 1984). Of these, 90% were injuries to the head, eye, face, or mouth. The male:female ratio of injured was 60:40. The estimates for 1991 were:

Internal injury including concussion	24%
Laceration	12%
Contusions or abrasions	48%
Other injuries	15%

The source given for these estimates is the CPSC NEISS injury estimates. The article discusses the NEISS estimates. These were national estimates based on projections from a probability sample of injuries treated in 91 sampled emergency departments. The authors cite Wellman & Paulson, 1984, Kavanaugh & Banco, 1982, and Fazen & Felizberto, 1982 for the proposition that in the early 1980s, studies indicated that infantnt walkers were associated with a significant risk of injury. In 1986 the ASTM promulgated a performance standard (No. F977-89) addressing the design of stability, seating, and folding mechanisms and requiring warning labels saying that w should never be used near stairs or stoves or heaters. The authors recommend consideration of prohibiting the manuf and sale of walkers and/or redesign of the w.

Infant walker related injuries: a prospective study of severity and incidence. Chiaviello CT, Christoph RA, Bond GR, Infant walker-related injuries: a prospective study of severity and incidence. Pediatrics 1994 Jun; 93(6 Part 1): 974-976. All infants brought to the U of Va Peds ER with walker-related injury were prospectively studied. 65 patients. Mechanisms associated with walker-related injuries included stairway falls in 46 infants (71%), tipovers in 14 infants (21%), falls from a porch in 2 infants (3%), and burns in 3 infants (5%). These injuries predominantly involved the head and neck region, with few injuries to the extremities (6%) and trunk (3%). Although the majority of the injuries were minor, significant injuries occurred in 19 infants (29%). These injuries included skull fracture, concussion, intracranial hemorrhage, full-thickness burns, c-spine fracture, and death. After excluding the burned patients, all the serious injuries resulted from falls down stairs. The annual incidence of injuries occurring in infants less than 1 year of age related to the use of walkers and resulting in ER visits was 8.9/1000 infants, and for serious injuries was 1.7/1000 infants. CONCLUSION: The incidence and sifnificance of infant walker-related injuries are unacceptably high.

Baby walkers -- an underestimated hazard for our children? Mayr J, Gaisl M, Purtscher K, Noeres H, Schimpl G, Fasching G. (Graz). Eur J Pediatr 1994 Jul; 153(7): 531-534.

1. A survey of 240 toddlers in Styria . 131 had used walkers. No benefits found.
2. Retrospective clinical study. 169 patients injured in walkers. In 143 there was a fall down stairs (83%). In 26 the walker tipped over (15%). 166/169 patients had a head injury. 48 required hospital admission for a mean duration of one week. The serious head injuries were:
 - 15 calvarial fractures
 - 2 basal skull fractures

All GCS were 13-15. All patients recovered without sequelae.

The authors quote an oral report by L Buterbaugh to the 1992 interim meeting of the AMA to the effect that six toddlers died in walker-related accidents in the US from 1991 to 1994.

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US Consumer Product Safety Commission. Baby walkers: advance notice of proposed rulemaking.

Federal Register 1994; 59: 39306-39311. 11 fatal Injuries 1989-1993 included 4 drowning, 4 fatal neck compression by the food tray, 3 fatal falls.

Bilateral fibula fractures from infant walker use. Sheehan KM, Gordon S, Tanz RR. Pediatr Emerg Care 1995 Feb; 11(1): 27-29

Injuries associated with infant walkers. Policy Statement No. RE9520. American Academy of Pediatrics Committee on Injury and Poison Prevention. Pediatrics 1995 May; 95(5): 778-780 Quotes figures from the National Electronic Injury Surveillance System (NEISS) to the effect that in 1993 25,000 children were treated in hospital emergency departments for injuries associated with the use of infant walkers. About ¼ of the injuries were described as “more serious.” (That would be about 6,000 a year.) These were overwhelmingly closed head injuries and fractures, resulting from falls. Stairs are involved in 75-80% of injuries and in almost all serious injuries. States that there were eleven deaths during the period 1989 through 1993, citing the US Consumer Products Safety Commission, *Baby Walkers: advance notice of proposed rulemaking*. Federal Register 1994; 59: 39306-39311. Cites Partington, 1991 for the view that because the infant tends to remain in the walker while falling, there is unprotected head exposure. Cites Lang-Runtz, 1983 for the proposition that the added mass of the walker and the higher starting speed (1 m/sec) gives more energy in the fall. [If you actually read Ms. Lang-Runtz’s article, it only states that a walker can achieve a speed of 1 m/sec across the floor; it does not discuss falling mechanics.] Cites Partington, 1991 for the proposition that walkers accounted for 45% of all falls down stairs causing head injury in children under 24 months, and that these falls caused more severe injury. Walkers have no positive benefits to balance the “considerable risk” of injury. Recommends that walkers be banned.

The association of vision-threatening ocular injury with infant walker use. Mark Koser MD and Patrick A DeRespinis MD, UMDNJ, Arch Pediatr Adol Med 1995 Nov; 149(11): 1275-1276. “Of 14 consecutive infant walker-related injuries evaluated ... over a period of one year, two children (14%) sustained skull fractures in association with serious ocular injury in the form of Terson’s syndrome. Terson’s syndrome may be defined as the association of preretinal and/or vitreous hemorrhage with an intracranial hemorrhage.” Patient 1 a 6 month old male fell down six stairs, suffered a right parietal skull fracture with epidural and subdural hematomas necessitating neurosurgical evacuation. A large preretinal hemorrhage surrounding the right optic nerve head and obscuring the macula. Followup examination revealed a persistent left hemianopsia related to a right-sided cortical infarct. Patient 2 an 8 month old female fell down a flight of stairs that had a gate which was apparently left open. Sustained a right frontoparietal SDH requiring evacuation. Dilated ophthalmoscopy revealed bilateral vitreous and preretinal hemorrhages covering both maculas (photo provided). The photo shows a very large vitreous and preretinal hemorrhage obscuring the macula and taking up 50 degrees of the field of view. This article is cited in Cindy Christian & A-C Duhaime’s 1999 article on household accidents and RH (see below.)

See Andrew Sirotiak’s letter, Arch Pediatr Adolesc Med 1996 Jun; 150(6): 652, referring to this article: “As a pediatrician trained in the medical aspects of child abuse, I am compelled to remind the readers that the association of intracranial hemorrhage and retinal hemorrhages in an infant or young child may be indicative of abusive head trauma....”

Limitations of child injury data from the CPSC's national electronic injury surveillance system: the case of baby walker-related data. Injury Prevention 1996 Mar; 2(1): 61-66. NEISS (National Electronic Injury Surveillance System) suffers from low sensitivity due to sampling error.

Fatal extradural hemorrhage following a fall from a baby bouncer. Claydon SM. Pediatr Emerg Care 1996 Dec; 12(6): 432-434. This was not a walker, but one of those spring-suspended seats from the ceiling. He considered non-accidental trauma in the differential. It was a thickly carpeted floor. He posits an accelerated fall due to rotation of the infant's body by the flipping of the seat.

[Patterns of use, popular beliefs, and proneness to accidents of a baby walker (go-cart). Bases for a health information campaign.] (Sp.) Santos Serrano L et al. An Esp Pediatr 1996 Apr; 44(4): 337-340. Baby walkers are a potential cause of accidents in infants 6 mos to 1 year of age. Questionnaire study done on 207 parents of infants. Of the infants who used walkers, 24.9% had experienced an accident (falls 76.2%, injuries 14.3%, hospital admission 4.8%). Dangers reported by parents included: 27.2% none, 33.5% leg deformities, 43% accidents (33.5% injuries and 12% falling down stairs).

Standard consumer Safety Performance Specification for Infant Walkers (ASTM F977-96). Philadelphia: ASTM, 1996. The 1996 revision of the voluntary standard. Modifying the 1971 voluntary standard which related to tipovers and warning labels by adding the voluntary structural standard (width 900 mm or stair brake) to prevent stair falls. According to the AAP Committee on Injury and Poison Prevention (2001) industry compliance is unevaluated.

Baby walkers -- still a major cause of infant burns. Cassell OCS, Hubble M, Milling MAP, Dickson WA. Burns 1997; 23: 451-453. These were 15 contact burns from grasping or spills. Do not appear to me to be walker-related. But Ken Feldman says the walkers need to have enough rim to confine exploring hands. He says more generally that walkers turn infants into "mobile unguided missiles."

Baby walker injuries continue despite warning labels and public education. Smith GA, Bowman MJ, Luria JW and Shields BJ. Pediatrics electronic pages, 1997 Aug; 100(2): e1. URL <http://www.pediatrics.org/cgi/content/full/100/2/e1> 271 patients with walker-related injuries admitted to the emergency room of Children's Hospital, Columbus. 96% fell down stairs. 26 skull fractures -- 3 depressed, 3 with intracranial hemorrhage including 2 with subdural hemorrhage.

- 271 walker-related injuries
- 159 contusions/ abrasions
- 35 concussions/head injuries
- 33 lacerations
- 26 skull fractures
 - 17 parietal
 - 8 frontal
 - 1 occipital
 - 3 depressed skull fractures
 - 2 of these had a second skull fracture present
 - 3 intracranial hemorrhage, including
 - 2 SDH
- 7 bloody noses
- 3 other fractures
- 4 knocked out teeth
- 1 burn

One SDH patient developed a chronic subdural effusion. One patient had possible generalized brain swelling, and one had frontal lobe edema. The average number of stairs fallen was 9.5. The number of stairs fallen down (ten or less versus more than ten) was significantly associated with skull fracture (P=.01) and hospital admission. The relative risk of skull fracture was more than threefold higher for children who fell down more than 10 stairs. Children who landed on concrete were more likely to sustain a skull fracture and be admitted, but not at a level of statistical significance (p=.20). There were also three clavicular

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fractures and one radius-ulna fracture. All patients survived. The article contains discussion of prevention issues, including the fact that warning labels haven't worked and neither has constant supervision by adults. It takes only a few seconds for a baby to cross the room in a walker at 1 m/sec. Stair gates don't work either -- they are commonly left open. Most of the infants injured were supervised at the time of injury.

Alexander, Levitt and Smith in *Reece, 2d ed.* (2001) at 64, quote this art to the effect that out of 260 walker-stairs falls, there were ten hospital admissions, all ten for skull fxx. Of these fxx, three were depressed, three had accompanying intracranial hemorrhage. That it made a difference how many stairs fallen.

Infant walker use, injuries, and motor development. Thein MM, Lee J, Tay V, and Ling SL. *Injury Prevention* 1997 Mar; 3(1): 63-66. 12.5% of users had one or more injuries. Most injuries were minor (e.g. bruises on the head and face).

Epidural hematoma and stroller-associated injury. Lee AC and Fong D. *J Paediatr Child Health* 1997 Oct; 33(5): 446-447. Case report and literature review. Case of an EDH in a 10 mo female who fell out of a stroller. No fx. Surgically evac, recovered. Five case reports of stroller injuries were found. Most injuries were mild; there were three deaths, two of which were child abuse. "life-threatening injuries are rare but these are potentially preventable if strollers are properly designed and safety recs are followed." See letter: Epidural hematoma and stroller-associated injury. Donald TG. *J Paediatr Child Health* 1998 Aug; 34(4): 402

A closer look at intracranial injuries due to falls in infants less than 2 years of age. 1998. Waarvik D and Levitt C, Midwest Children's Resource Center, St. Paul, Minn. 55102 612-220-6750. Research presentation at the Second National SBS Conf, 1998. (have abstract). N=30 documented falls. 9 walker stairs accidents: ages 5m-8m: 6 skull fractures: skeletal surveys negative: 4 SDH listed as "small:" 3 large intracerebral hemorrhages: 3 RH -- one multiple bilateral posterior-pole RH and two with just one flame hemorrhage. Opth was documented in all. None had peripheral hems, large hems, or traumatic retinoschisis. Quite a number of skull fractures and SDH's. No deaths.

Bilateral pediatric skull fractures: accident or abuse? Arnholz D, Hymel KP, Hay TC, Jenny C. *J Trauma* 1998 Jul; 45(1): 172-174 In a witnessed outdoor accident involving a fall out of a stroller on concrete steps for three feet, this 6 week old infant suffered symmetrical biparietal linear fractures with two separate and symmetrical areas of scalp hemorrhage. This the authors attributed to impact on the crown of the head and skull outbending on either side. See under Falls vs Inflicted.

Parental decisions to use infant walkers. Bar-On ME, Boyle RM, Endriss EK. *Injury Prevention* 1998 Dec; 4(4): 299-301. Residents at an ambulatory-care clinic interviewed 154 primary caretakers; 77% used walkers. 72% believed that walkers accelerated babies' development of walking skills. But they waited several months before buying one. Conclusion: "Until legislation can be passed banning walkers, this period of time may provide a window of opportunity for appropriate anticipatory guidance in the form of intense media-assisted antiwalker campaigns."

Consumer Product Safety Commission reports 34 deaths since 1973. In 1997 walkers were involved in 14,300 emergency room --treated injuries to children under 15 months. Most children sustained injuries when their walkers fell down stairs. See CPSC web site at <http://www.cpsc.gov> The citation is US Consumer Product Safety Commission. Baby Walkers. Washington, DC: US Bureau of Epidemiology; 1992. US Consumer Product Safety Fact Sheet 66.

Ages of young children who fall down stairs. Ridenour MV. (Biokinetics Research Laboratory of Temple University) *Perceptual and Motor Skills* 1999 Apr; 88(2): 669-675. (have) Children falling down stairs is a frequent household accident. Unprotected stairs is viewed here as the underlying remediable hazard. Falls in general are "the leading cause of death in children between 1 and 4 years old." (citing Hall et al, 1989 and Lewis, 1991) Infants are more likely than older children to die from falls. The purpose of this "review" of "a sample of emergency rooms" was to study the *age distribution* of children who fall down stairs. They also studied the difference between those who fell using walkers and those who fell using natural locomotion, because this has also not been well studied. There were 619 ER visits for falling down

stairs at ages 5 to 18 months during the first six months of 1995. 235 fell in walkers; 384 with natural locomotion. The walker victims were younger (average age 8 mos versus 13 mos.)

The methodology of this "review" is unclear. Under "methods," the article says, "These descriptions of incidents are from a probability sample of the approximately 6,000 hospitals that report having hospital emergency rooms or emergency visits." The sample was obtained from the National Injury Clearinghouse of the CPSC.

"Studies have indicated that gates were installed in over half the incidents involving a child falling down stairs in a walker." (citing AMA Trustees, 1991.) Usually when a child fell, the gate was left open. There is a risk of falling down stairs regardless of whether the child is using a walker or not. Need for constant supervision and parental education efforts.

Retinal hemorrhages caused by accidental household trauma. Christian CW, Taylor AA, Hertle RW, Duhaime AC. J Pediatr 1999 Jul; 135(1): 125-127. Reports 3 children with unilateral posterior-pole RH localized in the eye ipsilateral to intracranial hemorrhage. All recovered. "Retinal involvement was relatively mild, without peripheral retinal involvement, retinal folds, or detachment." Case 1 a walker-stairs 13 month old with a large R acute convexity SDH and right-sided posterior preretinal and intraretinal hems NOS. Case 2 a 9 month old hit the floor when father playing, had L SDH, left eye had multiple flame hems and round intraretinal hems in the posterior pole, and two small posterior vitreous hems. Case 3 a 7 month old fell off stairs onto the basement floor, large R convexity SDH with ML shift, a coronal diastatic fx, a R temporal linear fx and fracture contusion, evac SDH. Ophth on 3d hospital day showed R subretinal and preretinal hemorrhages in the posterior pole, (not further described), along with orbital cellulitis. "Differentiating hems caused by accidental injury from those caused by abuse may be difficult, because either cause can result in unilateral or bilateral hemorrhages and can involve all layers of the retina." 127

Childhood head injuries: accidental or inflicted? Reece RM and Sege R. Arch Pediatr Adol Med 2000 Jan; 154: 11-15. Retrospective chart review of 287 head trauma admissions to Tufts Univ Hosp aged 1 week to 6 1/2 years. They separated out walker injuries, of which they had 18. These were 6% of all the head injuries. Of these 18 pts,

13	simple skull fxx	=72%
4	complex skull fxx	=22%
1	SDH	= 6%
1	SGH	= 6%
2	contusions	=11%
2	concussions	=11%

No SAH
No RH
No deaths

Intussusception following a baby walker injury. Connors GP, Weber CE, Emmens RW. J Emerg Med 1999 Mar-Apr; 17(2): 269-271. Published abstract: Serious abdominal injury as a result of a fall in a baby walker has not been previously reported. We present the case of a 13-month-old boy who developed intussusception following a fall down five stairs in a baby walker. Attempted hydrostatic reduction was unsuccessful. At operation, a bowel wall hematoma, serving as a lead point, was identified. This case adds another type of injury to the list of those previously associated with baby walker use.

Reece & Ludwig, Child Abuse: Medical Diagnosis and Management, Second Edition (2001).

Alexander, Levitt & Smith in ch. 3 ("Abusive head trauma") p. 64: Levitt and McCormick (unpublished study) found that in 9 walker stairs falls, three had SDH, three had large intracerebral hematomas, two had small brain contusions, one had SAH.

Betty Spivack and Kent Hymel in ch. 1 ("Biomechanics of physical injury"), p. 18: "In specific circumstances, accidental pediatric falls may cause severe cranial injury (albeit rarely). These potentially dangerous circumstances include stairway falls in an infant walker, stairway falls in the arms of an adult, or falls from an elevated bunk bed." (citing Chiaviello, Walkers, 1994; Chiaviello, Stairways, 1994; Joffre & Ludwig, Stairways, 1988; Selbst, Bunk beds, 1990.)

Injuries associated with infant walkers. American Academy of Pediatrics, Committee on Injury and Poison Prevention. Pediatrics 2001 Sep; 108(3): 790-792. Stating that in 1999 8,800 children were tr in emergency rooms for walker injuries. Stating that 34 deaths were reported from 1973 through 1998 (from the NEISS data). The vast majority were from falls down stairs. A 56% decrease in injury reports between 1995 and 1999. (21,100 down to 8800 injury reports). Citing Rieder, 1986 and Partington, Swanson, 1991 for the proposition that walkers caused 45% of falls down stairways that caused head injury in children under 24 months, and these head injuries were more serious than non-walker-related stairway-fall head injuries, and that this is because of the larger mass and higher initial speed of the walker and because “the infant tends to remain in the walker while falling, resultin in unprotected head exposure.” Citing Smith, Bowman, 1977 for the proposition that stairs are implicated in 75% to 96% of all walker falls and in almost all of the severe injuries. Citing CPSC *Advance Notice of Proposed Rulemaking* , 1994 for the proposition that there were 11 walker deaths reported between 1989 and 1993, including 4 drownings, 4 fatal neck compressions by the food tray, and 3 falls. ASTM F977-96 is the revised voluntary standard requiring wider than 36 inches or a stair brake (because many walker-stairs falls were on basement stairs, and the doors to these are usually under 36” wide.) Seems to imply that this standard will prevent stair falls if complied with, but industry compliance unknown. Recommending a ban on the manufacture and sale. This Policy Statement was reaffirmed by the Academy in January 2005.

Accidents and resulting injuries in premobile infants: data from the ALSPAC study. Warrington SA, Wright CM, ALSPAC study team. Arch Dis Child 2001; 85: 104-107. A questionnaire survey to parents finding 3357 falls in 2554 infants under six months. Including fifty walker falls. No intracranial injuries.

Injuries from falls in the pediatric population: an analysis of 729 cases. Wang MY, Kim KA, Griffith PM, Summers S, McComb G, Levy ML, Mahour GH. J Pede Surg 2001 Oct; 36(10): 1528-1534. (see also under Head Injury -- Fall vs. Inflicted) (have) Retrospective chart review at CHLA of 729 falls between age 0 to 15 years admitted for injuries from falls. Included 42 walker falls (5.8% of all injuries from falls.)

There have been two recent CPSC voluntary recalls of walkers, as noticed on the web site of the Juvenile Products Manufacturers’ Association at <http://www.jpma.org> These were a Taiwan-made walker from SunTome Corp. of Los Angeles, recalled because it will fit through doorways and has no stair brake, <http://www.cpsc.gov/cpsc/pub/prerel/prhtml01/01203.html> and a Kolcraft rider-walker whose removable music center can break off, creating a choking hazard.

Imaging of child abuse. Nimkin K and Kleinman PK. Radiol Clin N Amer 2001 Jul; 39(4): 843-864. “Stairway injuries, injuries in infant walkers, and falls in the arms of a caretaker are important sources of accidental long-bone fractures.” 857 (citing Kleinman’s book at 214-224)

Femur fracture in infants: a possible accidental etiology. Grant P, Mata MB, Tidwell M. Pediatrics 2001; 108: 1009-1012. Report one seven month-old who was using an Exersaucer (an infant leg-exercise toy) and one 4 month-old also using an Exersaucer. These were Salter-Harris type II fractures, which extend obliquely *through* the physis rather than along it. Caused in these cases by twisting forces exerted by the baby itself (?) I looked up “Exersaucer” on the Consumer Product Safety Commission, and found that it was developed in the early 90’s by Evenflo Juvenile Furniture Company, Inc. The company’s president described its development in detail in an invited presentation at an official CPSC conference called “Safety Sells,” held on March 28, 1995. The president, Mr. George A. Harris, tells about how walkers were heavily criticized on safety grounds because they cause 20,000 accidnts a year. He goes on to say that “In spite of the number of accidents and negative publicity, there are still over 1.5 million walkers sold every year. Why? Because a walker allows a child to sit upright, keep an eye on Mom or Dad, and offers play value that entertains a child for loing periods of time. The challenge was to devleop a procut that provided all of the positive attributes of the walker, ... but which eliminated the mobility.” He says that the Exersaucer is a non-mobile walker; it spins instead of walks. Should offer substantially the same play value without the hazard of mobility. As such, it has won several awards. The CPSC web site does not contain any reports of injuries attributed to the Exersaucer.

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Babywalkers delay normal locomotor milestones in infants. Reuters Medical News on Medscape Jun 20, 2002, citing Dr Mary Garrett, (Dublin) BMJ 2002; 324: 1494 survey of the parents of 190 infants with milestones of head raising, rolling over, sitting with support, sitting alone, crawling, standing with support, walking with support, standing alone, and walking alone. 102 used walkers, 88 didn't. For each day of walker use there was a 3.7 day delay in standing alone and a 3.3 day delay in walking alone.

Infant-furniture-related injuries among preschool children in New Zealand, 1987-1996. Morrison L, Chalmers DL, Parry ML, Wright CS. J Paediatr Child H 2002; 38: 587-592. Reviewed 130 walker falls and compared them as to accident injury score with nonwalker stair falls and other nonwalker falls.

<u>fall</u>	<u>AIS</u>	
walker	15% serious	72% moderate
nonwalker nonstair	3% serious	59% moderate

Characteristics that distinguish accidental from abusive injury in hospitalized young children with head trauma. Bechtel K, Stoessel K, Leventhal JM et al., Pediatrics 2004; 114: 165-168 (see above under "RH -- In general"). This art (or actually the supplementation of it provided in response to a letter to the editor) reports one case of a 9 month old who had a witnessed fall down stairs in a walker, sustained a R frontal skull fracture with acute R SDH and a single intraretinal hemorrhage OD.

Femur fractures in resulting from stair falls among children: an injury plausibility model. Pierce MC, Bertocci GE, Janosky JE, Aguel F, Deemer E, Moreland M, Boal DKB, Garcia S, Herr S, Zuckerbraun N, Vogeley E. Pediatrics 2005 Jun; 115(6): 1712-1722. One of their closely studied 29 cases of stair falls was a walker-stairs fall in an 8 month-old. It received an injury plausibility score of 3 on a scale of 0 to 12, where 0 is highly plausible history and 12 is a totally implausible history. The fracture was in good alignment with minimal posterior displacement. The walker case was considered to be an innocent accident. See above under "Fractures -- Femur."

Deleted: Femur fractures in resulting from stair falls among children: an injury plausibility model. Pierce MC, Bertocci GE, Janosky JE, et al. Pediatrics 2005; 115: 1712-1722. See above under "Fractures -- femur." Walkers are a known risk for severe injury.

Shaken baby syndrome: theoretical and evidential controversies. Minns RA. Journal of the Royal College of Physicians of Edinburgh 2005; 35: 5-15. See above under "Shaken" for a detailed summary of this review article. "Baby walker injuries, with falls down stairs are potentially very serious, and falls from top bunks may also rarely produce SDH's."

Success in prevention of infant walker injuries: an analysis of national data, 1990-2001. Shields BJ, Smith GA. Pediatrics 2006; 117: e452-e459. From the *Quarterly* for Summer 2006. Significant reduction in injuries in US. As a result of standards set by the CPSC and the position paper of the AAP advising against the use of walkers. Injuries fell from 23,000 in 1991 to 3,000 in 2001. About 5% required hospitalization. Dr Chadwick in his review comments that the article mentions two fatalities for 2001, and he cites Chiaviello, 1994 and AAP, 2001 for the proposition that there were many more fatalities in earlier years. He goes on to comment that "While the mobile infant walker adds some height and possibly some horizontal velocity to a fall, the occurrence of a fatal head injury in such an event should, in this era, require a careful investigation. Many times an investigation will not be conclusive, and the citation of such cases for practical or scientific conclusions should be avoided." He is referring to the fact that the two fatalities cited in the article for 2001 were obtained from a note in the Federal Register by Commissioner Stevenson of the CPSC, a questionable source with no detail. [I'm not sure whether Dr Chadwick realizes that these are not same-level falls but stairway falls. -JKR]

DURA MATER AND NEOMEMBRANES

The origin of subdural neomembranes. I. Fine structure of the dura-arachnoid interface in man. Schachenmayr W and Friede RL. Am J Path 1978; 92: 53-68. From the abstract: "It was found that the cranial meninges of humans do not include a subdural space. Instead, there is a complex, tight layer of cells, the interface layer, composed in the innermost portion of the dura mater (the dural border cells) and

the outermost portion of the arachnoid (the arachnoid barrier layer)... The erroneous macroscopic impression of a subdural space results from an extraordinary lack of cohesion within the dura-arachnoid interface layer conditioned by (a) a complete absence of collagenous reinforcement within this zone, (b) the presence of large extracellular cisterns between the dural border cells, and (c) the paucity of intercellular contacts within that latter layer... [subdural lesions] form within a sheet of torn dural border cells and not within a preexistent tissue compartment.”

The origin of subdural neomembranes. II. Fine structure of neomembranes. Friede RL and Schachmayr W. *Am J Path* 1978; 92: 69-84. A neomembrane is the result of proliferation and excessive thickening of the normal layer of dural border cells.

Why do bridging veins rupture into the virtual subdural space? Yamashima T, Friede RL. *J Neurol, Neurosurg, Psychol* 1984; 447: 121-127. A body of neurosurgical opinion holds that there is no subdural “space.” There is only a potential space. Basically, their reason for saying this is that, histologically and electron micrographically, one finds dural border cells on the dural side of the arachnoid mater and the arachnoid side of the dura mater in cases of “subdural” hematoma. Therefore, they argue, such a hematoma is actually intradural.

On the question of a subdural space. Haines DE. *The Anatomical Record* 1991; 230: 3-21. From the abstract: “There is no [ultrastructural] evidence of an intervening space between the arachnoid barrier cell layer and the dural border cell layer that would correlate with what has been called the subdural space. When a tissue space is created in this general area of the meninges it is the result of tissue damage and represents, in most instances, a cleaving open of the dural border cell layer....A survey of reports describing the morphology of the inner and outer capsule of so-called subdural hematomas in humans reveals that dural border cells are found in both parts of the capsule. Also, experimental infusion of blood into this portion of the meninges in animals frequently dissects open the dural border cell layer. These data support the view that what has been called a subdural hematoma is most frequently a lesion found within the layer formed by dural border cells....”

Position paper on fatal abusive head injuries in infants and young children. NAME Ad Hoc Committee on Shaken Baby Syndrome. *Am J Forens Med Path* 2001 Mar; 22(2): 112-122. Mary Case, Michael Graham, Tracey Corey Handy, Jeffrey Jentzen, and James A. Monteleone. As to chronic SDH, discusses recurrent subdural bleeding in special categories of pts with enlarged subdural spaces, which can lead to rebleeds from “fragile capillaries” in granulation tissue. Child with a preexisting neomembrane wd have to be symptomatic before rebleed, bc there was brain injury present before rebleed. So rebleeds cannot just happen out of the blue. About 20% to 30% of asymptomatic neons have small amts of SAH and SDH during delivery., leading to neoms. Children with rebleeds into a chronic SDH will always (a) be in a susceptible group with an enlarged subdural space, *and* (b) have been symptomatic before the rebleed. I am informed that this position paper expired by its terms in 2003.

Dural haemorrhage in nontraumatic infant deaths: does it explain the bleeding in “shaken baby syndrome?” Geddes JF, Tasker RC, Hackshaw AK, Nickols CD, Adams CGW, Whitwell HL, Scheimberg I. *Neuropathology and Applied Neurobiology* 2003; 29: 14-22. ...We propose that, in such infants, a combination of severe hypoxia, brain swelling, and raised central venous pressure causes blood to leak from intracranial veins into the subdural space, and that the cause of the subdural bleeding in some cases of infant head injury is therefore not traumatic rupture of bridging veins, but a phenomenon of immaturity. This art is extensively discussed and refuted by Punt et al. in their 2004 critique, “The ‘unified hypothesis’ of Geddes et al. is not supported by the data,” above under “Shaken.” Also by me in a *Quarterly* review.

Head injury -- abuse or accident? Wilkins B. *Arch Dis Child (Br.)* 1997 May; 76: 393-397. As to minor trauma causing SDH, he references the Japanese concept of IASDH applying mainly to infants with an abnormally wide sub(dural?) space who suffer minor injury, and denying SBS, and summarizes that dispute by quoting Raimondi’s comment that “The conclusions of these authors are sound.”

Inflicted Childhood Neurotrauma. Proceedings of a conference sponsored by the Department of Health and Human Services, National Institutes of Health, National Institute of Child Health and Human

Development, the Office of Rare Diseases, and the National Center for Medical Rehabilitation. Edited by Robert M. Reece, MD and Carol E. Nicholson, MD. (2003) Available from <http://www.aap.org/>
This book contains relevant entries on fall vs. inflicted. Each is a very short (three to five page) summary of the state of knowledge as from peer-reviewed literature, plus analytical comments and recommendations. Each paper is followed by three invited responses. One is the literature summary by Dr Reece entitled, “The differential diagnosis of inflicted childhood neurotrauma.” (pp. 17-31, with responses from Ken Feldman, Robert Sege, and Cincy Christian.) Here is the differential diagnosis of inflicted head injury:

...
widened subarachnoid or subdural space

See also “Shaken” and “Neomembranes,” above.

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ENLARGED SUBARACHNOID SPACE

A pitfall in the diagnosis of child abuse: external hydrocephalus, subdural hematoma, and retinal hemorrhages. Joseph H. Piatt, Jr. Neurosurgical Focus 1999; 7(4): #4. See above under “Shaken” and “RH.” This 2 month old fell from own height hit head on carpeted floor, had immediate seizure and bilateral scattered dot-blot RH and several boat-shaped preretinal RH and a very small L frontal convexity SDH in the presence of a preexisting enlarged subarachnoid space whose etiology was unknown. Did well without treatment.

Position paper on fatal abusive head injuries in infants and young children. NAME Ad Hoc Committee on Shaken Baby Syndrome. Am J Forens Med Path 2001 Mar; 22(2): 112-122. Mary Case, Michael Graham, Tracey Corey Handy, Jeffrey Jentzen, and James A. Monteleone. Discusses recurrent subdural bleeding in special categories of pts with enlarged subdural spaces, which can lead to rebleeds from “fragile capillaries” in granulation tissue. Child with a preexisting neomembrane wd have to be symptomatic before rebleed, bc there was brain injury present before rebleed. So rebleeds cannot just happen out of the blue.

Controversial aspects of child abuse: a roundtable discussion. 43d annual meeting, Society for Pediatric Radiology. Pediatric Radiology 2001 Nov; 31(11): 760-774. Dr. Felman mentions the “well-known and reported entity” of subdural collections associated with an enlarged subarachnoid space. Citing:

Pediatric Neuroimaging, 2d ed. Barkovich AJ. New York: Raven Press, 1995 Cited by Felman, above, as showing examples of the association of small SDH’s with large SAS.

Magnetic Resonance Imaging of Children. Cohen MD, Edwards MK. Philadelphia: BC Decker, Inc., 1990. Cited by Felman, above, for the association.

Diagnostic Imaging of Child Abuse, 2d ed. by Paul Kleinman. pp. 133-134 discusses this entity and the related previously supposed entity of “benign subdural collections of infancy,” which were actually chronic frontal atrophy with enlarged subarachnoid space in macrocephalic infants born with congenital hydrocephalus and cerebral atrophy; previously thought to represent “subdural” collections because of confusion over nomenclature and archaic imaging techniques.

Intracranial hemorrhage and rebleeding in suspected victims of abusive head trauma: addressing the forensic controversies. Hymel KP, Jenny C, Block R. Child Maltreatment 2002 Nov; 7(4): 329-348. According to Dr Reece’s review in the Quarterly for Jan 2003, the authors address the fol q’s by means of a comprehensive literature review:

Barlow's disease. C.A.B. Clemetson. Medical Hypotheses 2002; 59(1): 52-56. Barlow's disease is infantile scurvy, with easy bruisability, broken bones (pathologic fractures) and sores that will not heal. In this paper and the next one, this professor of medicine at Tulane argues that infantile scurvy can mimic SBS. See above under "Differential diagnosis -- specific disease entities." [The problem is that it can mimic the subdural bleeding and fractures of SBS, but not the brain injury. -JKR] He points out that vitamin C is a needed cofactor in the catabolism of histamine, and vitamin C deficiency causes elevated histamine levels, which of course are an inflammatory mediator that separates endothelial cells causing increased capillary permeability (and fragility?). Here, he argues that giving multiple vaccinations to an already vitamin C-deficient baby (due to maternal illness during pregnancy and exclusive bottle feeding) could cause bleeding and bruisability, including RH and SDH, due to elevated histamine levels, which might be already elevated due to intercurrent infection. He further argues, in an addendum, that frank scurvy (which is necessary to explain the broken bones often seen in SBS) could develop rapidly if hemolysis of bruises and hematomas leads to rapidly falling vitamin C levels. He documents that hemolysis does cause falling vitamin C levels in rats. He discusses a specific court case in Florida where a father named Yurko was convicted of SBS upon a 10-week old infant with acute SDH, healing rib fractures, RH, and fatal brain injury, and argues that all of these findings could have been explained by infantile scurvy, since the mother was ill during pregnancy, lost weight, and may have had decreased vitamin C intake, and the infant was persistently jaundiced for unspecified reasons. [The problem is that (1) maternal vitamin C deficiency was never documented; (2) infantile vitamin C deficiency was never documented; (3) exclusive bottle feeding was never documented; (4) apparently the infant's growth and development were normal, which is against vitamin deficiency; (5) easy bruisability was not present: the infant only had one small bruise on his face and two discovered at autopsy on his temporal areas; nor were petechiae (a classical sign of scurvy) present; (6) scurvy, as noted above, might explain the SDH and the RH and the fractures, but would not explain the brain injury characteristic of SBS. -JKR] But the author's main point is that vaccination in the presence of intercurrent infection is dangerous; it further elevates the infected infant's already elevated blood histamine levels, resulting in acute vitamin C deficiency which may become symptomatic.

Inflicted Childhood Neurotrauma. Proceedings of a conference sponsored by the Department of Health and Human Services, National Institutes of Health, National Institute of Child Health and Human Development, the Office of Rare Diseases, and the National Center for Medical Rehabilitation. Edited by Robert M. Reece, MD and Carol E. Nicholson, MD. (2003) Available from <http://www.aap.org/> See Dr Reece's summary of "The differential diagnosis of inflicted childhood neurotrauma," p. 22, for a paragraph refuting the DPT theory.

Is it "Shaken baby," or Barlow's disease variant? C.A.B. Clemetson. Journal of American Physicians and Surgeons 2004 Fall; 9(3): 78- 80. Here Prof. Clemetson points out that both Caffey and Kempe included infantile scurvy in the differential diagnosis of SBS/ battered child syndrome in their original papers. Also points out classical obstetrical knowledge that hyperemesis gravidarum can cause scurvy and Wernicke's encephalopathy and retinal hemorrhages in the gravida.

See <http://www.vaers.org> for the Vaccine Adverse Events Reporting System and forms.

CEREBRAL EDEMA see also "seizures" under RH

Severe cerebral swelling without SDH can be a form of SBS -- the "DAI-type" SBS. -Kent Hymel.

Johnson et al. Pediatric Neurosurgery 1995; 23: 305-310. Asphyxia , post-traumatic apnea, shock, or other hypoxic events can cause massive brain swelling.

TIN EAR

Tin ear syndrome: rotational acceleration in pediatric head injuries. Hanigan WC, Peterson RA and Njus G. Pediatrics 1987; 80: 618-622.

See Hymel's "Biomechanics" paper and Reece, 2d ed.

EARS

See mention by Suzanne Starling in her chapter in the third edition of Giardino AP and Alexander R, *Child Maltreatment: A Clinical Guide and Reference*. St. Louis: G.W. Medical Publishing, 2005: "External injuries that should be screened for in suspected abusive [head] trauma include lacerations, abrasions, or contusions of the face, ears, or scalp." Citing Hanigan, above, and Ken Feldman, next below.

Patterned abusive bruises of the buttocks and the pinnae. Feldman KW. Pediatrics 1992; 90: 633-636.

ABDOMINAL INJURY

See also Cardiac. See also Abdominal Radiology.

"Many of these children have received repeated blows to the abdomen over time, and careful examination and microscopic sampling of the abdominal contents has revealed extensive fibrosis confirming subacute or remote injury.

-- Enid Gilbert-Barnes, ed., *Potter's Pathology of the Fetus and Infant*. C V Mosby, 1997, pp. 1609

Visceral Injuries in Battered Children McCort J and Vaudagna J.. Radiology 1964; 82: 424-428. 10 children with initially unexplained visceral trauma accompanied by evidence of neglect. 8/10 were found to have injuries to the duodenum, jejunum, or mesentery.

Abdominal visceral injuries in battered children Touloukian RJ.. Pediatrics 1968; 42: 642-646. 5 fatal cases.

- Case 1: retroperitoneal hematoma initially missed
- Case 2: duod/jej hematoma w mesenteric tears pres as 24h vom
- Case 3: DOA with tear at root of mes + fat necrosis
- Case 4: DOA with 1 week old retroperitoneal hematoma
- Case 5: perforated duod initially missed. Pres as pain & vom.

"The possibility of visceral injury from blunt trauma should be eliminated in any child with abdominal complaints who has characteristic bruises, whether or not skeletal fractures ... are present."

"A plea is made to suspect visceral injury in any abused child who has abdominal complaints."

Pancreatic pseudocyst occurring in the battered child syndrome. Bongiovi JJ and Logosso RD. J Pediatr Surg 1969; 4: 220-226. First case ever reported. 5 yr old boy with fever, anemia, pica, distension, vomiting, three healing fractures. Author reviews all of the 34 previously reported pseudocysts occurring in children. I would add that such a case is presented in Brogdon's *Forensic Radiology* and possibly another in the fourth edition of Kempe & Helfer's *The Battered Child*. --JKR

Patterns of injury in the battered child syndrome. O'Neill JA, Meacham WF, Griffin PP and Sawyers JL J Trauma 1973; 13(4): 332-339
110 patients. 49 w repetitive ST inj. 9 w intra-abdominal inj incl 7 in the area of the pancreas.

"The clinical presentation may be confusing and may mimic infectious or metabolic disease, malignancy, or CNS syndromes."

"Trauma to the abdomen was denied in every instance indicating that the physician was in the position of having to rely solely on clinical signs in order to make a dx of severe injury."

DISCUSSANT: "They present a case of a slowly developing intestinal stricture developing over 14 months."

Pancreatitis and the Battered Child Syndrome. Slovis TL, Berdon WE, Haller JO, Baker DH, and Rosen L. Am J Roentgenol 1975; 125: 56-461.

2 cases with bony fat necrosis after beatings. In the small bones of the hands and feet. The DDX wd be fractures. Commented on by Kleinman, 2d ed.

Small bowel strictures after blunt abdominal trauma. Marks CG, Nolan DJ, Piris J, Webster CU. Br J Surg 1979 Sep; 66(9): 663-664. Case report: two patients (?adults) with posttraumatic SB strictures. Mesenteric tear was not diagnosed until six weeks later.

Trauma to major visceral veins: an underemphasized cause of accident mortality. Cohen D, Johansen K, Cottingham K, et al. J Trauma 1980; 20(11): 928-932

Occult nonskeletal trauma in the battered-child syndrome. Kleinman PK, Raptopoulos VD, and Brill PW. Radiology 1981; 141: 393-396. (have) Three abused patients

None had any external signs of trauma. Radiology disclosed abd visceral injuries:

- Case 1: duod hematoma, s.b. stricture, mes scar. (This is also case 1 in "Resolving," infra.
- Case 2: pseudocyst
- Case 3: duod hematoma liver contus

Conclusion: "Visceral injuries due to child abuse are likely to go unrecognized when they occur without external signs of trauma. The diagnosis should be considered in any child with nonspecific abdominal signs and sx..." This article cited by ASCP Check Sample FP 03-9, infra, for the proposition that significant abdominal trauma from abuse is frequently without obvious external evidence of trauma.

Recognition and management of child abuse by the surgical pathologist. Buchino JJ. Arch Path Lab Med 1983; 107: 204-205. Cited by ASCP Check Sample 03-9, infra, for the proposition that "the child's abdominal anatomy, with wide costal margins and small anteroposterior distance, renders the pediatric abdomen more vulnerable than that of an adult."

[Delayed dx of traumatic peritonitis is not uncommon.] Robbs, J Trauma 1980; 20: 308

[Due to delay these delayed ruptures are highly mortal.] Ledbetter, Arch Surg 1988; 123: 1101; Cooper, J Trauma 1988; 28: 1483

Resolving duodenal-jejunal hematoma in abused children. Kleinman PK, Brill PW, and Winchester P. Radiology 1986; 160: 747-750. (have). (See also "Occult nonskeletal trauma," supra, in which case 1 was also case 1 in the present article.) Four cases:

Case 1 -- 3 y o boy presented with pain & distension. CT#1 showed two mural defects in the duod. CT #2 three weeks later showed they had gotten smaller. Four months later child was killed. Autopsy showed retroperitoneal fibrosis consistent with prior bleeding.

Case 2 a 2 y o boy w vom & sz, UGIS 5 d after adm sh duod fold thik.

Case 3 a 28 m boy w vom & abd pain, hematuria, hemoperitoneum on CT. UGIS 3 d after adm sh hematoma of duod, jej, and mes root.

Case 4 a 2 y boy 3day hx vom and a palpable mass. UGIS sh a large duod mass; laparotomy evac a hematoma of duod. 1 month later repeat UGIS sh a residual mural mass.

Authors' discussion: "Visceral injuries are relatively uncommon findings in abused infants and children. ... inj to the duod and jej are most frequently noted in abused chil. (gives history going back to 1957). The resolving mural hematoma shows up as smooth mural nodules or as diffuse fold thickening. "A conspicuous rounded defect inferior to the duodenal-jejunal junction may be noted due to an accumulation of blood at the root of the small intestinal mesentery extending into the wall of the bowel." ... "It is likely that some acute or nonobstructing resolving hematomas may be clinically inconsequential and therefore elude detection." ... "In all cases of suspected abuse in which vomiting and abnormal abdominal findings are initially present or subsequently develop, intramural small-bowel hematoma should be considered."

Bicycle handlebar injuries in children. Sparnon AL and Ford WDA. J Ped Surg 1986 Feb; 21(2): 118-119. (have) 30 admissions over ten years. Average delay of presentation 23 hours. Ten cases of traumatic pancreatitis, 4 with duodenal hematoma included, 3 leading to pseudocyst. See Clarnette & Beasley article 1997, below. See ASCP Check Sample FP 03-9, below.

Blunt abdominal trauma in children: special considerations in evaluation and management. Evans RD, Brotman S, Wilt E. Postgr Med 1986 Feb; 79(2): 169-174. Often presents diagnostic difficulties. Use CT.

Abdominal CT scanning in pediatric blunt trauma. Haftel AJ, Lev R, Mahour GH, Senac M, Shah SI. Ann Emerg Med 1988 Jul; 17(7): 684-689. Record review of 90 pediatric emergency trauma admissions at LA Children's who were stabilized in the ER and then went to abd CT with IV contrast instead of DPL. The CT results were compared with the operative or autopsy findings. Injury mechanisms: MVA 57 (ped 40, pass 12, bi 5), fall 23, BCS 4, other 6. Note that the great majority were road accidents. Found a high PVP with only one false positive. High PVN with only one false negative.

Major blunt abdominal trauma due to child abuse. Cooper A, Floyd T, Barlow B, Niemirska M, Ludwig S, Seidl T, O'Neill J, Templeton J, Ziegler M, Ross A et al. Reviewed 15 years surgical experience. 22 cases of major BAT due to child abuse -- less than 0.5% of all child abuse cases seen. Average age was 24 mos. The perpetrator was always either the father or the boyfriend, except in two cases it was the babysitter. Mortality was 45%. One case of pancr pseudocyst devel from a pancr hematoma. Three duodenal hematomas. Five peritonitis due to duodenojej rupture.

Visceral injury in battered children: a changing perspective. Carlos J Sivit, George A Taylor and Martin R Eichelberger. Radiology 1989; 173: 659-661. In the past, the only abd organ injuries that were detected by imaging were the hollow viscus. But CT in the ER has changed all that. "Use of CT ... has resulted in frequent ID of liver and spleen inj in ch w relativley minor or nonspecific abdominal sx." It has also changed our impression of how common visceral injuries are in the child-abuse setting, by showing that non-life-threatening solid-organ injuries are present when the symptoms are minor or nonspecific and the patient would not be taken to the OR. "The case-selection bias inherent in surgical reports underestimates the prevalence of less severe injury..."
"The present analysis reveals that lower thoracic and abdominal injuries are common in symptomatic battered children. Of children with abdominal signs and symptoms, 74% had nonskeletal injuries of the lower chest or abdomen noted at CT or autopsy; 67% of those examined with CT had a thoracic or abdominal abnormality."

Serial abuse in children who are shaken. Alexander R, Crabbe L, Sato Y, Smith W, Bennett T. Am J Dis Chil 1990 Jan; 144: 58-60. 24 SBS:
71% had prior neglect, abuse, or both
25% had ev of multiple prior shakings
16% had ev of prior extracranial abuse
33% had been previously shaken

Abdominal trauma -- child abuse (letter). Dworkind M, McGowan G, Hyams J, Pediatrics 1990 May; 85(5): 892. Reports the case of a 3 month infant with acute onset of vom, diarrh, fv and lethargy. Pres w a tense, distended abd. Lapar fd to hv mesenteric avulsion with a 12 cm seg of ischemic jj; postop skel

surv rev bilat healing spiral fxx of radii. Twin brother fd to have lac face, multiple fxx of d ages. Father confessed. "What makes this case particularly unusual is tha there wer no other clinical signs of battering." "Our experience should serve as a reminder that child abuse should be part of the differential diagnosis of the acute abdomen in infants and children even in the absence of more common signs of abuse." "Visceral injury is felt to occur in less than 2% of physically abused children. A review of the literature reveals that mesenteric avulsion is not an uncommon sequela in chiodren receiving BAT. Decelerating or whipping forces tear the mesentery and may disrupt the small intestine at the sites of ligamentous support."

Blunt trauma in children: significance of the peritoneal fluid Sivit CJ, Taylor GA, Bulas DI, Bowman LM, Eichelberger MR. Radiology 1991 Jan; 185-188.

790 children with BAT underwent CT abd. 16% (123) had CT evidence of peritoneal fluid collections: 93 MVA, 19 fall from a height, 6 assault, 5 other. The frequency of associated bowel injury is high. That's the significance of peritoneal fluid.

Intimal tears of the right atrium of the heart due to blunt force injuries to the abdomen. Cumberland GD, Riddick L, McConnell CF, Am J Forens Med Path 1991 Jun; 12(2); 102-104. (have) Presents six cases of both abused children and adults with BAT. Proposed mechanism as increased abdominal pressure resulting in hydrostatic force via a column of blood in the IVC. No photos given.

Safety belt injuries in children with ecchymosis: CT findings in 61 patients. Sivit CJ, Eichelberger MR et al. AJR 1991; 157: 111-114. Cf Bowkett & Kolbe under abd inj, infra.

Blunt trauma of the abdomen in children. Fossum RM and Deschenaux KA. JFS 1991 Jan; 36(1): 47-50. Injuries to the small intestine may take days or weeks to become apparent -- difficult to tell when the injury occurred or who caused it. Case report of the autopsy on a 2 ½ year old female with several months of intermittent vomiting who had been seen several times at her HMO receiving a dx of viral flu. Expired at home. Aside from bruises and abrasions of the face etc., the abdomen contained 300cc of pus, the loops of the SI were stuck together with inflammatory adhesions, , a perforation just distal to Treitz, and "The mesentery contained a firm, gray-white mass of fibrous tissue beginning at the base and extending throughout its length, at one point encircling the intestine." The fatal blow probably occurred 2-3 days before death, but earlier blows...

Markers for occult liver injury in cases of physical abuse in children. Coant PN, Kornberg AE, Brody AS, Edwards-Holmes K. Pediatrics 1992 Feb; 89(2): 274-277. Elevated transaminases in 4/49 children evaluated for possible abuse (8%). Three of them were found to have a liver laceration. None had any external evidence of abdominal trauma.

The child with acute abdominal pain and vomiting. Alford BA and McIlhenny J. Radiol Clin N Amer 1992; 30: 441-453. Gives a DDX of 9 neonatal conditions (reflux, sepsis, pyloric stenosis, atresia, malro, Hirschspr, mecon. appx), 12 infant conditions (colic, reflux, pyloric sten, gastroe, intuss, malro/volv, appx, hern, adh, Meckel's, duod hematoma), 9 childhood conditions (appx, UTI, gastroe, intuss, tumor, IBD, adh, HUS, HSP) and 6 adolescent diseases (gastroe, testic/ov dis, PID, ectopic, IBD, appx.) Only one (duodenal hematoma in infancy) relates to possible abuse. In the summary, they say, "Evaluation of the child who presents with abdominal pain and vomiting is difficult." "The incidence of abdominal injuries in children who are abused has been previously reported as 0.5% to 2%."

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Stenosis of the small bowel after blunt abdominal trauma. Vanderschot PM, Broos PL, Gruwez JA. Unfallchirurg 1992 Feb; 95(2): 71-73. Louvain. One pt was dxd 6 weeks later, one 26 years later.

Blunt abdominal aortic trauma in Children: case report. Amir A, Alexander JB, O'Malley KF, and Doolin E. J Trauma 1993 Feb; 34(2): 293-296. A 12 year old boy fell onto a retaining wall.

Adrenal hemorrhage in abused children: imaging and postmortem findings. Nimkin K, Teeger S, Wallach MT, DuVally JC, Spivack MR, Kleinman PK. Am J Roentgenol 1994 Mar; 162(3): 661-663

Five cases by CT. All five had R adr hemorrhage. Associated with right-sided visceral trauma. Right adrenal hemorrhage has been described in patients with accidental trauma.

"The presence of adrenal hemorrhage in a child should prompt a search for other associated injuries and raise the possibility of unsuspected trauma." See also the large autopsy study by Steven DeRoux and Nancy Prendergast, below (2000).

Chylous ascites: a sign of unsuspected child abuse. Olazagasti JC, Fitzgerald JF, White SJ, Chong SKF. Pediatrics 1994 Nov; 94(5): 737-739. From the abstract: "Chylous fluid accumulation in the peritoneal cavity is associated with pathology in the lymphatic system. Primary (congenital) chylous ascites develops during the first year of life ("leaky lymphatics"), while secondary (acquired) chylous ascites, resulting from lymphatic obstruction or trauma, can arise at any time during life. Both lead to spillage of chyle into the peritoneal cavity from ruptured lacteals and lymphatic ducts. Radiographic findings suggestive of intentional injury have been observed in most published cases of secondary chylous ascites resulting from child abuse. (Citing Stormo et al., 1966, Vollman et al., 1966, Roy et al., 1970, Viswanathan et al., 1974). We present a child with chylous ascites who was initially thought to have lymphangiectasia but who later was found to be a victim of abuse." The case presented is that of an 11 month-old male who presented for elective repair of an inguinal hernia. Chyle was found in the hernia sac. His past history included a femoral fracture at 3 weeks of age said to have been incurred when his three year-old sister jumped on him. His growth was below the 5th percentile. A chest x-ray done for placement of a central venous catheter disclosed subtle signs of rib trauma. An abnormal MRI of the head showing hemosiderin and focal encephalomalacia. The parents admitted abuse. They had presented as a very nice, caring family.

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Massive chylous ascites and transected pancreas secondary to child abuse. Hilfer CL et al. Pediatric Radiology 1995; 25: 117-119.

Mesenteric injury from blunt abdominal trauma. Nolan BW, Gabram SG, Schwartz RJ, Jacobs LM. Am Surg 1995 Jun; 61(6): 501-506. Reviewed 5 years of trauma admissions with BAT in all age groups. 27 cases of BAT in all age groups. Found that mesenteric injury in BAT is rare and difficult to diagnose; often missed by CT. The most common cause was MVA.

Posttraumatic chylous ascites in a child: recognition and management of an unusual condition Benhaim J, Strear C, Knudson M, et al. J Trauma 1995 Dec; 39(6): 1175-1177.
A 2 1/2 year old boy presented with distension and vomiting. Numerous healed burns. UGIS showed obstruction of the small bowel at proximal jejunum. CT + massive ascites. Laparotomy showed dense scar tissue surrounding the base of the mesentery. Several large lymphatics were leaking profusely.

Child abuse as a cause of traumatic chylothorax. Guleserian K et al. J Pediatr Surg 1996; 31: 1696-1697

Chylothorax as a manifestation of child abuse. Geismar SL et al. Pediatr Emerg Care 1997; 13: 386-389.
Case report of an 18 month girl with old fractures and FTT.
Ken Feldman comments that shaking can rupture the thoracic duct in association with compression of vertebrae which this child had.

Intestinal stricture following seat belt injury. Lynch JM, Albanese CT, Meza A, Wiener ES. J Pediatr Surg 1996; 31: 1354-1357. Two children: one to three weeks later they developed pain and bilious vomiting. The authors say delayed presentation of symptoms from a perforated hollow viscus is rare but should be alert for.
Comment: seat belt injury is similar to child abuse mechanism, because both involve crushing against the vertebral column, unlike the usual accident, which is inertial.

Stricture of the duodenum and jejunum in an abused child. Shah P, Applegate KE, Buonomo C. Pediatric Radiology 1997 Mar; 27(3): 281-283. Child had a duodenal hematoma and contained perforations of the duodenum and proximal jejunum. These were managed conservatively for three weeks, but due to persistent obstruction a laparotomy was required. This disclosed a calcified, fibrotic mesentery and

strictures of the distal duodenum and proximal jejunum. "To our knowledge, this unusual complication of BAT has not been described in association with child abuse."

Blunt abdominal injury: simultaneously occurring liver and pancreatic injury in child abuse Cameron CM, Lazoritz S, Calhoun A.D. *Pediatr Emerg Care* 1997 Oct; 13(5): 334-336. CT scans of four abused children with elevated liver enzymes and pancreatic enzymes. Their point is that if there is liver injury, there is possibly pancreatic injury and it should be looked for by means of pancreatic enzymes and CT. These children did well.

Lacerations of the hepatoduodenal ligament, pancreas and duodenum in a child due to blunt impact. Stephen J. DeRoux and Nancy C. Prendergast. *J Forensic Sci* 1998 Jan; 43(1): 222-224. A case report of an acute death of a 2 1/2 yr old male who presented in rigor mortis. There were no externally visible abd injuries, but there was a bruise over the lumbar spine and bruises on the chest, and x-ray showed multiple healing rib fxx. Intraabdominally he had a widely torn-open duodenum and a transected porta hepatis with 500cc of hemoperitoneum, which they seem to feel came from the portal vessels. Good diagram of fist punch to abdomen with crushing of the organs against the vertebral column.

Traumatic duodenal perforations in children: child abuse a frequent cause. Bowkett B and Kolbe A. *Aust NZ J Surg* 1998 May; 68(5): 380-382 He found seven cases in a retrospective review of a 14-year period at Starship Hospital. Five were abuse and two road-seatbelt accidents. In the abuse case, you get a circumferential avulsion of the junction of the third and fourth parts, because the organ is twisted against the left border of the vertebral column. In the traffic cases, you get a small hole in the inferior border of the third part, because the organ pops or blows out due to sudden increase in intraluminal pressure because of the lap belt. Gives diagrams of the two situations. The j of the 3d and 4th parts is located just to the left of the vertebral column. The 3d part is held in a nutcracker between the SMA and the vertebral column/aorta. In the abuse case, "the force, while localized, has a shearing effect, slicing the fourth part of the duodenum from the third part just to the left of the VC where the duodenum is held in its nutcracker. This may be associated with some ischaemia to the proximal duodenum, particularly if it has been avulsed from the pancreas..." "In all five cases where this shearing-type injury was found, there was no visible upper- or mid-abdominal bruising." There was a high readmission rate with obstruction. He notes the delayed presentation in his abuse cases. (see above under "time of injury") Also says, "... it can be difficult to make the diagnosis if a child presents early." Because the 3d pt d is retroperitoneal; no diffuse peritonitis. See Sivit, Eichelberger et al., Seat belt injuries in children, under "abdominal radiology."

Fracture-dislocation of the lumbar spine in an abused child. Gabos PG, Tuten HR, Leet A and Stanton RP. *Pediatrics* 1998 Mar; 101(3pt1): 473-477

Chylous ascites: a manifestation of blunt abdominal trauma in an infant. Beal AL, Gormley CM, Gordon DL, Ellis CMC. *J Pediatr Surg* 1998; 33: 650-652. See also Beshay, 2001, below.

GI tract perforation due to blunt abdominal trauma. *Pediatr Surg Int* 1998; 13: 259-264

Pancreatic trauma in children: mechanisms of injury. Arkovitz MS, Johnson N, Garcia VF. *J Trauma* 1997 Jan; 42(1): 49-53. Retrospective chart review of all pts adm to PICU w pancr inj 1980 – 1994. 26 pts. Mostly handlebar inj. Handlebar inj gave a unique pattern of isolated pancreatic injury, often complicated by a pseudocyst.

Handlebar injuries in children: patterns and prevention. Clarnette TD and Beasley SW. (Austr). *Aust NZ J Surg* 1997 Jun; 67(6): 338-339. Retrospective review of all pediatric handlebar admissions 1990-1995. 32 children. Injuries:

Spleen	9
Liver	4
Traumatic pancreatitis	3
Transection of pancr	2
Renal contus	2
Duod hematoma	1

Bowel perf	3
Urethra	3
Inguinal – scrotal lacerations	5

Management of blunt liver trauma in children. Br J Surg 1997 Jul; 84(7): 1006-1008.

Evaluation and management of pediatric major trauma. Cantor RM and Leaming JM. Emerg Clin N Amer 1998; 16: 229-256.

Hidden spears: handlebars as injury hazards to children. Winston FK, Shaw KN, Kreshak AK et al. Pediatrics 1998 Sep; 102(3): 596-601. These ER physicians collected 107 child bicyclist admissions; the caseload was divided between minor falls (handlebar-only injuries) and automobile collisions (non-handlebar-only injuries). There were 17 handlebar-only ER admissions and 90 non-handlebar-only. The handlebar-only consisted of minor falls where the handlebar went into the abdomen. There were six splenic lacerations, three kidney injuries, two pancreatic lacerations, one pneumothorax, one thigh impalement, one basal skull fracture, and one ulnar fracture. See also ASCP Check Sample No. FP 03-9 (FP-290), 2003, below.

Injury of the GI tract from child abuse. Brown CVR, Canty TJ Jr., Canty TJ Sr. (abstract). Pediatrics 1998; 102: 803 (Suppl.). 294 child abuse patients, 14 (5%) had injury to the GI tract. The presentations or modes of detection of the GI injury were: free air in 5, CT findings in 7, clinically obvious abdominal catastrophe in 2. Only five had abdominal wall contusions. The sites of injury were SI (10), duod (5), colon (3), stom (2). Perfs 10, contus 4, ischemic 2. Seven patients had associated solid-organ injury. Five had CNS injury.

Costochondral junction fractures and intra-abdominal trauma in non-accidental injury (child abuse). Ng CS and Hall CM. Pediatric Radiology 1998; 28: 671-676. Incidence of 4% in child abuse. These fxx of anterior CCJ ribs 6 – 9 bilateral. Rare compared to posterior and lateral rib fxx in child abuse, but are associated with blunt abdominal trauma -- here duodenal rupture, ileal serosal tears, pancreatic transection, portal vein tear, mesocolic hematoma, pseudocyst, splenic rupture.

Pneumatosis intestinalis and portal vein gas after blunt abdominal trauma. Gurland B, Dolgin SE, Shlasko E, Kim U. J Pediatr Surg 1998 Aug; 33(8): 1309-1311. (Mount Sinai). Case report of a 2 year old boy with BAT from child abuse. The findings establish mucosal injury but do not necessarily imply intestinal necrosis.

Injuries of the gastrointestinal tract from blunt trauma in children: a 12-year experience at a designated pediatric trauma center. Canty TG Sr., Canty TG jr, Brown C. J Trauma 1999 Feb; 46(2): 234-240. Retrospective review of 11,592 hospital admissions found 79 children aged 4 mos to 17 years:

22	passengers
15	pedestrians
15	child abuse
10	handlebars

Diagnosis was made quickly in 45 children, delayed more than four hours in 34 and beyond 24 hours in 17 chil. All six deaths were caused by head injury. Complications included two delayed abscesses and two cases of obstruction. “The majority of GI tract injuries (60%) are caused by a discrete point of energy transfer such as a seatbelt (19%), a handlebar (13%), or a blow from abuse (19%).... Although the dx may be difficult and often delayed, this did not result in excessive morbidity or mortality.” Nonoperative management. Discusses delayed diagnosis due to incomplete or false histories provided by abusing parents, causing increased morbidity.

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Nonaccidental trauma. Davis DP and McCaslin RI. J Emerg Med 1999 Jan-Feb; 17(1): 111-112

Medical complications in long-term survivors with X-linked myotubular myopathy. Herman GE, Finegold M, Zhao W, de Gouyon B, Metzberg A. J Pediatr 1999 Feb; 134: 206-214. This disease causes peliosis

hepatitis, which can cause fatal subcapsular hematoma of the liver with hemoperitoneum due to trivial trauma.

Identification of intra-abdominal injuries in children hospitalized following blunt torso trauma. Holmes JF, Sokolove PE, Land C, Kupperman N. Acad Emerg Med 1999; 6: 799-806. Chart review of 1040 admissions for BAT, mostly nonabusive. High risk factors for BAT were gross hematuria, abdominal pain, and altered LOC. AST was 604 versus 77, ALT 276 versus 39, WBC 16.3 versus 12.8, UA over 5 RBC's. Amylase and Hct were not discriminant. Discusses abdominal bruises and abrasions.

Traumatic adrenal hemorrhage in children: an indicator of visceral injury. Luchtman M, Breitgand A. Pediatr Surg Intern 2000; 16: 586-588. Three chil adm to trauma unit had adrenal hemorrhage + liver injury caused by MVA's. Dx by CT.

Adrenal lacerations in child abuse: a marker of severe trauma. Steven J. DeRoux and Nancy C. Prendergast, (NY CME) Pediatr Surg Int 2000; 16: 121-123. An autopsy study of all child abuse cases autopsied at NYC from 1990 through 1996. 121 cases. 50 had BAT. 5 had unilateral adrenal lacerations, four of them to the right adrenal gland. Discusses the CT appearances in abusive BAT. Cf Nimkin et al., 1994, above. Cf Hutchins & Natarajan, infra.

Blunt impact abdominal injury in child abuse. ASCP Check Sample no. FP 00-5, by K.D. Hutchins and Geetha Natarajan of the New Jersey ME. Makes the general point that the external and internal injuries have to be correlated in order to make DX, and this has to be correlated with the history re being consistent w hx. Further points: "...with increasing age there is a shift of site of the fatal injury from the head to the abdomen. Toddlers are at a particularly high risk for homicidal blunt impact abd inj bc of their inherent defenselessness and their capacity to inflame emotions with their increasing mobility." (citing Kleinman, 2d ed. at 248-284, and Cantor & Leaming, supra.) Frequent absence of external signs. Clinical dx difficult. Sometimes exact mechanisms are elusive. Describes "finger tip marks" of the skin. Gives a (meaningless) argument about biomechanics. The vulnerable points are the ligamentous attachments of the organs and the sources of blood supply. Comments on each organ. Describes lytic bone lesions in the case of pancreatic injury, predominantly in the small bones of the hands and feet, citing Kleinman, 1998 and citing Slovis, 1975, supra. Stom ruptures usually on the lesser curvature. Duod damage can occur at any location -- the fixed portion, over the vertebral column, or at the portion fixed at the l of Treitz. Colon rarely. Mesenteric hematomas and lac are common in both acc and inflicted. Adrenal commonly injured, more commonly R than L. Direct impact injury; shearing inj to the vessels; pressure-wave injury thr the adrenal vein from the IVC. Spl rarely in abuse but commonly in acc. Notes that handlebar injury can cause serious organ damage, but it's only a single site. Sports injury also serious but usually involves the spleen, K intestine. Birth injury usually the liver: but subc hematoma cd rupture 1 week after delivery. Also birth hematomas of the adrenal, K, spl, citing Schullinger, 1993, supra under Birth Injury. Comments on time of injury: see under Time of Injury -- Abdomen. In conclusion, "the eval ofr intraabd inj c by child ab is one of the most difficult and challenging issues in FP..."

Can falls on stairs result in small intestine perforations? (literature review). Huntimer CM, Muret-Wagstaff S, Leland NL. Pediatrics 2000 Aug; 106: 301-305. Reviewed the adult and pediatric literature excluding walker cases. Found three articles (1974, 1988, 1994). Had 677 cases of falls on stairs, involving 432 children and 245 other persons. No intestinal perfs. Such a history in a small child should be suspect.

Blunt abdominal trauma in children. Rance CH, Singh SH, Kimble R. J Paediatr Child H 2000; 49(11): 14. Cited in ASCP Check Sample FP 03-9 for the proposition that "[I]n multisystem trauma and significant abdominal injury, it has been demonstrated that even physical examination by physicians may be unreliable in up to 30% of cases."

Chylous ascites: a case of child abuse and an overview of a rare condition. Beshay VE, Beshay JE, Rosenberg AJ. J Pediatr Gastroe Nutr 2001 Apr; 32(4): 487-489. See also Beal, 1998.

Management of major pancreatic duct injuries in children. Canty TG, Weinman D. J Trauma 2001; 50: 1001-1007.

Duodenal perforation: a diagnostic pitfall in non-accidental injury. Champion MP, Richards CA, Boddy SA, Ward HC. Arch Dis Chil 2002; 87: 432-433. Three case reports of cases that presented with vomiting and scant history.. Recommends CT with IV and oral contrast as the method for Dx this retroperitoneal injury.

ASCP Check Sample No. FP 03-9 (FP-290). Corey TC and Wetherton AR. 2003. Case report of a 6 year old boy who had one day of vomiting and fatal dehydration following a bicycle accident. Case of handle bar injury causing duodenal perforation with fatal peritonitis. Found a circular handle bar mark on the skin of the abdomen. Also facial abrasions consistent with a fall.

Management of duodenal injuries in children. Clendenon JJ, Meyers RI, Nance M, Scaife E. J Pediatr Surg 2004 Jun; 39(6): 964-968. A retrospective chart review of all children treated for duodenal injuries over a 10 year period in two trauma centers. Had 42 children -- 33 blunt and 9 penetrating. No deaths. 24 required operative management and 18 had nonoperative management. 94% of the duodenal hematomas were managed nonoperatively. The operations used were primary repair, duodenal resection and gastrojejunostomy, and pyloric exclusion. Increased complication rates were seen in two groups: the group with delayed diagnosis and the group requiring operative intervention. In the delayed diagnosis group (>24 hour delay), the complication rate was 43%. The factors that were associated with delayed diagnosis were the presence of a foreign body, child abuse, and bicycle injury.

Duodenal injuries in children: beware of child abuse. Gaines BA, Shultz BS, Morrison K, Ford HR. (Pittsburgh) J Pediatr Surg 2004; 39(4): 600-602. An 8-year retrospective review to test the hypothesis that a significant number of duodenal injuries in young children are the result of nonaccidental trauma. Thirty children had injury to the duodenum -- 20 duodenal hematomas and 10 perforations. Average age 7.6 +/- 4.4. Of those under 4 (n=8), all were abuse victims. Other mechanisms seen in older children included MVA (9), bicycles (4), and ATV's (2). The abstract does not state that the findings reached statistical significance, but it concludes that in the young child, a large percentage is potentially the result of child abuse.

Abdominal injury due to child abuse. Barnes PM, Norton CM, Dunstan FD et al. Lancet 2005; 366: 234-235. (From Dr Reece's review in the Winter 2006 *Quarterly*) Compared 20 children 0-14 yrs with abusive abdominal trauma to 164 accidental abdominal traumas (112 road accidents and 52 falls). Found that the relative risk of an injured hollow viscus (perforation, contusion, or hematoma) in abuse was 2.2 x that of road accidents and 5.7 x that of falls. Also, children under 5 never suffered an injured hollow viscus from a fall. The abusive hollow viscus injuries were 5 perforations and 6 contusions or hematomas. The solid organ injuries were 6 spleens, two pancreases, 2 kidneys, 7 livers. Ten children had combinations of injuries. 5/20 patients had no visible bruises of the external abdominal wall. Six abused died. 17/20 had other injuries, such as bites, fractures, torn frenula, burns. Dr Reece notes that these other injuries "were seen as vital in making the diagnoses."

Distinguishing inflicted versus accidental abdominal injuries in young children. Wood J, Rubin DM, Nance ML, Christian CW. J Trauma Inj Infect Crit Care 2005; 59: 1203-1208. A ten-year retrospective study to test the hypothesis that delayed rescue is predictive of inflicted. Had n = 121. According to Dr Reece's review in the Spring 2006 *Quarterly*, had 77 high-velocity accidents (MVA's and falls greater than ten feet), 31 low-velocity accidents (bicycle accidents, household accidents, and falls less than ten feet), and 13 inflicted. How did they tell which were inflicted? This diagnosis was made at the time by the child protection team on the basis of : other unexplained injuries, confession, or child's disclosure. The authors excluded cases where they could not tell for sure whether the injuries were inflicted or not. Delayed rescue was stratified into under two hours, over two hours, and over twelve hours. Found that in the high-velocity accidents, 99% of the rescues were under two hours. In the low-velocity rescues, however, only 42% of the rescues were under two hours and 35% were over twelve hours. In the inflicteds, 86% were over two hours and 54% were over twelve hours. The predictive value of a delay over two hours was only 67%.

Solid organs were more common than hollow organs in abuse: 92% versus 46%. (See also the below article by Roaten et al.)

[I would say that in developing this predictive value of delayed rescue, one should have excluded the high-velocity accidents, since these are always eyewitnessed and are not really in the differential diagnosis of abdominal abuse. If the authors had done this, what would have been their PVP? --JKR.]

Visceral injuries in nonaccidental trauma: spectrum of injury and outcomes. Roaten JB, Partrick DA, Bensard DD et al., *Am J surg* 2005; 190: 827-829. According to Dr Greeley's review in the Spring 2006 edition of the *Quarterly*, had n = 265 chil with nonaccidental trauma, of which 24 had visceral injuries (9%). Solid organs were the most commonly injured, just as in the above study by Wood et al. Had 71% solid organs and 29% hollow organs. The solid organ was liver 33% of the time. As to hollow organs, had

stom	4%
duod	25%
jej/il	29%

Patient and injury characteristics in abusive abdominal injuries. Trokel M, Discala C, Terrin NC, Sege RD. *Pediatric Emergency Care* 2006; 22: 700-704. Used the National Pediatric Trauma Registry to identify 664 chil who were admitted or died of abdominal trauma, after excluding MVA's. (Note that by definition, these were the most severe injuries, since they were admitted or died.) Of these, the causes were

child abuse	40%
falls	37%

But in the fatal cases, 83% were due to suspected abuse. Dr Lindberg notes in his review in the Spring 2007 *Quarterly* that ¾ of the hollow-viscus injuries and 2/3 of the pancreatic injuries were suspicious for abuse. In his comment, Dr Lindberg points out the risk of circular reasoning in the category "suspicious for abuse." (The same problem we have in some cases of AHT.) He points out the main point: that once auto accident has been excluded, the chance that the injury is abuse is about 50%.

TIME OF INJURY -- ABDOMEN

The assessment of the duration of illness in children unexpectedly dead. John L. Emery. *Med Sci Law* 1964; 4: 39-42 (have) Thymus, liver, and CCJ histologic changes over hours and days to weeks post onset of illness or injury. See under "Autopsy technique."

Traumatic duodenal perforations in children. Bowkett et al. (see under "Abdominal Injury"). "When child abuse is considered as a mechanism of injury, it is important to make an attempt to establish the exact time of injury in relation to presentation to hospital. This information may be essential if one is to establish whether or nto an assault has taken place and by whom. In the present series we have evidence from case 4 that the delay may be up to 4 days (despite extensive duodenal injury) before significant clinical deterioration occurs and the child presents to hospital. This is probably due to the fact that the third part of the duodenum is a retroperitoneal structure and leakage may occur for some time before significant peritoneal contamination occurs." P 382

[Peritonitis due to intestinal rupture may evolve slowly.] Engray. *J Trauma* 1975; 15:854

[Delayed dx of traumatic peritonitis is not uncommon.] Robbs, *J Trauma* 1980; 20: 308

[Due to delay these delayed ruptures are highly mortal.] Ledbetter, *Arch Surg* 1988; 123: 1101; Cooper, *J Trauma* 1988; 28: 1483

Recognition and management of child abuse by the surgical pathologist. Buchino JJ. *Arch Path Lab Med* 1983; 107: 204-205.

<u>Hennes</u>	Pediatrics 1990; 86: 87-90	Timing of transaminases
<u>Coant</u>	See below	transaminases
<u>Isaacman</u>	Pediatrics 1993; 92: 691-694	Lab detec of intraabd inj
<u>Stalker</u>	Am J Roentg 1990; 154: 118-119	Hematuria

Blunt impact abdominal injury in child abuse. By K. D. Hutchins and Geetha Natarajan of NJ ME, ASCP Check Sample FP 00-5, (2000). See under Abdominal Injury. "This may be accomplished by extrapolating from the time at presentation at the hospital...to the time it may take to develop symptoms, given the injuries found at autopsy. If the solid abdominal organs are damaged, sx may appear immediately bec of massive hemorrhage and shock from large stellate lacerations. [I wonder if this is correct; has he read the liver literature? JKR] In the case of subcapsular hematomas, presentation may be delayed bc of late reup. Hollow visceral ruptures may lead to chemical and/or bacterial peritonitis, which frequently becomes evident within 6 to 24 hours, but can be delayed even longer" (citing Cooper's chapter in Ludwig & Kornberg, 2d ed, 1992.) Says that many excellent texts are available to address the issue of using histology to determine time of occurrence, citing Janssen W., *Forensic Histopathology*. New York: Springer Verlag, 1984.

Unpublished study by Amy Baxter, MD, 2001, Children's Hospital of the King's Daughters, Norfolk, VA, on the rates of decline of AST and ALT in the four days after admission. They both decline post-injury. AST is higher early; ALT is higher late. ALT greater than AST is specific for an injury more than twelve hours old. In this retrospective search of 192 pediatric admissions with uncomplicated liver trauma, of which 16 were abuse. Recommend obtaining serial liver enzymes to form a curve to infer when the injury occurred. Graphs out to 96 hours. Have graphs saved in computer.

Delayed presentation of handlebar injuries in children. Lam JPH, Eunson GJ, Munro FD, Orr JD. BMJ 2001 May; 322: 1288-1289. Injuries to the spleen, liver, or K's are evident soon after acc; inj to the small intestine and pancr often present late and result in greater morbidity. Why? Because the first group of injuries cause blood loss, which irritates the peritoneum and produces early peritoneal signs. The second group of injuries do not cause any early peritoneal signs because there is no blood loss, the pH of the fluid is neutral, and the bacterial count is low; as a result, only 46-62% of these perfs have peritoneal signs at presentation. Bicycle injuries account for 5-14% of all BAT in children. Handlebar inj acct for 14-20% of small intestine perfs and 25-48% of all pancr inj in chil. Ultrasound suffers from false negatives. Also, there is very little air in the small intestine, so that x-rays are usually negative; only 15-46% have free air. Pts with major pancr inj usually have a persistently high amylase. "Repeated clinical examination remains the most important tool for early idagnosis; we recommend a period of observation for all chil who haave sx after such an inj."

Duodenal injuries in children: beware of child abuse. Gaines BA, Schultz BS, Morrison K, Ford HR. J Pede Surg 2004; 39: 600-602. From Dr Reece's review in the Autumn 04 issue of the *Quarterly*, had 30 cases of duodenal, with 8 being abuse. The most common single cause was auto accident. All the abuses had delayed convalescence due to infection due to delayed rescue.

Traumatic bowel perforation: analysis of CT findings according to the perforation site and the elapsed time since accident. Kim HC, Shin HC, Park SJ et al. J Clin Imag 2004; 28: 334-339. Had 57 patients of all ages (7 - 64) with confirmed traumatic bowel perforations. The time of injury was known. They classified them as "early" or "late" based on whether they were more or less than seven hours old by history. The radiologists retrospectively were able to tell the location of perforation (duodenal, jejunoileal, colonic) in 82% of the cases overall, but were much less accurate with colonic perfs than with UGI perfs. They were unable to distinguish early from late cases. The only single finding that came close to doing this was extraluminal air, which was present in 80% of the late cases and only 43% of the early cases.

ABDOMINAL RADIOLOGY

Abdominal CT in children with neurologic impairment following blunt trauma. Taylor GA and Eichelberger MR. *Ann Surg* 1989; 210: 229-233. Examines the role of neurological impairment as an indication for CT examination of the abdomen in children after blunt trauma. 482 patients. Found that they had a high frequency of thoracoabd trauma, but every child who had + abd injury on CT also had specific clinical abdominal signs. Therefore, coma by itself was a poor indication for abdominal CT.

AAP Policy Statement, Diagnostic imaging of child abuse (RE9204). *Pediatrics* 1991 Feb; 87(2): 262-264. (have) Skeletal. Intracranial. Thoracoabdominal.

Mesenteric injury from blunt abdominal trauma. Nolan BW, Gabram SG, Schwartz RJ, Jacobs LM. *Am Surg* 1995 Jun; 61(6): 501-506. Reviewed 5 years of trauma admissions with BAT in all age groups. 27 cases of BAT in all age groups. Found that mesenteric injury in BAT is difficult to diagnose; often missed by CT.

Massive chylous ascites and transected pancreas secondary to child abuse. Hilfer CL et al. *Pediatric Radiology* 1995; 25: 117-119.

Harris VJ, Lorand MA, Fitzpatrick JJ, Soter DK, *Radiographic Atlas of Child Abuse: a case studies approach.* New York: Igaku-Shoin, 1996. ISBN 0-89640-258-4. Presents radiographs in ten cases of abusive abdominal injury.

Pneumatosis intestinalis and portal vein gas after blunt abdominal trauma. Gurland B, Dolgin SE, Shlasko E, Kim U. *J Pediatr Surg* 1998 Aug; 33(8): 1309-1311. Case report of a 2 year old boy imaged after BAT by child abuse. The finding indicates mucosal injury. See also Koutouzis & Lee, 2000, *infra*.

CT appearance of clinically occult abdominal hemorrhage in children. Donnelly LF, Frush DP, O'Hara SM, Johnson ND, Bisset GS III. *AJR* 1998; 170: 1073-1076. This is not a study of abuse, but of bleeding from all causes. Intraluminal bleeding, intramural bleeding, solid-organ bleeding, and hemoperitoneum. Active bleeding sites are delineated by contrast enhancement. See Ken Feldman's review in *Child Abuse Quarterly* Oct 98.

Adrenal lacerations in child abuse: a marker of severe trauma. *Pediatr Surg Int* 2000; 16: 121. Comments on the abdominal CT findings of periportal tracking, adrenal hematoma, periadrenal hematoma, retroperitoneal streaky edema, retroperitoneal blood. See below under Abdominal Injury.

Portal venous gas: an unusual finding in child abuse. Wu JW, Chen MY, Auringer ST. *J Emerg Med* 2000 Jan; 18(1): 105-107.

Diagnostic imaging of child abuse. AAP, Section on Radiology. *Pediatrics* 2000; 105: 1345-1348.

Blunt abdominal trauma resulting in pneumatosis intestinalis in an infant. Koutouzis T, Lee J. *Ann Emerg Med* 2000; 36: 619-621. A table fell on this two year old. Ludwig (*Child Abuse Quarterly*, fall 2001) comments that the article does not detail that child abuse was ever ruled out in this case. Apparently there have been two previous reported cases, both child abuse -- one of them being Gurland's case from 1998, *supra*.

Imaging of child abuse. Nimkin K, Kleinman PK. *Radiol Clin N Amer* 2001 Jul; 39(4): 843-864.

Imaging in suspected child abuse: what to expect and what to order. Care M. *Pediatric Annual* 2002 Oct; 31(10): 651-659.

Radiology of nonaccidental trauma. Rustamzadeh E, Truwit CL, Lam CH. Neurosurg Clin N Amer 2002 Apr; 13(2): 183-199. Review article.

Brogdon BG, Vogel H, McDowell JD. *A Radiologic Atlas of Abuse, Torture, Terrorism, and Inflicted Trauma.* CRC Press, 2003. (In Neuro lab)

Child Abuse: radiologic-pathologic correlation. Lonergan AJ, Baker AM, Morey MK, Boos SC. From the Archives of the AFIP. RadioGraphics 2003; 811-845. (See also above under "Fractures -- in general.") Visceral injury -- small bowel, pancreas, other. Small bowel injury tends to occur in the duodenum and proximal jejunum. Perforation presents with pain and fever. Only 33% had free air on CT. Ascites is the most common CT finding [but in this context, the term "ascites" means either blood, purulent fluid, or pancreatic-related fluid. JKR] Children with hematoma of the bowel wall present most often with pain and vomiting from obstruction. Pancreatic injury presents with vomiting, fever, and elevated amylase. Pancreas was normal on CT in 71% of patients. The most common CT finding was fluid.

Traumatic bowel perforation: analysis of CT findings according to the perforation site and the elapsed time since accident. Kim HC, Shin HC, Park SJ et al. J Clin Imag 2004; 28: 334-339. See above under "Time of injury -- abdomen."

BLADDER

Child abuse presenting as pseudorenal failure with a history of a bicycle fall. Yang JW, Kuppermann N, Rosas A. (Cal Davis). Ped Emerg Care 2002 Apr; 18(2): 91-92. Bladder rupture.

CHEST INJURY

Homicidal cardiac lacerations in children. Cohle SD, Hawley DA, Berg KK et al. JFS 1995; 40: 212-218.

Homicidal commotio cordis in children. Denton JS and Kalelkar MB. JFS 2000; 45: 734-735.

Reece, Child Abuse: Medical Diagnosis and Management, 2d ed. (2001). P. 160, cardiac trauma: "Direct cardiac injury is rare in pediatric trauma and in particular in child abuse." (citing Marino & Langston, case report, 1982) Cites Dowd & Krug, Pediatric blunt cardiac injury, 1996, that over a ten year period there were only 184 chil younger than 18 years in 16 trauma centers, of which only three were from an assault. There were rib fractures in 23% of the 184 pts. P. 457, lacerations: (in chapter entitled, "Unusual manifestations of child abuse): Cohle Hawley, six cases, (above). All had evidence of other significant trauma. "The authors pointed out that this type of injury is the result of motor vehicle accidents or very violent assault, and not minor trauma." P. 483, intrathoracic injuries, in the chapter, "Pathology of child abuse," by the late Robert H. Kirschner and Harry Wilson: "Cardiac injury is rare. Abusive cardiac trauma usually presents as a laceration of the heart, most commonly at the junction of the vena cava with the right atrium, but also may involve the left ventricle. (citing Cohle Hawley). Presents a case of an 11 month old who suddenly became unresponsive in the care of a babysitter who had previously injured two other children. Victim had a lacerated RA at its junction with the IVC with 80 – 100 ml hemopericardium. Victim had abrasions of the face and small contusions of varying ages on the chest and abdomen. "The defense retained a forensic pathologist who claimed that the child had died of a 'viral' illness, although no evidence of such was noted clinically or at autopsy. He attributed the atrial laceration to CPR. The jury failed to bring in a conviction." Dr Kirschner goes on to refute the viral theory on anatomical and clinical grounds, and to refute the CPR theory in part on the basis that "[D]espite thousands of episodes of CPR of infants by untrained persons, we are unaware of any documented cases of cardiac laceration during this procedure. A single case report of cardiac laceration after CPR provides a sketchy clinical history that is open to question (Reardon et al., 1987). The force necessary to produce such an injury is clearly beyond the bounds of therapeutic chest compression. It is most likely caused by stomping on the child or by severe

prolonged compression with the hands.” Presents another case of a blow to the sternum with a telephone handset by a babysitter that was successfully resuscitated and had an intramyocardial hematoma of the anterior IVS. Discusses *commotio cordis*, refers to sports-related cases reported by Maron et al. and others. But “We have seen several cases of children who have similarly collapsed and died after receiving an inflicted blow to the sternum of only moderate force... There is usually no evidence of chest wall contusion, and there is no evidence of physical trauma to the heart.”

Marvin S. Platt, Daniel J. Spitz, and Werner U. Spitz, The abused child and adolescent. In: Spitz & Fisher's Medicolegal Investigation of Death, 3d ed. (2006), p. 394. “Although abusive chest injuries are less common than abdominal injuries (citing Cohle, Hawley, homicidal cardiac lacerations, (above) and Denton, two cases of abusive commotio, 2000, above), a blow to the chest can cause a contusion or laceration of the heart, cardiac dysrhythmia, and cardiac arrest. Victims of chest trauma are typically struck with a fist, stomped, and/or kicked.” Reviews Cohle Hawley's six cases of homicidal cardiac lacerations -- five right atrial lacerations. Mechanism could be direct chest trauma, indirect abdominal trauma, puncture by a fractured rib, or delayed rupture of an earlier necrotic area. Five of their cases had rib fractures. Book says most chest trauma victims have other evidence of abuse. “The scene investigation and comments made by the caregiver were critical in the workup. External findings in such child may include ev of poor nutrition and dehydration, as well as recent and remote bruises in the skin indicating ongoing abuse and neglect... careful search for injuries... Take sections of the chest wall skin and of the anterior walls of the heart for occult hemorrhages or injuries. Discusses accidental fatal cardiac injuries, which are much more common than abusive. A bruise to the chest wall may or may not be present. Sports, traffic, falling objects. Mentions the CPR defense and cites Price et al., 2000, for the proposition that this happens rarely if at all.

Flail chest in a neonate resulting from nonaccidental trauma. Gipson CL, Tobias JD. Southern Medical Journal 2006; 99: 536-538. Acc to Dr Brooks's review in the Autumn 2006 *Quarterly*, this 21 day old male neonate whose father admitted grabbing the child, presented with a flail chest and also a femur fracture and a pelvic fracture. Survived, placed in a foster home.

CPR

Child abuse, CPR, and rib fractures. Feldman KW and Brewer DK. Pediatrics 1984; 73: 339-342. Cited by Dr Reece (2002) for the proposition that CPR cannot cause posterior rib fxx bc it does not bring the rib arc posterior to the plane of the transverse process.

Visceral injuries following external cardiac massage. Barrowcliffe MP. Anesthesia 1984; 39: 347-350

RH after CPR. Kanter RK J Pediatr 1986; 108: 430-432

Mesenteric laceration complicating the Heimlich maneuver. (letter) Ann Emerg Med 1986 Jan; 15(1): 105-106 A 56 year old man aspirated an aspirin tablet at home and received a bystander abdominal thrust which was excessively forceful. Autopsy showed a 13 cm tear in the root of the mesentery and 3L of blood in the peritoneal cavity. The tablet was successfully dislodged by the maneuver.

Rupture of the esophagus caused by the Heimlich maneuver. (letter) Ann Emerg Med 1986 Jan; 15(1): 106-7. A 62 y o man.

Resuscitation and petechiae. Hood I, Ryan D, Spitz WU. Am J Forens Med P 1988; 9: 35-37. Petechiae occur in adults from CPR. (But not in infants, according to Byard & Krous, 2001, see under “Petechiae,” above.)

Gastric trauma following CPR. Hulewicz B. Med Sci Law 1990; 30(2): Case report of a 5 month old female SIDS case where autopsy showed a 2 ¾ inch vertical midline laceration of the anterior wall of the

stomach with postmortem spillage of milk as a result of insufflation during CPR. The author was able to reproduce the lesion in cadavers by transesophageally insufflating the stomach.

RH in an infant after CPR. Weedn VW, Mansour AM, Nichols MM. Am J Forens Med Path 1990; 11: 79-82

Patterns of facial resuscitation injury in infancy. Kaplan JA and Fossum RM. Am J Forens Med Pathol 1994; 15(3): 187-191 Nine cases described. Facial abrasions, under-chin abrasions, refs.

Rib fractures in children: resuscitation or child abuse? Betz P, Liebhardt E. Int J Legal Med 1994; 106(4): 215-218. Cited by Reece (2002) for the proposition that CPR cannot cause posterior rib fxx bc it is on a flat surface and therefore does not cause the posterior rib arc to pass the horizontal plane of the transverse process.

Cardiopulmonary resuscitation and rib fractures in infants: a postmortem radiologic-pathologic study. Spevack MR, Kleinman PK, Belanger PL, Primack C, Richmond JM. JAMA 1994; 272(8): 617-618. Citeg by Reece (2002) for the proposition that CPR does not cause posterior rib fxx bc it does not cause the rib arc to pass the plane of the transverse process.

Pediatric injuries from CPR. Bush CM, Jones JS, Cohle SD, Johnson H. Ann Emerg Med 1996 Jul; 28(1): 40-44 Studied 211 PICU pts who died and had in-hosp CPR. SIDS, drown, CHD, pn. Only 3% had medically significant injuries from CPR at autopsy: 2 retroperitoneal hemorrhages, 1 pneumothorax, 1 pulmonary hemorrhage, 1 epicardial hemorrhage, 1 gastric perf, 1 CCJ rib fx. "Significant iatrogenic injuries are rare in children who receive CPR; they occur in approximately 3% of cases.... Regardless of resuscitation history, abuse should be considered whenever traumatic injuries are encountered."

Artifactual injuries of the larynx produced by resuscitative intubation. Raven KP, Reay DT, Harruff RC. Am J Forens Med Path 1999 Mar; 20(1): 31-36. Numerous petechiae of the laryngeal and epiglottic mucosa, contusions of the base of the tongue, and hemorrhages of the thyrohyoid m. "[W]e conclude that resuscitative intubation can cause artifactual injury that may mimic inflicted injuries caused by neck compression, including strangulation and neck holds."

Artifactual injuries of the larynx produced by resuscitative intubation. Raven KP, Reay DT, Harruff RC. Am J Forens Med Path 1999; 20: 31-36. Did neck dissections on 50 adult cases. Ten had ocular petechiae. Two had neck skin abrasions. None had lx fxx. 7 had strap m hems, 18 had Lx mucosal contus etc. Inj mimic strang Be cautious.

ASCP Check Sample FP vol 42, no. 5, 2000, FP 00-5 BAT in Child Abuse, by K. D. Hutchins and Geetha Natarajan of New Jersey ME. "Severe injury of abdominal organs occurs rarely, if ever, during CPR. Isolated injuries including gastric perforation, liver and spleen laceration, and hemoperitoneum have been reported, but these injuries are rarely medically significant." (citing Bush, Jones & Cohle, 1996.)

Characteristics of child abuse by anteroposterior manual compression versus cardiopulmonary resuscitation: case reports. Gunther WM, Symes SA, Berryman HE. Am J Forens Med Path 2000 Mar; 21(1): 5-10. Shows finger bruises and nail marks on the anterior chest from gripping from behind, with posterolateral rib fxx on the inner curvature plus anterior callus fractures. All victims had CPR. One had hemosiderin in lungs.

CPR – related injuries and homicidal BAT in children. Price EA, Rush LR, Perper JA, Bell MD. Am J Forens Med Path 2000 Dec; 21(4): 307-310. Does closed-chest CPR result in fatal BAT that can be mistaken for child abuse injuries? Answer: no. This retrospective study of all children over a 17 year period in Florida, excluding head injury deaths. 33 child homi's with fatal BAT, of which 24 received CPR. 324 naturals, all had CPR. No traumatic intra-abdominal injuries were found in any of the naturals. As far as the homis, no detectable differences were observed between the abdominal injuries of the 24 who had CPR and the 9 who did not have CPR. Conclusion: CPR does not cause abdominal injuries. Cited by

Marvin S. Platt, Daniel J. Spitz, and Werner U. Spitz in Spitz & Fisher, ed ed. (2006), p. 395 for the proposition that this “occurs rarely if at all.”

Jane D. Kivlin, Ophthalmic Manifestations of Shaken Baby Syndrome. In: Lazoritz S and Palusci VJ, eds., *The Shaken Baby Syndrome: A Multidisciplinary Approach*. New York: The Haworth Press, 2001, p. 142-143. Brief review of the literature. “No patient who has had traumatic or atraumatic CPR has been found to have the extensive number and degree of hemorrhages that shaken babies commonly have. Thus extensive, numerous hemorrhages, particularly with large subhyaloid hemorrhages or a vitreous hemorrhage are very unlikely to have been caused by CPR.”

Reece, Child Abuse: Medical Diagnosis and Management, 2d ed. (2001). P. 483, intrathoracic injuries, in the chapter, “Pathology of child abuse”, by the late Robert H. Kirschner and Harry Wilson: “Cardiac injury is rare. Abusive cardiac trauma usually presents as a laceration of the heart, most commonly at the junction of the vena cava with the right atrium, but also may involve the left ventricle. (citing Cohle Hawley). Presents a case of an 11 month old who suddenly became unresponsive in the care of a babysitter who had previously injured two other children. Victim had a lacerated RA at its junction with the IVC with 80 – 100 ml hemopericardium. Victim had abrasions of the face and small contusions of varying ages on the chest and abdomen. “The defense retained a forensic pathologist who claimed that the child had died of a ‘viral’ illness, although no evidence of such was noted clinically or at autopsy. He attributed the atrial laceration to CPR. The jury failed to bring in a conviction.” Dr Kirschner goes on to refute the viral theory on anatomical and clinical grounds, and to refute the CPR theory in part on the basis that “[D]espite thousands of episodes of CPR of infants by untrained persons, we are unaware of any documented cases of cardiac laceration during this procedure. A single case report of cardiac laceration after CPR provides a sketchy clinical history that is open to question (Reardon et al., 1987). The force necessary to produce such an injury is clearly behind the bounds of therapeutic chest compression. It is most likely caused by stomping on the child or by severe prolonged compression with the hands.”

What the literature tells us about rib fractures in infancy. Reece RM. SBS Quarterly, Fall 2002, pp. 2,3,6. See above under “Rib fractures.”

1. It takes “major forces” to fracture infant ribs bc of the flexibility (citing Garcia, Gotschall, 1990, which found that rib fxx were associated with the most severe trauma in a retrospective study of injured children 0-14 years of age.)

2. CPR or being stepped on, or other flat-surface compression does not cause posterior rib fxx bc Kleinman’s “lever arm” mechanism (1987a) involving the transverse process requires that the rib pass the horizontal plane of the transverse process in order for a frac to happen. (citing Feldman & Brewer, 1984; Betz, Liebhardt, 1994; Spevack, Kleinman, 1994).

Do resuscitation attempts in children who die cause injury? Ryan MP, Young SJ, Wells DL. J Emerg Med 2003; 20: 10-12. Australians did autopsies on 204 children who died of non-trauma: 153 resusc, 51 no resusc.

	Resusc	No resusc
Injury found at autopsy	42%	12%

The resusc injuries were 63 superficial bruises or abrasions, 23 ecchymoses from IV’s, 18 airway injuries, 9 lip injuries, 7 pulmonary contusions, 1 splenic hematoma, and 1 broken tooth. Obviously there were no fatal or life-threatening injuries, and that is the point.

Knight’s Forensic Pathology, 3d Edition (2004). “...[I]n lesser degrees of trauma, it may be impossible to differentiate deliberate battering from inexpert rough handling, albeit contributed to by exasperation, panic or even attempts at resuscitation.” (p. 461)

Skeletal chest injuries secondary to CPR. Hoke RS, Chamberlain D. Resuscitation 2004; 63: 327-338. A literature review of five articles on CPR in pediatric populations. Finds that out of 770 children undergoing CPR, there were three cases of rib fractures. Two of these were infants (SIDS cases). One was a five year-old. One of the SIDS cases, aged 3 months, had CCJ fractures. The other SIDS case, aged 2 months, had fractures in the MCL. Paul Kleinman states in his review for the Winter 2006 *Quarterly*, "The authors concluded that manual CPR rarely causes skeletal chest injuries in infants and toddlers and that posterior or lateral rib fractures have never been described."

Resuscitation injuries complicating the interpretation of premortem trauma and natural disease in children. John Plunkett. JFS 2006 Jan; 51(1): 127-130. Case report of two children (a 6 year-old and a 21 month-old) with resuscitation injuries that were initially interpreted as homicidal but later shown to be CPR. Murder charges were filed and then dismissed in case 1 and led to an acquittal in case 2.

In his introduction, Dr Plunkett reviews the published reports of significant CPR trauma to infants: right **atrial rupture** (Reardon et al., 1987), tracheal perforation and gastric rupture (Hulewicz, 1990), hepatic, splenic, and pancreatic lacerations (Waldman et al., 1985), and retroperitoneal hemorrhage (Corbett et al., 1997). Also Krischer et al., 1987, finding liver and spleen lacerations and ruptured stomach.

Case #1: A 6 year old boy who according to the coroner's investigation was in good health was alone with mother's bf and found him collapsed at 1 pm. The 911 operator instructed the bf in CPR. Autopsy showed a 2.5 cm laceration of the liver, a 2 cm laceration of the right adrenal gland, and 300 ml of intraabdominal blood. No vital reaction histologically. Boyfriend charged with murder. Defense investigation revealed that the boy had unstable asthma and was on albuterol. Lung histology showed severe asthma with interstitial emph. Charges dismissed.

Case #2: A 21 month old boy living with mother and mother's bf, said to fall out of bed at 8:15. (19" to carpeted floor.) Taken by private car to hospital, hypotensive, bradycardic, rectal temp 91.7, CT head showed a depressed fracture of the R temporal bone and temporal lobe lacerations and SDH which continued to enlarge. An abdominal CT performed 2.5 hours after admission was normal. At 4.5 hours after admission the child arrested and had CPR, followed by four more arrests with CPR. Finally expired 17h after admission. Autopsy showed abraded contusions of the lips, multiple cutaneous contusions, a fresh posterior fracture of the right 10th rib, 100 ml of intraperitoneal blood, a splenic laceration, 300 ml retroperitoneal hemorrhage, ecchymosis of the scrotum, and a healing fracture of the right inferior pubic ramus. Bf was indicted for murder and they would seek the DP. BUT: Defense showed that the coroner had failed to know about or consider the abdominal CT scan, which showed no hemorrhages, rib fractures, or injuries. The jury acquitted.

Author's discussion: In case #1, the pathologist was not told that the boy had asthma, or that he was seen alive & well just shortly before the death. In case #2, the pathologist failed to consider the abdominal CT scan, which made it highly unlikely that the intraabdominal trauma was present at the time of the CT scan, and the lip and scrotal injuries could have been from CPR. Dr Plunkett has a hard time explaining the pubic fracture or the multiple bruises. He explains away the skull fracture by suggesting that a 3 ½ year old brother might have jumped on the boy, but this explanation came forth only years afterward. The author concludes concerning the legal evaluation of the head injury, that "It was likely ... that a single unidirectional force caused the head injury, allowing a reasonable nonabuse explanation to be considered." Author's conclusion: "Resuscitation is an uncommon if not rare cause for significant injury in a child. Most cases that appear to be abuse are abuse. However, some are not. The only way to differentiate between the alternative conclusions is to consider the unique characteristics for each case. If there is new information, the conclusion must be re-evaluated."

GLUTARIC ACIDURIA TYPE I

(see also Hydroxyglutaric aciduria, below)

Diagnosis and management of glutaric aciduria type I. Baric I, Zschocke J, Christensen E et al. J Inher Metab Dis 1998; 21: 326-340 (have). Autosomal recessive congenital deficiency of glutaryl co-A dehydrogenase. 1:30,000. Common in the Amish and in Arabs and possibly Sephardic jews. Accumulates glutaric acid and 3-hydroxyglutaric acid. Mutation of a small enzyme gene on the short arm of

chromosome 19. The clinical features are congenital macrocephaly, subtle early developmental delay, a sudden catastrophic deterioration (coma, seizures, subdural bleeding) at age 4-18 months, often in response to a fever, followed by a characteristic picture of athetoid cerebral palsy with CT atrophy of the temporal and frontal lobes with widening of the lateral fissures and atrophy of the basal ganglia, most notably the caudate nucleus and putamen. CT scans at that stage show subdural effusions with or without acute blood. A high index of suspicion should be triggered by a suggestive family history (e.g. previous SIDS or mental retardation), macrocephaly. If a high index of suspicion, do urinary organic acids or quantitative urinary glutaric acid (these are 80% SENS), or serum or vitreous quantitative glutaric acid level. If a low index of suspicion, may do Neo-Gen only; a low or negative index of suspicion is appropriate if no macrocephaly and no characteristic CT picture and no family history. If a very high index of suspicion, get metabolic disease consult. GA-1 is in the differential dx of SBS (and a significant index of suspicion exists) only if the clinical findings of trauma are equivocal; it is not in the DDX if there is a large acute SDH, characteristic RH, or other clearcut indicia of physical abuse. [Above summary thanks to oral presentation by Stephen Goodman MD and Andrew Sirotiak MD at the Second National SBS Conference, 1998].

Phenotypic variability in glutaric aciduria type I: report of fourteen cases in five Canadian Indian kindreds Haworth JC, Booth FA, Chudley AE et al.. J Pediatr 1991; 118: 52-58

Subdural haematoma in a child with glutaric aciduria type I. Kohler M. Pediatric Radiology 1998; 28: 582

Subdural hemorrhage as an initial sign of glutaric aciduria type I: a diagnostic pitfall. Woelfle J, Kreft B, Emons D, Haverkamp F. Pediatric Radiology 1996; 26: 779-781

Glutaric aciduria and suspected child abuse. Morris AAM, Hoffmann GF, Naughten ER, Monavare AA, Collins JE, Leonard JV. Arch Dis Child 1999 May; 80(5): 404-405. Discusses signs which also occur in AHT. GA-1 usually presents with the sudden onset of severe encephalopathy during infancy (as summarized by Betty Spivack). CT shows frontotemporal atrophy, widening of the lateral fissures, and sometimes SDH. RH have also been reported. They never have fractures. They never have SDH unless there is frontotemporal atrophy. Get urinary organic acids, blood glutaryl carnitine, total plasma carnitine, and free plasma carnitine. Then confirm dx with cultured leukocyte or fibroblast glutaryl CoA dehydrogenase activity. If there is no frontotemporal atrophy, need not bother to work up for GA-1.

Glutaric aciduria type I: ultrasonic demonstration of early signs. Forstner R, Hoffman GF, Gassner I et al. Pediatr Radiol 1999; 29: 138-143. Macrocephaly and anterior frontotemporal cerebral atrophy are the early signs within the first few weeks of life. Then you look for glutaric acid in the urine, and the diagnosis is made.

Ocular findings in glutaric aciduria type I. Kafil-Hussain NA, Monavari A, Bowell R et al. J Pediatr Ophthalm Strab 2000; 37: 289-293. Studied 15 living patients aged one week to 24 months. One pt had RH; this was a pt with the typical acute encephalopathic crisis of the disease.

Atypical and variable clinical presentation of glutaric aciduria type I. Zafeiriou DI et al. Neuropediatrics 2001; 31: 303-306.

Glutaric aciduria type I and nonaccidental head injury. Hartley LM, Khwaja OS, Verity CM. Pediatrics 2001; 107: 174-176. Report a case: eight week old who was dropped. Bilateral possibly chronic subdural collections with an acute L parietal SDH, no fx, one tiny RH, SPNBF R humerus, retarded mother known to DCFS. GA1 confirmed by fibroblasts. So: possible NAI on top of GA1. The overall frequency of GA1 is 1/30,000, but watch out for discrete subpopulations and kindreds.

Kathy Makaroff of Cincinnati Children's has published a literature review as of August 2001.
http://www.chmcc.org/programs_services/152/tools/update.asp

A 9-month-old baby with subdural hematomas, retinal hemorrhages, and developmental delay. Soden SE, Dasouki MJ, Walsh IR. Pediatr Emerg Care 2002 Feb; 18(1): 44-47. Case report of GA-1. From Dr

Sirotnak & Reece's review in the *Quarterly* for January 2003: 9m fell backwards and hit his head, immed LOC. CT neg, sent home. Next day vom, sent home again. 2d later vom CT sh subacute parietal SDH; sent home. Six weeks later fell sz LOC: CT acute SDH + extraaxial fluid collections. Neom seen. Devel delay ID'd. Skel surv neg. GA-1 dxd by chemistry. The reviewers point out that Dr Walsh is a pediatric ophthalmologist and she discusses the RH aspect of this. [She was the author of the RH section in the first edition of Reece's textbook. -JKR]

Intraretinal hemorrhages and choric subdural effusions. Glutaric aciduria type I can be mistaken for shaken baby syndrome. Gago LC, Wegner RK, Capone A Jr., Williams GA. *Retina* 2003; 23: 724-725. Case report of a 6 month old boy who presented with developmental delay, an enlarging head, and subdural effusions. Gives good fundus photographs. Intraretinal and vitreous hemorrhages.

GLUTARIC ACIDURIA TYPE II

See page 850 in *Potter's Pathology of the Fetus and Infant*. This is a form of fatty acyl-Co-A dehydrogenase deficiency. "is associated with deficiency of several mitochondrial acyl-co-A dehydrogenases and is characterized by acidosis, nonketotic hypoglycemia, organic aciduria, hyperammonemia, and accumulation of lipid in the liver, myocardium, and renal tubular epithelium." See also our refs to disorders of fatty acid oxidation, under SIDS BIOLOGY, above.

D-2-Hydroxyglutaric aciduria and subdural haemorrhage. Kwong KL, Mak T, Fong CM, Poon KH, Wong SN, So KT. (HK) *Acta Paediatrica* 2002; 91(6): 716-718. SDH had not previously been reported in this rare disorder. Case report of a boy with bilateral SDH in whom NAI was initially suspected. No further details in the abstract.

HYDROXYGLUTARIC ACIDURIA

D-2 Hydroxyglutaric aciduria and subdural hemorrhage. Kwong KL, Mak T, Fong CM, Poon KH et al. (Hong Kong) *Acta Paediatrica* 2002; 91(6): 716-718. The first reported case of SDH in this rare disease. Nonaccidental injury was initially suspected.

MENKES' DISEASE

see also under DDX

Massive subdural hematomas in Menkes disease simulating shaken baby syndrome. Nassogne M-C, Sharrard M, Hertz-Pannier L et al. *Child's Nervous System* 2002; 18: 729-731. See Dr Reece's review in the *Quarterly* for April 2003. A male infant 9 weeks old who presented with dehydration, hypothermia, and sz. CT sh bilateral multifocal hypodense areas. No RH. +low ceruloplasmin, +high intracellular copper. Child went on to develop macrocephaly, cerebral atrophy with extraaxial fluid collection, hypotonia, pyramidal syndrome. Dr Reece characterizes it as a classical case of Menkes disease in which SBS was never in the differential.

Knight's Forensic Pathology, Third Edition (2004) has a brief discussion under "bone diseases" in Chapter 22, "Fatal Child Abuse."

PYRUVATE CARBOXYLASE DEFICIENCY

Presents with lactic acidosis, elevated lactate: pyruvate ratio, ketonuria, hyperammonemia, normal glucose, normal LFT's. May do serum amino acid levels. Definitive dx depends on skin fibroblast culture. These patients have a "crisis" followed by significant cerebral atrophy (reminds us of GA-1). The crisis tends to occur during an intercurrent illness. Between crises, their levels will be normal, making the condition notoriously difficult to diagnose. Thanks to Betty Spivack MD for this info. It enters the DDX of child abuse because there have been reports of subdural hematoma in this condition.

Treatment of pyruvate carboxylase deficiency... Ahmad A et al. Am J Med Genet 1999; 87: 331-338. Presents an 11 week old infant with a R frontal SDH, cerebral atrophy, no coagulopathy, presented febrile with limpness, myoclonus. The authors refer to two previous reports of ICH and state that condition could be confused with SBS. Thanks to Dr. Alex Levin for this info. See:

Rutledge SL et al, *Pediatr Neurol* 1989; 5: 249-252 (SAH)

Hoffman GF et al, *Neuropediatr* 1996; 27: 115-123 (SDH)

BIOTINIDASE DEFICIENCY

A group of autosomal recessive disorders of the release of biotin from its carrier protein. Presents as biotin deficiency with limpness, seizures, mental retardation, platybasia, and tomato urine. See Nelson's Pediatrics and material in file box.

COAGULATION DISORDERS

See also under DDX -- Specific Entities

Disorders of coagulation misdiagnosed as nonaccidental bruising. Jim R Harley. *Pediatr Emerg Care* 1997; 13: 347-349. Two cases of ITP and one hemophilia that were dx'd as child abuse. Subtle morphologic features distinguish, but also need coagulation studies.

Persistent scalp bleeding due to fetal coagulopathy following fetal blood sampling. Pachydakis A, Belgaumkar P, Sharmah A. *Int J Gyn Ob* 2006; 92(1): 69-70. This one-page case report shows that a fetus bled profusely from the scalp sampling wound because of an unspecified fetal coagulopathy.

HEADBANGING and SELF-MUTILATION (JACTATIO NOCTURNA)

In normal children is considered to be a developmental disorder. But it occurs in severe mental retardation, traumatic encephalopathy, Gilles de la Tourette syndrome, possibly in the Lesch-Nyhan syndrome, and other metabolic disorders, also seen in medical examiners.

Also called "jactatio nocturnis." It is not benign; it can cause significant head injury, including subdural hematoma.

Jactatio nocturna after head injury. Drake ME Jr. *Neurology* 1986 Jun; 36(6): 867-868. A sleep disorder, usually a developmental disorder. Here reported in a patient with global encephalopathy and frontal lobe dysfunction secondary to an unspecified closed head injury.

EEG and brainstem auditory evoked potentials in brain-injured patients with rage attacks and self-injurious behavior. Cannon PA, Drake Me JR., *Clin Electroencephalogr* 1986 Oct; 17(4): 169-172. Did these tests on 10 normals, 10 TBI with rage attacks, and 10 TBI with self-injurious. EEG was nondiscriminatory. BAERS were abnormal.

Self-injurious behavior and the Gilles de la Tourette syndrome: a clinical study and review of the literature. Robertson MM, Trimble MR, Lees AJ. ----- 1989 Aug; ----- : 611-625 Studied 90 patients, of whom 30 had self-injurious behavior. 14 headb. **One nonstudy pt (reported in detail) died of fatal SDH due to headb.** 3 had permanent visual imp due to injuring their own eyes.

Neuropathological observations in a case of autism presenting with self-injury behavior. Hof PR, Knabe R, Bovier P, Bouras C. Acta Neuropathol (Berlin) 1991; 82(4): 321-326. 24 year old severely autistic woman with headbanging since childhood, at autopsy had neurofibrillary tangles in various areas including the orbitofrontal cortex and the amygdala. No neuritic plaques or amyloid. Compares it as similar to those observed in boxers and soccer players.

Children with self-injurious behavior. Cataldo MF. Pediatrics 1990 Mar; 85 (3 Pt 2): 437-441. A serious problem not uncommon in severe mental retardation. Here reports 97 children and young adults, predominantly school-aged. Headbanging, biting, head hitting, body hitting, scratching. 77% had physical injury, including hematomas, scars, excoriations.

The coming of age of self-mutilation. Favazza AR. J Nerv Ment Dis 1998 May; 186(5): 259-268. Divided by this author into four forms: Cultural SM such as tattooing. Major SM such as self-castration is associated with psychosis. Stereotypic SM such as headbanging and self-biting seen in Gilles de la Tourette syndrome and severe mental retardation. Superficial-moderate SM such as trichotillomania, cutting and burning is psychiatric.