

THE MEDICAL LITERATURE OF CHILD ABUSE: AN ANNOTATED BIBLIOGRAPHY
MAY 2007 EDITION

James K. Ribe, MD, JD
Senior Deputy Medical Examiner
Los Angeles County Department of Coroner
jribe@coroner.co.la.ca.us

Updated 5/19/07

Definitions	4
Reporting	6
Classic articles	6
Differential diagnosis	8
Specific disease entities	12
Metabolic disease screening	20
Epidemiology and risk factors	20
Birth injury	31
Fetal injury	34
Battered child syndrome	36
Bruises	38
Fractures	42
In general	
Femur	
Ribs	63
Flail	
Tibia	
Pelvis	
Clavicle	
Humerus	
Back	
Mandible	
OI and Rickets	73
TBBD	78
Folk customs	81
Ritual abuse	81
Burns	82
MSBP	86
Medea complex	89
MCAD	89
SUDC	92
SIDS v. Suffocation	95
SIDS biology	122
SIDS genetics	160
SIDS epidemiology	163
Twin SIDS	173
Cosleeping	174
Pacifiers	186
Blood in nose/ face	186
Petechiae	188
Pulmonary siderophages	193
ALTE	204
Central hypoventilation	207
Brugada's syndrome	208

Car seats	209
Infant botulism	211
Recurrence risk	212
Vaccination	212
Smoking	213
Neomembranes	213
Cervical Spine/ spinal cord	214
Cardiac (+ Stress Cardiomyopathy)	217
Autopsy technique	220
Retinal hemorrhages (RH)	225
Unusual causes	226
In general	231
Histology	263
Optic nerve sheath hemorrhage (ONSH)	270
Unilateral	272
CPR	274
Seizures	276
SIDS	276
Traumatic retinoschisis (TR)	277
Terson's syndrome	286
Vitreous hemorrhage	299
Purtscher's syndrome	300
Birth hemorrhages	301
Hemosiderin	303
Long term sequelae	303
Shaken (= AHT)	304
Biomechanics	377
Head injury -- fall versus inflicted	390
Time of injury -- head	437
Bunk beds	449
Walkers	451
Dura mater and neomembranes	463
Enlarged subarachnoid space	463
Vaccine defense to SBS	465
Cerebral edema	466
Tin ear	466
Ears	466
Abdominal injury	466
Time of injury -- abdomen	475
Abdominal radiology	477
Bladder	478
Chest injury	478
CPR	480
Glutaric aciduria type I	483
Glutaric aciduria type II	484
Hydroxyglutaric aciduria	484
Menkes' disease	485

Pyruvate carboxylase deficiency	485
Biotinidase deficiency	485
Coagulation disorders	485
Headbanging and self-injurious behavior	486
Sexual abuse	486
Female circumcision	529
Cocaine babies	531
Foreign body	535
Drowning	536
Bath seats (tub seats & rings)	541
Buckets	542
Dental & Face	543
Esophagus	545
Strangulation and neck	546
Heat injury	548
Positional asphyxia	548
Neglect	549
Sibling abuse	560
Drug abuse	560
Domestic violence	561
Battered Women's Defense	565
Parental rights	566
Parental alienation syndrome	566
Water intoxication	567
Cough medicine	568
Stickler's syndrome	570
Sandifer's syndrome	570
Handicapped children	570
Foster child	571
Long term sequelae	571
Medicolegal	573
Child Death Review	575
Unclassifiable	576
Web sites	584

DEFINITIONS

California Penal Code Sec. 111656.6 "Child abuse means a physical injury which is inflicted by other than accidental means on a child by another person." The law defines child abuse as: (1) Physical abuse, (2) Physical neglect, (3) Sexual abuse, (4) Emotional abuse.

California Attorney General: <http://www.safestate.org>

The California Child Abuse and Neglect Reporting Act, Penal Code Sec. 11164-11174.3, as amended by SB525 (1999) (Chapter 1012, Statutes of 1999, as amended, 20020 defines the terms "child abuse," "child neglect," "general neglect," "severe neglect," "wilful cruelty to a child," "unlawful corporal punishment," "sexual abuse," "sexual assault," and "sexual exploitation." As to the sexual items, see below under "Sexual abuse." As to neglect, see below under "neglect." See also SB 580 (2003) which mandated use of certain protocol exam forms for physical abuse, now Sec 11161.2.

Sec. 11161.2 Mandated examination forms (SB 580)

Sec. 11165.1 Sexual abuse

Sec. 11165.2 Neglect

Sec. 11165.3 Willful cruelty or unjustifiable punishment

Sec. 11165.4 Unlawful corporal punishment defined.

Sec. 11165.5 Abuse in out-of-home care

Sec. 11165.6 Abuse defined: (As amended in 2000) “As used in this article, ‘child abuse’ means a physical injury that is inflicted by other than accidental means on a child by another person. The term, ‘child abuse or neglect’ includes sexual abuse as defined in Sec. 11165.1, neglect as defined in Sec. 11165.2, willful cruelty or unjustifiable punishment as defined in Sec. 11165.3, unlawful corporal punishment or injury as defined in Sec. 11165.4, and abuse or neglect in out-of-home care as defined in Sec. 11165.5. ‘Child abuse or neglect’ does not include a mutual affray between minors. ‘Child abuse or neglect’ does not include an injury caused by reasonable and necessary force used by a peace officer acting with the course and scope of his or her employment as a peace officer.”

Old Sec. 11165.6: REPEALED in 2000 said, “As used in this article, ‘child abuse’ means a physical injury which is inflicted by other than accidental means on a child by another person. ‘Child abuse’ also means the sexual abuse of a child or any act or omission proscribed by Sec. 273a (willful cruelty or unjustifiable punishment of a child) or 273d (unlawful corporal punishment or injury.) ‘Child abuse’ also means the neglect of a child or abuse in out-of-home care...”

Sec. 666.7(j)-6: Willfully causing or permitting any child to suffer, or inflicting on the child unjustifiable pain or injury that results in death under circumstances or conditions likely to produce great bodily harm or death, or, having care or custody of any child, willfully causing or permitting that child to be injured or harmed under circumstances likely to produce great bodily harm or death, when that injury or harm results in death.

Penal Code Sec. 273a-(a). “Any person who, under circumstances likely to produce great bodily harm or death, willfully causes or permits any child to suffer, or inflicts thereon unjustifiable physical pain or mental suffering, or having the care and custody of any child, willfully causes or permits the person or health of that child to be injured, or willfully causes or permits that child to be placed in a situation where his or her person or health is endangered, shall be punished by imprisonment in a county jail not exceeding one year, or in the state prison for two, four, or six years.

Sec. 273a-(b). “Any person who, under circumstances or conditions other than those likely to produce great bodily harm or death, willfully causes or permits any child to suffer, or inflicts thereon unjustifiable physical pain or mental suffering, or having the care and custody of any child, willfully causes or permits the person or health of that child to be injured, or willfully causes or permits that child to be placed in a situation where his or her person or health may be endangered, is guilty of a misdemeanor.

Sec. 273ab. “Any person who, having the care and custody of a child who is under eight years of age, assaults the child by means of force that to a reasonable person would be likely to produce great bodily injury, resulting in the child’s death, shall be punished by imprisonment in the state prison for 25 years to life. Nothing in this section shall be construed as affecting the applicability of subdivision (a) of section 187 or section 189.

Sec. 273d. “Any person who willfully inflicts upon a child any cruel or inhuman corporal punishment or an injury resulting in a traumatic condition is guilty of a felony...”

See also the California Elder and Dependent Adult Civil Protection Act, Welfare & Institutions Code Sec. 15610 et seq., which contains definitions of “physical abuse,” “neglect,” and “reasonable suspicion.”

California Office of Criminal Justice Planning. Required protocols for medical examination of physical and sexual abuse victims, Promulgated under the 2003 amendments to Sec. 11160 et seq., above.
<http://www.ocjp.ca.gov/>

The Child Abuse and Treatment Act (CAPTA) Public Law 104-235, Section III; 42 U.S.C. 51106(g) defines child abuse and neglect as any recent act or failure to act

- resulting in imminent risk of serious harm, death, serious physical or emotional harm, sexual abuse, or exploitation
- of a child
- by a parent or caretaker who is responsible for the child's welfare.

See Mignon SI et al., *Family Abuse: Consequences, Theories and Responses*. Boston: Allyn & Bacon, 2002, p. 19. Sexual abuse is also defined by CAPTA; see under Sexual Abuse.

Chapter 1 in Ludwig S and Kornberg AE, *Child Abuse: A Medical Reference, 2d ed.* New York: Churchill Livingstone, 1992. States that the definition is elusive because of differing cultural and individual standards. Legal definition: "Abuse is what the law says it is." (see below). Quotes Pennsylvania statutory definition as serious injury that is not explained by the history. Goes on to discuss other forms of abuse and neglect.

A national definition is offered by ivillage.com at <http://pages.ivillage.com/sacboard/>. "An 'abused child,' under the law, means a child less than 18 years of age whose parent or other person legally responsible for the child's care inflicts or allows to be inflicted upon the child physical injury by other than accidental means which causes or creates substantial risk of death or serious disfigurement, or impairment of physical health, or loss or impairment of the function of any bodily organ. It is also considered 'abuse' if such a caretaker creates or allows to be created situations whereby a child is likely to be in risk of the dangers mentioned above. Injuries may include:

- bruises
- welts
- cuts
- fractures
- burns
- internal injuries
- severe beatings
- biting
- strangulation
- scalding
- scars

"Physical abuse can be one or two isolated incidents or can occur over a prolonged period of time."

When inflicted skin injuries constitute child abuse. AAP Committee on Child Abuse and Neglect. Pediatrics 2002 Sep; 110-(3): 644-645. Any injury beyond temporary reddening of the skin. Because it is a precursor of more severe abuse. See below under "Bruises." This policy statement was RETIRED by the academy in October 2006. See notice published at page 405 of the February 2007 issue of *Pediatrics*.

Elder abuse and neglect. American Medical Association, Council on Scientific Affairs. JAMA 1987; 257: 966-971. "An act or omission that results in harm or threatened harm to the health or welfare of [an elderly person.]"

Elder abuse and neglect. Collins KA, Bennett AT, Hanzlick R, and the Autopsy Committee of the College of American Pathologists. Arch Int Med 2000 Jun; 160(11): 1567-1568. "Physical abuse is an act carried out with intention of causing physical pain or injury, such as hitting, slapping, or stalking with objects."

Knight's Forensic Pathology, Third Edition (2004), p. 461: "Though definitions are variable, it can be said that the 'child abuse syndrome' (also known as the 'battered baby' or 'non-accidental injury in childhood') exists when an infant or child suffers repetitive physical injuries inflicted by a parent or guardian, in circumstances that exclude accident."

REPORTING

Child Abuse Hot Line
CAHL
3075 Wilshire Blvd 5th floor 90010

800-540-4000

DCFS online reporting web site <http://dcfs.co.la.ca.us/>

PANDA Prevent Abuse and Neglect through Dental Awareness Hotline 501-661-2595, an important resource on dental abuse & neglect

CLASSIC ARTICLES

Etude medico-legale sur les services et mauvais traitements exerces sur des enfants. Tardieu A. (Auguste-Ambroise Tardieu) Ann Hyg Publ Med Leg 1860; 13: 361-398. Recognizing subdural hemorrhage as a form of child abuse. According to Dr. Tongue, Tardieu published the results of autopsies performed on 32 children who died of injuries, mainly inflicted by their parents. She cites the following secondary source: Heins M. The "battered child" revisited. JAMA 1984; 251: 295-300. Another discussion of this work is given in B. G. Brogdon's 1998 book, *Forensic Radiology* (see below). Brogdon presents a photograph of the first page of this paper, as well as the title page of Tardieu's 1879 book entitled *Etude Medico-Legale sur les Blessures*, in which this paper was reprinted. Brogdon's photograph (copied from Silverman's book, *q.v.*) shows the abstract (in French), which I will translate here as follows:

"Among the numerous and diverse findings which make up the medicolegal material of injuries and wounds, there exists a separate grouping which has been left in complete obscurity until now, and which deserves to be given a name in order to be brought to light. I am referring to the findings diagnostic of neglect and maltreatment, of which children are the particular victims at the hands of their parents, their teachers, and those who, in a word, exercise more or less direct authority over them."

Brogdon states that the paper presented 32 cases of child abuse and set forth "all the salient features," but that it had little impact at the time.

The book is described also in Reece & Ludwig, 2d ed. (2001) at 123, in chapter on skeletal manifestations by Daniel R. Cooperman and David F. Merten. These authors say that Tardieu "described multiple fractures and other injuries in children that were inflicted by parents or others with authority over the victims. This report appears to be the first inclusive concept of the medical, demographic, social, and psychiatric features of child physical abuse that would be defined more than a century later as the battered child syndrome." The work is also discussed by Chadwick et al. in the 2005 reference next below. It is also discussed in more detail in the articles by Labbe and Roche et al., below.

Ambroise Tardieu: The man and his work on child maltreatment a century before Kempe. Labbe J. Child Abuse & Neglect 2005; 29: 311-324. Reviewed by Mary Case in the Autumn 2005 issue of the *Quarterly*. Mary states that physicians did not believe Tardieu's diagnoses and dismissed his findings as due to fanciful accounts by children.

The work of Ambroise Tardieu: the first definitive description of child abuse. Roche AJ, Fortin G, Labbe J, Brown J, Chadwick D. (Montreal) Child Abuse & Neglect 2005 Apr; 29(4): 325-334. The first important monograph describing the battered child syndrome, 1860. French forensic physician. These authors present a complete translation, followed by a brief summary of the 32 cases presented in the work. Reviewed by Mary Case in the Autumn 2005 issue of the *Quarterly*.

Multiple fractures in the long bones of infants suffering from chronic subdural hematoma. Caffey J, Am J Roentg (AJR) 1946; 56: 163-173 (have). [cf. Lazoritz, Has Caffey's syndrome changed... Child Abuse & Neglect 1997; 21(10): 1009-1014.] See below under "Shaken." In this article Caffey considered

accidental as a possible mechanism, but broached the question of intentional injury. His later reading of Ommaya's 1968 work on acceleration brain injury led to the hypothesis of shaking.

The roentgen manifestations of unrecognized skeletal trauma in infants. Silverman FN. Am J Roentgenol (AJR) 1953 Mar; 69: 413-427. First proposed that children with multiple unexplained fractures may be the victims of trauma intentionally inflicted by their caretakers.

Some traumatic lesions in growing bones other than fractures and dislocations: clinical and radiological features. Caffey J. Br J Radiol 1957; 30: 225-238. The original description of corner fractures and bucket-handle fractures.

Slaughter of the innocents: study of forty six homicides in which the victims were children. Adelson L, NE J Med 1961; 264: 1345-1349

The battered-child syndrome. Kempe CH, Silverman FN, Steele BF, Droegmueller W, and Silver HK: JAMA 1962 Jul; 181: 17-24 (have) Symposium organized by Dr. Kempe of Denver, presenting the syndrome as repetitive injury inflicted by caregivers.

Infantile subdural hematoma and its relationship to whiplash injuries. Guthkelch AN, Br Med J 1971; 2: 430-431 (have)

Unrecognized trauma in infants, the battered child syndrome, and the syndrome of Ambroise Tardieu. The Rigler Lecture. Silverman FN. Radiology 1972 Aug; 104: 337-353. (have)

The parent-infant traumatic stress syndrome; Caffey J, Am J Roentgenol Radium Ther Nucl Med (AJR) 1972; 114: 216-229

On the theory and practice of shaking infants. Its potential residual effects of permanent brain damage and mental retardation. Caffey J. Am J Dis Child 1972; 124: 161-169

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; 54: 396-403

Cerebral concussion and traumatic unconsciousness: correlation of experimental and clinical observations on blunt head injuries. Ayub K, Ommaya and Gennarelli TA. Brain 1974; 97: 633-654

Injuries resulting when small children fall out of bed. Helfer RE, Slovis TL, Black M. Pediatrics 1977; 60: 533-535. See under "Head injury: fall v. inflicted." See also abstract given on p. 65 of the AAP *Guide to References and Resources in Child Abuse & Neglect, 2d ed.* (1998.)

Sexual abuse of children: a clinical spectrum. Summit R and Kryso J. Am J Orthopsychiatry 1978 Apr; 48(2): 237-251

The cutaneous manifestations of child abuse & neglect. Ellerstein NS, Am J Dis Child 1979; 133: 906-909

Subtle fatal child abuse. Zumwalt RE and Hirsch CS. Human Pathology 1980 Mar; 11(2): 167-174

The child sexual abuse accomodation syndrome. Roland C. Summit. Child Abuse & Neglect 1983; 7: 177. Identifying recantation as the standard course the child victims usually follow once the consequences of their disclosure become apparent to them. A copy of this article appears as an appendix to the two-volume ring-binder manual, *Investigation and Prosecution of Child Abuse, 2d ed.*, put out by APRI.

The mistaken diagnosis of child abuse: a form of medical abuse? Kirschner RH and Stein RJ. *Am J Dis Child* 1985; 139: 873. (These are ER resident cases from Cook County Hospital in the 1960's; today wd be considered obvious diagnoses.)

The shaken baby syndrome: a clinical, pathological, and biomechanical study. Duhaime A-C, Gennarelli TG, Thibault LE, Bruce DA, Margulies SS, and Wiser R. *Journal of Neurosurgery* 1987; 66: 409-414

Child abuse by drowning. Griest KJ and Zumwalt RE. *Pediatrics* 1989 Jan; 83(1): 41-46. See below under "Drowning."

Serial abuse in children who are shaken. Alexander R, Crabbe L, Sato Y, Smith W Bennett T, *Am J Dis Child* 1990; 144: 58-60. (risk of subsequent injury if not recognized)

Inflicted injury versus accidental injury. Charles Felsen Johnson. *Peds Clin N Amer* 1990; 37: 791-811. Includes burns & scalds.

Inflicted skeletal injury: a postmortem radiologic-histopathologic study in 31 infants. Kleinman PK, Marks SC jr, Richmond JM, Blackbourne BD. (excluded skull fxx) *AJR* 1995; 165: 647-650 (have)

Child abuse and neglect. (review article). Wissow LS. *N E J Med* 1995; 332(21): 1425-1431

Current concepts: Nonaccidental head injury in infants -- the "shaken-baby syndrome." Duhaime A-C, Christian CW, Rorke LB, and Zimmerman RA. *N E J Med* 1998 Jun 18; 338(25): 1822-1829. Cf Editorial, *Lancet* 1998 Aug 1; 352: 335.

Introduction to Paul Kleinman's *Diagnostic Imaging of Child Abuse, 2d ed.* (1998). Gives a short summary of the history of the field and the seminal articles.

Abusive head trauma? A biomechanics-based approach. Hymel KP, Bandak FA, Partington MD and Winston KR. *Child Maltreatment* 1998 May; 3(2): 116-127

Retinal haemorrhages in child abuse. Levin AV. In: T J David, ed., *Recent Advances in Paediatrics 18*. London: Churchill Livingstone, 2000. ISBN 044306430X

Child Maltreatment, Third Edition. Angelo P. Giardino and Randell Alexander. ISBN 1-878060-54-6. Vol. 1: Clinical Guide. Vol 2: Photographic Reference. Saint Louis: G.W. Medical Publishing, Inc., 2005. <http://www.gwmedical.com>

DDX -- DIFFERENTIAL DIAGNOSIS

Role of the Medical Examiner in Fatal Cases. Michael Graham, MD and Tracey Corey, MD. In: Giardino AP and Alexander R, *Child Maltreatment: A Clinical Guide and Reference, 3d ed.* St. Louis: G.W. Medical Publishing, Inc., 2005. Includes a discussion of "Conditions that are not abuse." Includes: trivial scrapes and bruises, birth injuries, SIDS, mongolian spot, OI, neoplasm, Caffey's disease, congenital syph, kinky hair syndrome, coagulopathies, metabolic disorders, connective tissue disease, hematologic disease (particular as regards AHT), hemophagocytic lymphohistiocytosis, leukemia, and injuries related to therapeutic intervention. Under this, shows neck conditions from CPR and catheterization injury of the introitus. But notes that "CPR-related facial bruises are distinctly uncommon." (citing DiMaio's *Forensic Pathology*.) Discusses the issue of iatrogenic rib fractures: "In contrast to adults, rib fractures caused by CPR are very rare in children without a predisposing bone disease. (citing Ken Feldman et al., 1984). CPR-related rib fractures are anterior or anterolateral and are not posterior. Visceral injuries such as hepatic

laceration can occur during resuscitation...” Also mentions folk medicine such as *cao giao* (coining), dermabrasion, cupping, plumbism, moxibustion, and *caida de mollera*. Also discusses postmortem insect bites.

See Table 2 from Nimkin & Kleinman, Radiol Clin N Amer 2001 Jul, which Lists the following conditions for bony lesions:

- Congenital indifference to pain
- Myelodysplasia
- Osteomyelitis
- Congenital syph
- Rickets
- Scurvy
- Vitamin A intox
- Caffey’s disease
- Leukemia
- Prostaglandin E-1 therapy
- Methotrexate
- Menkes’
- Copper deficiency
- Metaphyseal and spondylometaphyseal dysplasias
- Erythema multiforme
- Hemophagocytic lymphohistiocytosis
- Berloque dermatitis
- hypogammaglobulinemia (vs. neglect)
- cystic fibrosis (vs. neglect)
- panhypopituitarism (vs. neglect)
- malabsorption (vs. neglect)
- pyloric stenosis (vs. neglect)
- glycogen storage diseases (vs. neglect)
- chronic tonsillitis (vs. neglect)
- chromosomal disorder (vs. neglect)

Birth trauma

Cephalhematoma, Apnea of prematurity, clavicle and long bone fxx, rib fxx, scleral hems, Rickets of prematurity, HDN, benign subdural collections of infancy, vacuum extraction

Nutritional disorders

Rickets incl neonatal rickets or rickets of prematurity/TPN, scurvy, copper deficiency, FTT, malabs, CF, pyloric stenosis, malabsorption

Metabolic diseases

OI, Ehlers, Menkes, Storage diseases, Cockayne's, Alagille's, Biotinidase deficiency, GA, CF with malabs/ bruiseability, glutaric aciduria, panhypopituitarism, glycogenopathy, congenital disorder of glycosylation

Infections

TORCH, impetigo, tinea, STSS, Bruton's etc., meningitis, pox, Bruton's, Behcet's

Autoimmune diseases

Idiopathic pulmonary hemosiderosis (see SIDS-Suffocation), Pulmonary vasculitis (part of systemic vasculitis or Goodp)

Neuromuscular disorders

Congenital insensitivity to pain, spina bifida, cerebral palsy, fractures caused by physical therapy, MD, X-linked congenital myotubular myopathy

Pulmonary diseases

Heiner's syndrome (cow's milk allergy causing bleeding into the lungs and GI tract)

Skeletal disorders

OI, osteodysplasias, osteomyelitis, Caffey's disease (Infantile Cortical Hyperostosis), pseudofractures, fragility of extreme prematurity, rickets due to extreme prematurity, nutritional osteopenia, hypervitaminosis A, syph, rickets due to phenytoin or phenobarbital, scurvy (Barlow's disease)

Hematologic
 Any coagulopathy, Henoch, hemoph, VonW (VWD), ITP, Leukemia, Tumor, HDN, Petechiae caused by violent coughing, histiocytosis X, purpura fulminans, SSA/ hand-foot syndrome, salicylism, DIC, HUS, Wiskott-Aldrich?, Vitamin K deficiency, hemophagocytic lymphohistiocytosis, hypogammaglobulinemia

Dermatologic
 Mongolian spots, hypersensitivity angitis, erythema multiforme, Phytophoto, hemangioma, impetigo, tinea, epidermolysis bullosa, Eczema, erythema nodosum, erythema marginatum, Perianal Streptococcal cellulitis, urticaria pigmentosa, alopecia areata, Tricotillomania, loose anagen hair syndrome, Berloque dermatitis

Innocent trauma
 Sunburn, curling irons, chemical burns, seat belt burn, toddler's fx Nursemaid's elbow, hair-throat tourniquet syndrome, straddle Injury

Genital conditions
 Straddle injury, varicella, labial adhesions, perianal Strep, Anomaly, lichen sclerosus (more common, molluscum, allergic rxn, hemangioma, prolapse, caruncle, diaper rash, lichen planus, Crohn's disease, anal signs of HUS, Behcet's disease

Folk remedies
 Ca giao, cupping, caida de mollera, moharram, African facial scarification, female circumcision, moxib

Self-inflicted injury
 Self-mutilation

Medication effects
 Hypervitaminosis A, dilantin, warfarin

Neurological
 Ruptured AVM, sickle cell anemia

DDX -- specific diseases on which I have articles in hand.

Acute hemorrhagic edema of infancy
 Adenovirus (ocular petechiae)
 Alagille's syndrome
 ALL
 Beckwith's syndrome (pathologic fractures incl. rib fxx)
 Behcet's syndrome
 Berloque dermatitis
 Biotinidase deficiency
 Caffey's disease
 Coagulation disorders
 Congenital disorder of glycosylation
 Congenital melanocytic nevus
 Congenital X-linked myotubular myopathy
 Eczema
 Glutaric aciduria
 Glycogenopathy (vs. neglect)

Hemophagocytic lymphohistiocytosis
 Hemophilia
 Henoch
 HDN
 Hermansky-Pudlak syndrome
 Holocarboxylase synthetase deficiency (see biotinidase deficiency)
 Hypogammaglobulinemia (vs. neglect)
 ITP
 Idiopathic pulmonary hemosiderosis
 Incontinentia pigmenti
 Infantile cortical hyperostosis
 Langerhans cell histiocytosis
 Lichen sclerosus
 Malabsorption (vs. neglect)
 Menkes' syndrome
 Neonatal rickets
 OI
 Panhypopituitarism
 Phytophotodermatitis
 Pyloric stenosis (vs. neglect)
 Scurvy
 Streptococcal TSS
 Trichotillomania
 Valsalva's hemorrhagic retinopathy

GENERAL ARTICLES ON DIFFERENTIAL DIAGNOSIS

The cutaneous manifestations of child abuse and neglect. Ellerstein N. Am J Dis Child 1979; 133:906-909

The battered child syndrome: pitfalls in radiological diagnosis. Radkowski MA. Pediatric Annals 1983; 12: 894-903

The mistaken diagnosis of child abuse: a form of medical abuse?. Kirschner RH and Stein RJ. AJDC 1985; 139: 873-875

Pseudoabuse: the misdiagnosis of child abuse. Kaplan JM. JFS 1986; 31: 1420-1428

The conflict of underdetection and overreporting. Silverman FN. Pediatrics 1987; 80: 441-443

Mistakes in diagnosing non-accidental injury: 10 years' experience. Wheeler DM, Hobbs CJ. BMJ 1988; 296: 1233. Includes a case of Valsalva's hemorrhagic retinopathy.

Brittle or battered? Carty H. Arch Dis Child 1988; 63: 350-352

Mistakes in diagnosing non-accidental injury: ten years' experience. Wheeler DM and Hobbs CJ. Br Med J 1988; 292: 1233-1236

Advances and retreats in the protection of children. Krugman RD. NE J Med 1989; 320: 531-532

Is it sexual abuse? Adams JA and Horton M. Clin Pediatr 1989; 28: 146-148

The mistaken diagnosis of child abuse: a three-year USAF Medical Center analysis and literature review. Wardinsky T and Vizcarrando F. Mil Med 1995; 160: 15-20. Gives the figure that 7% of children with

physical signs suggestive of abuse actually have some other medical condition. Dr Reece considers this figure suspect. See his review of a Menkes case, under “DDX -- Specific Conditions, below.”

Conditions mistaken for child abuse: part I. Stewart GM, Rosenberg NM. *Pediatric Emergency Care* 1996; 12: 116-121

Jean Bays, Conditions mistaken for child physical abuse. In: Reece & Ludwig, 2d edition (2001)

When inflicted skin injuries constitute child abuse. *Pediatrics* 2002; 110(3): 644-645. If (a) they are nonaccidental, and (b) there is any injury beyond temporary reddening of the skin. (In other words, all nonaccidental deep bruises are child abuse. –JKR) See below under “Bruises.” This policy statement was RETIRED by the Academy in October 2006. See announcement at page 405 of the February 2007 issue of *Pediatrics*.

Child Abuse: Radiologic-Pathologic Correlation. From the Archives of the AFIP, pp 811-845. *RadioGraphics* 2003; 23: 811-845. Gael J. Lonergan, LTC USAF MC, Andrew M. Baker, MD, Mitchell K. Morey, MD, Steven c. Boos, LTC USAF MC. Covering bony and soft-tissue injuries.

Underdiagnosis of child abuse in emergency departments. Kunen S, Hume P, Perret JN, Mandry CV, Patterson TR. *Acad Emerg Med* 2003 May; 10(5): 546. It is likely that more than 75% of all child abuse cases presenting to EDs are being missed, and many of these missed cases involve fractures and dislocations in infants. In this chart review of 7,827 infants under two y o seen in ER, 127 had fractures or dislocations that were *not* diagnosed as abuse in the ER. We randomly selected 50 of these charts and rated them for likelihood of abuse: found that 30% of them were highly suspicious for abuse.

Child Abuse and Neglect: Guidelines for Identification, Assessment, and Case Management. Marilyn Strachan Peterson and Michael Durfee, eds. Volcano, California: Volcano Press, 2003. 370 pp. ISBN 1-884244-21-1. Updates: <http://www.volcanopress.com/>.

Child abuse and the otolaryngologist, parts I and II. Crouse CD, Faust RA. *Otolaryngol Head Neck Surg* 2003 Mar; 128(3): 305-310, 311-317. Discusses, for otolaryngologists, conditions of the head & neck that may be mistaken for abuse.

Children with signs of abuse: when is it not child abuse? Laposata ME and Laposata M. *Am J Clin P* 2005 Jun; 123 Suppl: S119-S124. Child with bruises turned out to have a coagulopathy. Vasculopathy/collagen disease is also in the differential. See also Barlow's disease and scurvy, below under “Specific disease entities.” “Overdiagnosis is as catastrophic as underdiagnosis.”

SPECIFIC DISEASE ENTITIES

Infantile cortical hyperostosis. Caffey J. *J Ped* 1946; 29: 541-559 (see Classic Articles)
In the 1962 article, "The battered-child syndrome," (above), Kempe et al. include this entity (Caffey's disease) in the differential diagnosis of the radiographic picture of battered child syndrome. They state that it is differentiated by two features: (A) metaphyseal lesions do not occur in ICH. (B) mandibular hyperostosis does not occur in BCS.

Congenital syphilis mimicking battered child syndrome. Fisher RH, Kaplan J, Holder JC. *Clin Pediatr* 1972; 11: 305-307

Kinky hair syndrome: serial study of radiologic findings with emphasis on the similarity to the battered child syndrome. Adams PC, Strand RD, Bregman MJ et al. *Radiology* 1974; 112: 401-407

Subdural hematoma following open-heart operations. Krous HF, Tenckhoff L, Gould NS, Stamm SJ. Ann Thoracic Surg 1975; 19(3): 269-276. Because of anticoagulation. Lori Frazier suggests that that would probably not happen today. [Yes, that would be Tenckhoff of the Tenckhoff catheter and Stamm of the Stamm gastrostomy.]

Misdiagnosis of neglect in a child with bleeding disorder and cystic fibrosis. Carpentier U, Gustafson LP, and Haggard ME. Southern Med J 1978; 71: 854-855

Vitamin D deficiency rickets simulating child abuse. J Pediatr Orthop 1981;

Hypersensitivity vasculitis presenting as suspected child abuse: case report and literature review.

Waskerwitz S, Christoffel KK, Hauger S. Pediatrics 1981; 67: 283. Looked like bruises. Required a skin biopsy to differentiate. See Collins, letter, 1981, below under "Bruises," saying why don't you just keep this zebra to yourself, it causes problems in court. But Waskerwitz et al. reply that this is not as rare as you think. Also, we are by no means contending that biopsies should be done in every case. In this case, we only did one because we observed evanescent urticaria, which suggested something else was going on, other than abuse.

Rickets presenting as multiple fractures in premature infants on hyperalimentation. Geffer WB, Epstein DM, Anday EK et al. Pediatric Radiology 1982; 142: 371

Von Willebrand's with ICH: Aust NZ J Surg 1982; 521: 86; Surg Neurol 1986; 26: 457

Erythema multiforme: confusion with child battering syndrome. Roddy et al. Pediatrics 1983; 72: 718.

Pseudoabusive burns in asian refugees. Feldman KW. AJDC 1984; 138: 768-769

Bleeding disorders and non-accidental injury. O'Hare AE, Eden OB. Arch Dis Child 1984; 59: 860-864

Ehlers-Danlos syndrome type IV mimicking non-accidental injury in a child. Br J Derm 1984; III: 341-345

Henoch-Schoenlein Purpura misdiagnosed as suspected child abuse: a case report and literature review. Brown J and Melinkovich P. JAMA 1986; 256(5): 617-618

A case of panhypogammaglobulinemia masquerading as child abuse. Copeland AR. JFS 1988; 33(6): 1493-1496 Looked like starvation neglect.

Brittle or battered? Carty H. Arch Dis Chil 1988; 63: 350-352

Copper deficiency and non-accidental injury. Shaw JC. Arch Dis Chil 1988; 63: 448-455

Clinical presentations of Ehlers-Danlos syndrome type IV. Arch Dis Chil 1988; 63: 1016-1025

Undiagnosed, untreated acute lymphoblastic leukemia presenting as suspected child abuse. McClain JL, Clark MA, Sandusky GE. JFS 1990 Jul; 35(3): 735-739.

Criteria for diagnosis of Behcet's disease. International Study Group for Behcet's Disease. Lancet 1990; 335: 1078-1080. "The clinical triad of uveitis with oral and genital ulceration was probably first recognized by Hippocrates, but bears Behcet's name..." Variable inv of many organ systems. Five sets of diagnostic criteria are in current use. This study group was appointed in 1985 to compare the different sets as to diagnostic performance. Results: the only diagnostic features that showed useful discriminating value were:

Genital ulcers

Eye lesions
Positive pathergy test
Folliculitis
Erythema nodosum

So a new set of diagnostic criteria, to be known as the International Study Group (ISG) criteria was put forth:

Oral ulcers (recurring at least 3 times in any 12-month period) +

Two of the four features:

Genital ulcers
Eye lesions
Positive pathergy test (read by physician at 24-48h)
Either folliculitis or erythema nodosum

Case report: multiple neonatal fractures -- dietary or deliberate? Duncan AA and Chandy J. Clin Radiol 1993; 48: 137-139

Patterns of facial resuscitation injury in infancy. Kaplan JA and Fossum R. Am J Forensic Med Path 1994; 15(3): 187-191 (see CPR)

Acute hemorrhagic edema of infancy. (case report). Caliskan S, Tasdan Y, Kasapcopur O et al. Arch Pediatr Adol Med 1995 Nov; 149: 1267-1268

Fatal intramuscular bleeding misdiagnosed as suspected nonaccidental injury. Wetzel RC, Slater AJ, Dover GJ. Pediatrics 1995; 95(5): 771-773. See below under RH -- other. A supposed case of late-onset HDN with severe RH and interhemispheric SDH, fatal in a 10 week old homebirth breastfed f who recd no VK, presented earlier with unexplained bruising, recently with rectal bleeding, now adm decerebrate with SDH, hgb 3.0, no head trauma, markedly elev PTs, very low factor VII. Has multiple large RH (photo).

Cutaneous necrosis after contact with calcium chloride: a mistaken diagnosis of child abuse. Zurbuchen P, Lecoulture C, Calza A-M, Halperin D. Pediatrics 1996 Feb; 97(2): 257-258. This child got in contact with an industrial defrosting agent which caused deep dermal necrosis of the thigh.

Two cases of incontinentia pigmenti simulating child abuse. Ciarello L and Paller AS. Pediatrics 1997 Oct; 100(4):e6. Figure 1 shows linear streaks of pigmentation (brown) and tiny erythematous vesicles following Blaschko's lines on the arm. In figure 2 the back and buttocks have purple stripes composed of pigment, erythema, and lines of vesicles. Could be mistaken for burns or abrasions. Not only that, but it has CNS lesions -- a hemorrhagic white matter encephalitis -- and also cotton-wool spots and RH, which can lead to blindness. Also have strabismus and glaucoma. This X-linked dominant condition is lethal in boys and affects girls variably.

Phytophotodermatitis mimicking child abuse. Hill PF, Pickford M, Parkhouse N. J R Soc Med 1997 Oct; 90(10): 560-561 A blistering burn to the buttocks in a 3 y o girl. Caused by the herb called "rue" growing in the child's wading pool. This produces a contact dermatitis which is photosensitive. Here are some herbs which can cause this: celery, parsnip, parsley, various garden weeds.

Langerhans cell histiocytosis mimicking child abuse. Papa CA, Pride HB, Tyler WB, Turkewitz D. J AM Acad Derm 1997 Dec; 37(6): 1002-1004 deep anal fissures.

Disorders of coagulation misdiagnosed as nonaccidental bruising. Jim R Harley. Pediatr Emerg Care 1997; 13: 347-349 Two cases of ITP and one case of hemophilia misdiagnosed as child abuse.

Two cases of incontinentia pigmenti simulating child abuse. Ciarello L and Paller AS. *Pediatrics* 1997 Oct; 100(4):e6. Figure 1 shows linear streaks of pigmentation (brown) and tiny erythematous vesicles following Blaschko's lines on the arm. In figure 2 the back and buttocks have purple stripes composed of pigment, erythema, and lines of vesicles. Could be mistaken for burns or abrasions. Not only that, but it has CNS lesions -- a hemorrhagic white matter encephalitis -- and also cotton-wool spots and RH, which can lead to blindness. Also have strabismus and glaucoma. This X-linked dominant condition is lethal in boys and affects girls variably.

Biotinidase deficiency: two cases of very early presentation. Haagerup A, Andersen JB, Blichfeldt S, et al. *Dev Med Child Neurol (GB)* 1997 Dec; 39(12): 832-835. For a discussion of biotinidase deficiency (= multiple carboxylase deficiency), see page 167 in Stocker & Dehner's *Pediatric Pathology* (1992), where it is stated that biotinidase removes biotin for recycling. (See below). They state that the symptoms mimic biotin deficiency (*q.v.*). "Patients are acidotic and ketotic; they develop hyperammonemia, hypoglycemia, and hyperglycinemia, have seizures, and may become comatose. A tomcat odor and an erythematous rash occur. ..." *Nelson's Pediatrics*, 15th Ed. explains this further: biotin, a cofactor for all carboxylases, is bound to the carboxylases. From them it must be released for recycling. If not, the result is biotin deficiency leading to organic acidemia. The incidence of biotinidase deficiency is 1:60,000 (autosomal recessive). (A related disorder is holocarboxylate synthetase deficiency, which prevents binding of biotin to its carboxylases. This one strikes in the first few weeks of life with respiratory difficulty or apnea, hypotonia, sz, vom, FTT.)

Syphilis or abuse: making the diagnosis and understanding the implications. Connors JM, Schubert C, Shapiro R. *Pediatr Emerg Care* 1998; 14: 139-142. Syphilis is the great mimic. In one case, a six year old male, he had perianal condyloma lata, flat painless coalescent grayish-white papules that were caused by sexual abuse. These were mistaken for HPV warts. The other case was a six month old infant with fever vomiting irritability and decreased movement of the right upper extremity with swelling of the right elbow. There was metaphyseal erosion of the distal humerus consistent with a healing fracture, but also bilateral tibial changes of Syph. There was a high risk for congenital infection because the mother had a history of gonorrhea and chlamydia during the pregnancy, although she had been RPR negative, as had the cord blood. But retesting disclosed RPR+ baby and FTA +++. The author says ruling out Syph is not straightforward, may require repeated testing. See also under "Fractures," below.

Major hemorrhage in children with ITP: immediate response to therapy and long-term outcome. Medeiros D and Buchanan GR. *J Pediatr* 1998; 133: 334-339. Only 2 of 68 major hemorrhages were intracranial. Dr. Reece comments that the markedly depressed platelet count is diagnostic and should avoid confusion. See Lozance et al., Role of coagulopathy in patients with head trauma, *J Clin Neuroscience* 1998; 5: 394; Also *Pediatrics* 1997; 99: 371.

Streptococcal toxic shock syndrome presenting as suspected child abuse. Nields H, Kessler SC, Boisot S, and Evans R. *Am J Forens Med Path* 1998; 19: 93-97. Red sores transiently on her back that could have been mistaken for cigarette burns. Dr. Kirschner comments that child abuse was really never in the differential for this child; she died from lack of early medical care.

Streptococcal toxic shock syndrome presenting as suspected child abuse. Nields H, Kessler SC, Boisot S, and Evans R. *Am J Forensic Med Path* 1998; 19(1): 93-97

Urticaria pigmentosa mistaken for child abuse. (letter). Gordon EM, Bernat JR jr, Ramos-Caro FA. *Pediatric Dermatology* 1998 Nov-Dec; 15(6): 484-485

Clinical features of pediatric Behcet's disease. Eldem B, Onur C and Ozen S (Ankara). Disease is a constellation of lesions including oral ulcers, genital ulcers, erythema nodosum, arthritis, eye involvement, and, rarely, CNS involvement with dural sinus or cavernous sinus thrombosis, pseudotumor cerebri. Report twenty patients. 65% had genital ulcers. Lori Frasier comments that these genital ulcers can mimic herpes lesions or trauma, and that Behcet's should be considered in the DDX of sexual abuse. See also slide 48 of the AAP slide set, "Visual Diagnosis of Child Sexual Abuse" prepared by Carol Jenny and

Deborah Lowen, (1998). This slide shows redness and edema of the inner genital area with some raised umbilicate wart-like ulcers each about 0.5 cm..

Delayed-onset profound biotinidase deficiency. Wolf B, Pomponio RJ, Norrgard KJ et al. J Pediatr 1998 Feb; 132(2): 362-365.

Henoch-Schoenlein purpura in a child at risk of abuse. Daly KC, Siegel RM. Arch Pediatr Adol Med 1998 Jan; 152(1): 96-98. Case report of a three year old girl. Purpuric lesions (ecchymoses) of the buttocks and legs and a limp with R knee pain. Mother IVDA, brother had SBS, father ETOH. Some NVD. Swel near R eye w rash. New lesions appeared in the hospital. Dx of HSP confirmed by skin bx.

Spontaneous subdural hematoma in severe hemophilia A. Abstract: [Spontaneous subdural hematoma in an 18 day-old male newborn with severe hemophilia A] Ries M, Klinge J, Rauch R, Chen C, Deeg KH, Klinik mit Poliklinik fur Kinder und Jugendliche, Universitat Erlangen-Nurnberg. Klin Padiatr 1998 May-Jun; 210(3): 120-124. Abstract: "Most intracranial bleedings in hemophiliacs occur in patients more than 6 months of age. In the neonatal period, this complication is rare and almost always observed in the first week of life. Based on a review of the literature, intracranial hemorrhage is an exceedingly rare occurrence in infants with hemophilia aged 2 weeks to 6 months. We report on a male infant with hemophilia A who was referred to our hospital on day 18 because of pallor and jaundice. The neurological examination was normal. A cerebral ultrasound showed a left-sided subdural hematoma with a shift of the midline structures to the right. PRBC's and factor VII were given and a craniotomy with evacuation of the hematoma was performed. The postoperative course was uneventful. On discharge, the neurological examination was considered normal."

Thalidomide responsiveness in an infant with Behcet's syndrome. Lee BW et al. Pediatrics 1999; 103: 1295-1297. Classical triad of oral and genital ulceration with recurrent uveitis, first reported by Behcet in 1937. 28% have vasculitis.

Late-form hemorrhagic disease of the newborn: a fatal case report with illustration of investigations that may assist in avoiding the mistaken diagnosis of child abuse. Ruttly GN, Smith CN, Malia RG. Am J Forens Med Path 1999 Mar; 20(1): 48-51. 9m male FTT term NSVD bottle fed got VKx2, no bruising, recent ten days of diarrh & vom, pres comatose w large interh SDH and severe RH. No head trauma. Elevated PT PTT with normal thrombin time and fibrinogen. They did factor testing on antemortem plasma and ruled in PIVKA (protein induced by vitamin K absence or antagonism). Dr. Kirschner terms this an excellent report. (Child Abuse Quarterly, Jan. 2000; vii: 16.) Cf Dr Brousseau et al's 2005 report, below.

When "NAI" means "not actually injured." Mayes C and Macleod C. BMJ 1999; 318: 1127-1128. Symmetrically located congenital melanocytic nevi on the temples looked like bruising unexplained by the history in a 3 month old male.

Medical complications in long-term survivors with X-linked myotubular myopathy. Herman GE, Finegold M, Zhao W, de Gouyon B, Metzenberg A. J Pediatr 1999 Feb; 134: 206-214. This disease causes peliosis hepatis, which can cause fatal subcapsular hematoma of the liver with hemoperitoneum due to trivial trauma.

Treatment of pyruvate carboxylase deficiency. Ahmad A et al. Am J Med Genet 1999; 87: 331-338. Case report of an 11 wk infant w fever, lethargy, limpness, myoclonic, and old R frontal SDH. State that cd be confused with SBS.

Congenital skull fracture as a presentation of Menkes disease. Ubhi T, Reece A, Craig A (Leeds). Dev Med Child Neurol 2000 May; 42(5): 347-348. Case report of a congenital skull fx and intracerebral hemorrhage, seizures, diagnosed at 3 months of age. Progressing to death at 3 years. Menkes requires treatment with copper-histidine early in life to avoid this. Congenital skull fracture has never before been reported in this disease. (have abstract). For retinal hemorrhages in Menkes' disease, see Levin AV, Retinal haemorrhages and child abuse. In: David TJ, ed., Recent advances in Paediatrics 18. Edinburgh:

Churchill Livingstone 2000; 199-200, where he says that one patient had peripheral RH with peripheral subhyaloid hemorrhage, “a very unusual [anatomic] distribution not commonly seen even in SBS.” Probably due to a vascular abn.

Parietal pseudofracture and spontaneous intracranial hemorrhage suggesting nonaccidental trauma: report of two cases. Fenton LZ, Sirotak AP, Handler MH. *Pediatr Neuros* 2000; 33: 318-322, reviewed in *Child Abuse Quarterly*, July 2001. **Parietal fissures** are sinuous fibrous defects in the calvaria that can be mistaken for healing fractures. I recall Dr. Golden encountered one of these in a child in 1999 or 2000. In this case report, one 35 day old breast-fed infant bled intracranially with ICH and a R interhemispheric SDH due to HDN, and was found to have a parietal fissure. Second case had ICH due to congenital herpes simplex encephalitis and had a parietal fissure. In these cases there were no RH, but there were massive SDH which these guys were able to explain away on grounds of natural disease.

Fracture rates in patients with growth hormone deficiency. Wuster C. *Hormone Research* 2000; 54 Suppl 1: 31-35. Panhypopituitarism causes obesity, diabetes, osteopenia, and pathologic fractures. See also Stocker & Dehner, 2001, at p. 1019.

A fatal case of infantile scurvy. Mimasaka S, Funayama M, Adachi N, nata M, Morita M. *Int J Legal Med* 2000; 114(1-2): 122-124. We initially suspected that the bleeding was due to violence. But it was due to neglect. The parents locked the child alone in a room while they went out during the day.

Laxative-induced dermatitis of the buttocks incorrectly suspected to be abusive burns. Leventhal JM, Griffin D, Duncan KO, Starling S, Christian CW, Kutz T. *Pediatrics* 2001; 107: 178-180. Four children with large buttock burns. They had taken Ex-Lax and had primary irritant contact dermatitis. Dr. Ricci, reviewing this in *Child Abuse Quarterly*, July 2001, says they are not sure whether Ex-Lax contains a chemical irritant or if it is digestive enzymes in the stool. Notes that the parents may not know about the Ex-Lax ingestion if the child gets into it surreptitiously. Jan Bays and Ken Feldman in *Reece 2d ed.*, p. 418 (Child abuse by poisoning), state that Ex-Lax has been reformulated to contain sennasides (senna), which are related to the anthracene dyes, a black dye, and looks and tastes like chocolate candy. Stools contain increased sodium and chloride. Ken Feldman comments that in one case where a child ate twelve squares, there were burns on the lips.

Hermansky-Pudlak syndrome presenting with subdural haematoma and retinal haemorrhages in infancy. Ressell-Eggitt IM, Thompson Da, Khair K et al. *J Roy Soc Med* 2001; 93: 591-592.

Idiopathic pulmonary hemosiderosis. See Chapter 349 of *Nelson's Textbook of Pediatrics* (15th ed.), and page 546 of *Stocker & Dehner's Pediatric Pathology*. Stocker says, “Hemosiderin in the lung usually indicates previous hemorrhage or aspiration of blood and is thus relatively nonspecific (referring to a table of differentials). Macrophages containing hemosiderin can be found in alveolar or interstitial regions in association with conditions such as infection, blood dyscrasia, chronic heart failure, pulmonary hypertension, and neoplasia.” He then goes on to discuss IPH as “a group of rare disorders,” usually affecting children 1 to 6 years of age but can be seen as early as 4 months, nearly always associated with anemia and sometimes with eosinophilia. Nelson's says four types of PH: an idiopathic form, a cow's milk allergy form, a myocarditis form, and a Goodpasture's form. (Only the idiopathic form is called “idiopathic PH.” As far as the idiopathic form, “Onset usually occurs in childhood, rarely later than early adult life.” “Symptoms are those of recurrent or chronic pulmonary disease and include cough, hemoptysis, dyspnea, wheezing, and occasional cyanosis associated with fatigue and pallor.”

Subconjunctival haemorrhage and respiratory distress. Chiu C-H et al. *Lancet* 2001 Sept 1; 358: 724. Thought to be from adenovirus infection.

Pertussis and coughing as causing facial petechiae. Byard RW and Krous HF. *Sudden Infant Death Syndrome: Problems, Progress & Possibilities*. New York: OUP, 2001, p. 230.

Rectorrhagia and lichen sclerosus in childhood. (letter) Labandeira J et al. Pediatric Dermatology 2001; 18: 543-545. Case report of an eight year old girl with rectal bleeding.

Pediatric ribs: a spectrum of abnormalities. Glass RBF, Norton KI, Mitre SA, Kang E. Radiographics 2002; 22: 87-104. Complete review of the radiology and abnormalities of the ribs.

Berloque dermatitis mimicking child abuse. Gruson LM, Chang MW. Arch Pediatr Adol Med 2002 Nov; 156(11): 1091-1093. Berloque dermatitis is a photocontact dermatitis reaction to perfumed skin products containing bergamot or a psoralen reacting with sunlight. It is linear in pattern, corresponding to the areas of application. In the acute phase it has erythema and even blistering; in the subacute phase it has hyperpigmentation.

Periosteal reaction with normal-appearing underlying bone: a child abuse mimicker. Ved N, Haller JT. Emergency Radiology 2002 Nov; 9(5): 278-282. From the abstract: Any irritation or disruption to the underlying bone will cause a periosteal reaction and result in new periosteal bone deposition. Hypervitaminosis A, prostaglandin therapy, cortical hyperostosis (Caffey's disease), hypertrophic osteoarthropathy, osteomyelitis, leukemia, trauma, syphilis -- the last four involve bone destruction the others do not.

Eczema mimicking child abuse: a case of mistaken identity. Heider TR, Priolo D, Hultman CS, Peck MD, Cairns BA. J Burn Care Rehab 2002 Sep-Oct; 23(5): 357-359. Looked like an abusive burn. Advocates multidisciplinary eval before child abuse is diagnosed.

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. This disease seems to be reemerging when "Infants with borderline vitamin C depletion are assaulted with too many inoculations at one time." This guy is a professor of medicine at Tulane. Vitamin C deficiency also causes increased blood and tissue levels of histamine, because of impaired catabolism of histamine, for which ascorbic acid is a needed cofactor. This in turn causes capillary fragility through separation of the endothelial cells, something that histamine does as an acute-phase reactant. Obviously, infection increases histamine. So does vaccination. Vitamin C deficiency can be caused in bottle-fed infants due to boiling or heating of formula and a lack of orange juice or vitamin supplements in the diet. [The good professor does not seem to realize that orange juice will not go through a baby nipple. -JKR] "Subdural hemorrhages, multiple bone fractures, and sub-periosteal hemorrhages do occur in infantile scurvy, (citing Barlow's original work published in 1883 and Hart, Lessing, book in German about scorbutism, 1913), but all too often now the sub-periosteal hemorrhages lifting the growing sheath right off the bone, are thought to be the result of fractures, instead of being recognized as revealing their cause. Even this X-ray finding, formerly known as being characteristic of the healing phase of infantile scurvy, is now often said to be indicative of child abuse." Goes on to discuss the Florida court conviction of one Alan Yurko, convicted of shaken baby syndrome. Case report: premature infant male 10 weeks of age. Pregnancy complicated by maternal illness (?hyperemesis) resulting in significant maternal weight loss and failure to take vitamins. Infant received multiple inoculations at 8 weeks of age, eleven days later developed a high-pitched cry, then stopped breathing while being cared for by father. Had delayed rescue, which the present author explains by saying that the parents had been warned at the time of vaccination that such symptoms might occur and so ignored them. Had severe anemia, bruises on head and face, acute right-sided SDH, right-sided RH, fever, healing costochondral junction fractures and a fresh 10th rib fracture. Expired. Problem: mere "borderline" vitamin C deficiency (which was not documented in this infant) would not be expected to cause bone fragility or fractures; only frank scurvy would do that. The author handles this difficulty as follows: "The escape of blood into the tissues soon causes hemolysis and hemolysis rapidly destroys ascorbic acid (citing his own book on vitamin C), leading to frank scurvy." See also next below.

Cerebral hemorrhage in Henoch-Schoenlein syndrome. Chiaretti A et al. Child's Nervous System 2002; 18: 365-367. Vasculitis rarely causing neuro sequelae, but this time a girl presented with abdominal pain and purpura of the legs and buttocks with hematuria. Followed by spontaneous intracerebral hemorrhage. Made a complete recovery on steroids.

Hemophagocytic lymphohistiocytosis masquerading as child abuse: presentation of three cases and review of central nervous system findings in hemophagocytic lymphohistiocytosis. Rooms L, Fitzgerald N, McClain KL. Pediatrics 2003 May; 111(5 Pt 1): e636-640. Disease is characterized by fever, hepatosplenomegaly, lymphadenopathy, CNS dysfunction. This art collects three cases. Two had SDH + RH. Other had periosteal NBF in two long bones. The disease causes elevation of the PTT and a hemorrhagic diathesis.

Fractures in biliary atresia misinterpreted as child abuse. DeRusso PA, Spevak MR, Schwarz KB. Pediatrics 2003 Jul; 112(1 Pt 1): 185-188. Biliary atresia is associated with metabolic bone disease. There are numerous reports of rickets, osteopenia, and fractures.

Purpuric gloves-and-socks syndrome caused by parvovirus B 19 infection. Messina MA, Ruggeri C, Rosano M. Pediatric Infectious Disease Journal 2003; 22: 755-756.

Intraretinal hemorrhages and chronic 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.

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. In this further development of the hypothesis offered in Barlow's disease (above), the author notes that Caffey, in his original work on SBS, considered scurvy in the differential diagnosis, as did Harry Kempe. He also brings up Fung et al., Unexplained subdural hematoma in young children: is it always child abuse? (2002), who criticize the child abuse diagnosis of isolated subdurals with RH as "a self-fulfilling prophecy." [But note Randy Alexander's criticism of that characterization in his *Quarterly* critique of Fung's article, as to which see below under "Shaken." Randy points out that it's not just the subdural that is unexplained -- it's the brain injury.] How would scurvy cause brain injury? In any event, Prof. Clemetson discusses how vitamin C deficiency causes increased histamine levels, which in turn cause hemorrhage and fragility. Cites Lund and Kimble, 1943, for the proposition that hyperemesis gravidarum can cause dangerously low levels of vitamin C and can also cause retinal hemorrhages in the gravida (!) See below under "RH -- Unusual causes."

Childhood panhypopituitarism presenting as child abuse: a case report and review of the literature. Marcus BJ, Collins KA. Am J Forens Med P 2004 Sep; 25(3): 265-299. Diabetes, growth failure, decreased bone density, obesity. Case of a 3 year old black girl who died in the ER with a healing fracture of the surgical neck of the left femur and severe growth retardation. At autopsy, all the endocrine glands were hypoplastic. Status post group B streptococcal meningitis at age six weeks.

Child abuse and trichotillomania. Saraswat A. (Lucknow) BMJ 2005 Jan; 330(7482): 83-84.

Blinding keratoconjunctivitis and child abuse. Ong T, Hodgkins P, Marsh C, Taylor D. Am J Ophth 2005 Jan; 139(1): 190-191. (Great Ormond Street) This is an inflicted injury, consistent with child abuse. Also called inferior half keratoconjunctivitis. Case report of two unrelated infants. One of them had an eye that had perforated. They recovered quickly in the hospital with no specific treatment. Child abuse workup showed that the infants had other signs of physical abuse, and in one family the sibling was found to suffer from severe abuse. Conclusion: Inflicted corneal injuries are nonspecific, and unexplained keratoconjunctivitis, especially in the lower half of the conjunctiva and cornea in infants should alert the cl to the possibility of child abuse, but by itself cannot be taken as pathognomonic. See also under "Dental and face."

Lichen sclerosus mistaken for child sexual abuse. Al-Khenaizan S, almuneef M, Kentab O. Int J Dermatol 2005 Apr; 44(4): 317-320. (King Fahd National Guard Hospital) Case report of a 3 year old girl with an asymptomatic white discoloration of the vulva present for three months, which after two more months developed itching and a yellow-green drainage and dysuria. The father was an alcoholic and drug addict. Abuse suspected. Examination showed sharply demarcated hypopigmented atrophic plaques symmetrically surrounding the labia majora with three sharply demarcated 0.5 cm reddish macules on the medial aspect

and two healing erosions of the introitus. "The hymenal ring could not be seen." (??) Culture for STD's was negative. Vaginal culture grew out beta hemolytic Strep.. A skin biopsy was diagnostic for LS. Showed atrophic dermatitis with follicular plugging, homogenized eosinophilic amorphous collagen replacing the papillary and upper reticular dermis. Treatment with mometasone furoate resolved all the symptoms except for mild hypopigmentation.

Vitamin K deficiency mimicking child abuse. Brousseau TJ, Kissoon N, McIntosh B. J Emerg Med 2005; 29: 283-288. As reviewed by Dr Hibbard in the Spring 2006 issue of the *Quarterly*, gives a case report of bruising and intracranial hemorrhage with the laboratory abnormalities found. Can present as early as the first 24 hours of life, or in its classic form on days 1 to 7 of life, or in its late-onset form at ages 2 weeks to 6 months. Maternal medications during pregnancy can exacerbate the normal VK deficiency of the newborn. So can placental conditions, since VK has trouble crossing the placenta. Breast milk does not provide. Cf Dr Ruddy's 1999 article, above.

Menkes disease mimicking non-accidental injury. Bacopoulou F, Henderson L, Philip SG. Arch Dis Child 2006;doi:10.1136/adc.2005.081836 (UK). Dr Reece summarizes this case report of a 3 mo male w status epilepticus, bilat SDH, cerebral and cerebellar atrophy, "metaphyseal spurs" at the distal ends of long bones, scanty colorless scalp hair, high-arched palate, pale lax skin, hypotonia in the Autumn 2006 *Quarterly*. He comments, as he has before (see above), that this diagnosis would be apparent to any pediatrician and does not, in fact, mimic child abuse.

Fracture rate in children with cerebral palsy. Stevenson RD, Conaway M, Barrington JW et al. Pediatric Rehabilitation 2006; 9: 396-403. Found a 45 fracture rate, correlated with obesity and gastrostomy and with history of a previous fracture.

Intracranial hemorrhage as the initial manifestation of a congenital disorder of glycosylation. Cohn RD, Eklund E, Bergner AL, Casella JF, Woods SL, Althaus J, Blakemore KJ, Fox HE, Hoover-fong JE, Hamosh A, Braverman NE, Freeze HH, Boyadjiev SA. Pediatrics 2006 Aug; 118(2): e514-e521. From the abstract: "Intracranial hemorrhage in a term neonate is a rare event in the absence of an identifiable precipitating factor such as severe thrombocytopenia, mechanical trauma, asphyxia, infections, or congenital vascular malformations. Congenital disorders of glycosylation are a genetically and clinically heterogeneous group of multisystem disorders characterized by the abnormal glycosylation of a number of glycoproteins. Although bleeding caused by abnormal glycosylation of various coagulation factors is a well-known clinical complication of several types of congenital disorders of glycosylation, intracranial hemorrhage has not been reported as an initial manifestation of this entity. Here we report the detailed history of a family with 2 consecutive male infants, both born at term with intracranial hemorrhage diagnosed within the first 24 hours of life. The diagnosis of a congenital disorder of glycosylation was established in the second infant by an abnormal glycosylation of serum transferrin detected by electrospray-ionization mass spectrometry. Both infants showed significant neurologic deterioration during the first month of life, and both died at 5 months of age. Intracranial hemorrhage in a term neonate without a potential precipitating factor represents yet another clinical feature that should raise the suspicion for a congenital disorder of glycosylation." Vince Palusci comments that these were sick infants (brothers) who were already deteriorating before they bled, and would not be confused with AHT infants.

METABOLIC DISEASE SCREENING

Newborn screening now covers by tandem mass spec approximately 100 diseases, of which a list is available at <http://www.dhs.ca.gov/gdb> The results for the Los Angeles area can be obtained from the hospital where the baby was born. The phone list as of late 2005 is available at <http://www.dhs.ca.gov/pcfh/gdb/html/NBS/NBSCoordinatorsPhoneList.htm> Here are the local numbers they give:

Kaiser Southern California
UCLA

626-564-3322
310-826-4458

Harbor-UCLA

310-222-3751

Pediatrix Screening P O Box 219 Bridgeville, PA 15017 Pediatrix_screening_information@pediatrix.com
or <http://www.pediatrixscreening.com>

California's experience implementing a pilot newborn supplemental screening program using tandem mass spectrometry. Feuchtbaum L, Lorey F, Faulkner L, Sherwin J, Currier R, Bhandal A, Cunningham G. Pediatrics 2006 May; 117(5): S261-S269. This was a pilot program of the California State Department of Health Services, run between Jan 2002 and Jun 2003. They offered tandem mass spec screening to just over half of California's 755,000 newborns during that year and a half. (In other words, they screened 353,894 newborns). They screened for amino acids and acylcarnitines to detect metabolic disorders classified as amino acid disorders, organic acid disorders, or fatty acid disorders. They identified 51 newborns with detectable disorders, or one for every 6,939 infants screened. The overall California population prevalence of detectable disorders (excluding PKU) was 1/6,500. The false-positive rate as determined by followup testing was .07%. They express concern about the fact that they left unscreened half the population, which certainly included some + cases, in fact, as per the coroners, two MCAD cases. MCAD usually doesn't have symptoms during the first year of life.

EPIDEMIOLOGY / RISK FACTORS

See also under SHAKEN and SEXUAL ABUSE and DRUG ABUSE and SIDS EPIDEMIOLOGY

Child maltreatment 1996: reports from the states to the National Child Abuse and Neglect Data System.
Available at [Http://www.acf.dhhs.gov/programs/cb/](http://www.acf.dhhs.gov/programs/cb/).

U.S. Department of Justice, Bureau of Justice Statistics.
<http://www.ojp.usdoj.gov/bjs/homicide/children.htm>

WISQARS Web Injury Statistics and Reporting System. National and state injury data.
<http://www.cdc.gov/ncipc/wisqars>

CDC WONDER cause of death data by county. <http://wonder.cdc.gov>

Expert Health Data Programming, Inc. California maps and epidemiologic data. <http://ehdp.com/>

California State Department of Health Services data site. <http://www.applications.dhs.ca.gov/>

FBI homicide for infants. <http://www.ojp.usdoj.gov/bjs/homicide/homtrnd.htm>

Decline in the number of homicides of children. "Increased over the past two decades, but has recently declined." As for infanticide, it has shown a modest decline since 1976. White was 2.4 per 100,000 in 1976 and in 1999 was 2.2 Black was 10.2 and 7.4. The source is the FBI's Uniform Crime Reporting Program (UCR.) This comes from law enforcement agencies and is based on (a) victim reports and (b) observation. (have)

An investigation of socio-demographic factors surrounding childhood drowning accidents. Nixon J and Pearn J. (Aust) Soc Sci Med 1978; 12: 387-390.

[Decrease in estimated incidence of physical abuse from 1975 to 1985]. Straus MA et al., Journal of Marriage and the Family 1986; 48: 480, reprinted in Straus MA and Gelles, *Physical Violence in American Families*, 1990. Decrease continuing to 1992 (Murray A. Straus, unpublished data). (Murray A. Straus, Professor of Sociology and Co-Director, Family Research Laboratory, University of New Hampshire, Durham NH 03824. See web site at unh.edu/frl. Dr. Straus believes there is a drop in actual prevalence as well as reporting. Reasons for the decrease wd include later marriage and fewer children.

He warns that there are some sectors of society that may have experienced an increase during the period that the overall pattern was a decrease.

Race and the shaken baby syndrome: experience at one hospital. Brenner SL, Fischer H, Mann-Gray S. JNMA 1989; 81(2): 183-184

Socioeconomic differentials and neonatal mortality: racial comparison of California singletons. Gould JB, Davey B, LeRoy S. Pediatrics 1989 Feb; 83(2): 181-186

Safety practices and living conditions of low-income urban families. Santer LJ and Stocking CB. Pediatrics 1991; 88: 1112-1118

Abusive head trauma: the relationship of perpetrators to their victims. Starling S, Holden JR, Jenny C. Pediatrics 1995 Feb; 95: 259-262. Retrospective chart review of 151 abusive head trauma children aged 3 weeks to 2 years. 35 were fatal, 116 nonfatal. 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.

In 127 cases, the perpetrator was identified:

	Male perp	Female perp
Fatal	23	8
Nonfatal	<u>64</u>	<u>32</u>
	68%	32%

Overall in the 127 identified cases, men were 2.2x more likely to be the assailant. Usually fathers and boyfriends.

Father	37%
Boyfriend	20%
Babysitter	17%
Mother	12%
Male BBS	4%
Stepf	3%

See also the mail survey of child fatality review teams conducted by the Utah Child Abuse Prevention Center, newsletter Autumn 1998, *infra*.

Dr Reece cites this article along with others in his critique of Leestma's 2005 article on confessed shaking publications for the proposition that since 2001 there have been more confessed shaking cases published than in all the years before 2001 (which were the material chosen by Leestma). See the *Quarterly* for end of 2005. Now in the present article, as to confessions, the article does not say how many confessions there were. The article gives zero detail about the confessions or the histories or the findings in the individual cases. This is an epidemiology article, not a clinical article.

Neonaticide, infanticide, and filicide: a review of the literature. Bull Am Acad Psych Law, vol 23, no. 3, 1995

Onset of physical abuse and neglect: psychiatric, substance abuse, and social risk factors from prospective community data. Chaffin M, Kelleher K and Hollenberg J. Child Abuse & Neglect 1996; 20(3): 191-203. (have). Social and demographic variables were found to be limited predictors of maltreatment, while substance abuse disorders were strongly associated with onset of both abuse and neglect. (relative risks = 2.90 and 3.24 respectively.) Depression was found to be a strong risk factor for physical abuse (relative risk = 3.45)

Preventing family violence. Browne KD, Herbert M. Chichester: Wiley, 1997. Cited by Ellaway et al. (2004) for the risk factors of domestic violence and mental illness.

Homicide and the baby-sitter. Dix, J. Am J Forens Med Path 1998 Oct; 19(4): 321-323.

Victim, perpetrator, family, and incident characteristics of 32 infant maltreatment deaths in the United States Air Force. Brewster AL, Nelson JP, Hymel KP. Child Abuse & Neglect 1998; 22(2): 91-101

Bias in child maltreatment reporting: revising the myth of classlessness. Drake B and Zuravin S. Am J Orthopsychiatry 1998; 68: 295-304 Review of the literature on class bias in CPS databases and the class epidemiology of child abuse. Finds that the reported high levels of child abuse among the poor are real, and not an artifact of biased reporting or case-finding.

The child-abuse lottery -- will the doctor suspect and report? Physician attitudes towards and reporting of suspected child abuse and neglect. Van Haeringen AR, Dadds M, Armstrong KL. Child Abuse & Neglect 1998; 22: 159-169. A survey of physicians in Queensland. 43% reported having at one time or another decided not to report. Reluctance was based on (a) problems differentiating accidental from abusive, (b) defining emotional abuse, (c) perceived adverse consequences of reporting for both the physician and the family.

Unemployment Rates, single parent density, and indices of child poverty: their relationship to different categories of child abuse and neglect. Gillham B, Tanner G, Cheyne B, Freeman I, Roney M, Lambie A. (Study from the U.K.) Child Abuse & Neglect 1998 Feb; 22(2): 79-90

1. Is there a rel betw unemployment, single parent, poverty, and any forms of child ab?
2. Is there a specific rel for different forms of abuse?

Results:

1. Yes there is a rel betw all of these indices of deprivation and all forms of abuse. Male unemployment strongest correl and strongest with physical abuse. Sexual abuse and neglect showed weaker correl. Male unemployment correl strongly with physical abuse and less strongly with other forms of abuse. Other indicia of deprivation were weakly correlated. Male unemployment explained two-thirds of the variance in both abuse and neglect.

Are battered women responsible for protection of their children in domestic violence cases? Wilson C. Journal of Interpersonal Violence 1998; 13(2): 289-293. The dynamics of domestic vi interacts with that of child abuse. This article talks about the fact that these two phenomena have been managed by two separate treatment subspecialties. The dom vi subspecialty argues that the mother is not "responsible for" her child's abuse, because saying that would imply that she is an aider and abettor of the (male) perpetrator and would punish her for her decisions to remain in the home and to cover up the abuse, which would in effect "blame the victim." The child ab subspecialty argues that the mother is "responsible for" her child's abuse because her "decisions" helped cause it and are helping continue it.

Identification of child maltreatment with the parent-child conflict tactics scales: development and psychometric data for a national sample of American Parents. Straus M, Hamby S et al. Child Abuse & Neglect 1998; 22: 249-270.

A Nation's Shame: Fatal Child Abuse and Neglect in the United States. U.S. Advisory Board on Child Abuse and neglect. Washington, D.C.: U.S. Department of Health and Human Services, 1995. Cited by Susan Broderick, J.D. in *APRI Update*, vol 17, no. 8, 2004, for the proposition that an estimated 2000

children die from abuse and neglect each year, and that approximately 40% of those children are under one year old, and the majority are under five years old.

David Finkelhor, personal communication, 1998:

Contends that child maltreatment reports are levelling off. "While child abuse 'reports' are up since 1980, in fact reports have stopped their sharp increases since the early 1990's. Sexual abuse reports are actually in decline, ... child abuse fatalities have been fairly flat for the last few years." Comparing this view to the oft-quoted findings from the NIS-3 study (National Incidence Study of Child Abuse & Neglect), which estimated that maltreatment nearly doubled between 1986 and 1993, from 1.4 million children to 2.8 million, with serious injury children quadruple from 143,000 to 570,000, Dr. Finkelhor states that NIS-3 measured just two points in time (1986 and 1993) and captured an increase in reports that was still occurring in the late 1980's. See communications from Drs. Finkelhor, Caldwell, and Trocme, Nov. 4th, 5th, 6th, 1998 in file.

Impoverishment and child maltreatment in African-American and European-American neighborhoods.

Korbin JE, Coulton CJ, Chard S et al. Dev Psychopathol 1998 Spring; 10(2): 215-233. There were differences in prevalence of maltreatment reports by ethnicity. Impoverishment had a weaker effect on the report rate in African-American neighborhoods than in European-American neighborhoods. "Ethnographic data point to the importance of the social fabric in accounting for differences in child maltreatment report rates by predominant neighborhood ethnicity."

A longitudinal analysis of risk factors for child maltreatment: findings of a 17-year prospective study of officially recorded and self-reported child abuse and neglect. Brown J, Cohen P, Johnson JG, Salzinger S. Child Abuse & Neglect 1998; 22(11): 1065-1078. Logistic regression analysis of state records, self-report instruments, and surveys administered to 644 families in upstate New York four times between 1975 and 1992 showed that there were different sets of risk factors for sexual abuse, physical abuse, and neglect. But maternal youth and maternal sociopathy predicted all three. The risk factors were somewhat predictive: the prevalence of maltreatment was 3% when no risk factors were present, and 24% when four or more risk factors were present.

Neuropsychological functioning: comparison of mothers at high and low risk for child physical abuse.

Nayak MB and Milner JS. Child Abuse & Neglect 1998; 22(7): 687-703. The main factors were IQ and depression.

Epidemiology of sexual abuse of children: old problems, new directions. Leventhal JM. Child Abuse & Neglect 1998; 22(6): 481-491. A literature review by a well-known academic researcher.

See Profile of an abused burned child, below, under BURNS.

Predicting child maltreatment in the first four years of life from characteristics assessed in the neonatal period. Kotch JB, Browne DC, Dufort V, Winsor J. Child Abuse Negl 1999 Apr; 23(4): 305-19. Low social support at the time of birth increased the risk of a maltreatment report by 4X.

Trends in intentional injury deaths in children and teenagers (1980-1995). (UK) Roberts I, Li L, Baker M. J Public Health Med 1998 Dec; 20(4): 463-466. In the UK accidental deaths declined but intentional deaths did not. There is a steep class gradient in intentional death rates, which has widened over time.

Risk factors for infant homicide in the United States. Overpeck MD, Brenner Ra, Trumble AC, Trifiletti LB, Berendes HW. NE J Med 1998; 339: 1211-1216. Cited in Hymel et al., 2007 (see under "Shaken") for the proposition that AHT is two-thirds of infant homicides.

Underascertainment of child abuse mortality in the United States. Herman-Giddens ME, Brown G, Verbiest S, Carlson PJ, Hooten G, Howell E, Butts JD. JAMA 1999; 282: 463-467. A ten-year retrospective study of medical examiners records in North Carolina pertaining to child abuse homicides 1985-1994. Concludes in part "using medical examiner data, we found that significant underascertainment of child abuse homicide in vital records systems persists despite greater societal attention to abuse

fatalities.” In criticising this article, Dr. Edmund Donoghue and subsequently Dr. JD Butts of the NC OCME point out that the NC vital records office (not the CME’s office) uses a restrictive definition of child abuse homicide, requiring that either the ME list abuse, beating, or other maltreatment as a condition on the DC, or that the DC must otherwise list conditions consistent with previous abuse. This gave rise to a lower incidence than a broader definition applied by the NC Child Fatality Prevention Team. This discrepancy in definitions need not amount to underascertainment. The vital records definition leads to an underascertainment of *child abuse mortality* if you only query death certificate data. It does not lead to an underascertainment of *child homicides*. (Donoghue1@AOL.com and jbutts@OCME.UNC.EDU on NAME-L Aug 16 99).

Risk factors for infant homicide in the United States. Overpeck MD, Brenner RA, Trumble AC, Trilifetti LB, Bernendes HW. NE J Med 1998 Oct; 339(17): 1211-1216. A death-certificate study from the NCHS. 2,776 infant deaths from homicide (includes UNDET) during the period 1983-1991. Homicide is the leading cause of infant deaths due to injury. Reviewed birth certificates and death certificates. Half the infant homicides occurred by the fourth month of life. Homicide rate = 8/100,000 live births. 27% of all fatal infant trauma was homicidal. 5% occurred on the first day of life; 50% occurred in the first three months.

Maternal risk factors:

Age under 19, education under 12 y, timing of 1st PNC, no PNC, single, Black or Indian,

Infant risk factors:

LBW, prematurity, birth order

From the abstract:

Risk factors:

2d or higher birth-order child of a

Mother under 17	relative risk	10.9 compared to m 25 y o
-----------------	---------------	---------------------------

Mother under 19	relative risk	9.3 “ “
-----------------	---------------	---------

No PNC	relative risk	10.4
--------	---------------	------

Less than 12 yrs educ	relative risk	8.0 compared to 16 y educ
-----------------------	---------------	---------------------------

There are comments at 1998 Oct 22 339(17): 1239-1241; 1999 Mar 18; 340(11): 895.

Annual summary of vital statistics -- 1998. Guyer B et al. Pediatrics 1999; 104: 1229 Homicide is the third leading cause of death of children between 1 and 4. (note, homicide, not child abuse).

Deaths attributable to injuries in infants, United States, 1983-1991. Brenner RA, Overpeck MD, Trumble AC, DerSimonian R, Berendes H. Pediatrics 1999; 103: 968-974. The risk factors for all types of trauma to infants under one year. The leading causes of death were:

Homicide

Suffocation

MVA

Choked on something

Homicide rate increased over the period. The main risk factors for trauma were:

No PNC

Mother less than 12 yrs ed

Mother under 20

Birth order over 3

Indians

Child abuse and unintentional injuries: a 10-year retrospective. DeScala C, Sege R, Li G, Reece R., Arch Pediatr Adol Med 2000 Jan; 154: 16-22. Analyzed ten years of the National Pediatric Trauma Registry (NPTR) for hospital admissions for trauma in children 0-4 years old. 1,997 abuse admissions (10%) and 16,831 accident admissions (90%). Abuse victims were younger (13 vs. 25 months). Accidents leading to admission were overwhelmingly falls and MVA’s (58% and 37%). Abuse had much more intracranial injury (42% vs 14%) and much more RH (28% vs .06%).

Rates of child abuse and neglect continue to decline for fifth straight year in US. (Reuters Medical News on Mescap, dated April 12, 2000. <http://www.pediatrics.medscape.com/reuters/prof/>

Says the US DHS has announced that number of abused & neglected ch fell to 903,000 for 1998, with an incidence of 12.9 per thousand, "the lowest rate in over 10 years." Announcemdnets was by Secy. Shalala. Total no. is 11% below the 1993 peak. 54% neglect, 23% physical abuse, 12% sexual abuse. 1,100 deaths. 80% of all maltreatment was by "parents," [?i.e. caregivers]. HHS attributed the fall in rates to the Adoption and Safe Families Act of 1997 and community-based prevention programs. The name of an official is given. Also see next.

Identification of violence in the home: pediatric and parental reports. Kerker BD, Horwitz SM, Leventhal JM, Plichta S, Leaf PJ. Arch Ped Adol Med 2000 May; 154(5): 457-462. In this questionnaire study of parents in 19 private pediatric practices, 21% of mothers reported leaving a mark, while only 0.5% of patients were identified by the pediatricians as physically abused. Mothers experiencing domestic violence were 1.6X more likely to report leaving a mark, but even when the physicians knew there was DV, they had no increased rate of diagnosing child abuse. Result: parents report substantially more DV and child abuse than pediatricians detect. Pediatricians should ask parents directly about DV and CA. (have abstract)

Evidence inconsistent for intergenerational continuity of child abuse. Reuters Medical News on Medscape, September 5th, 2000. Citing Ertem IO et al., Lancet 2000 Sep 2; 356: 814-819. A literature review of ten published studies of the "cycle of abuse." It says that most of the studies were of poor quality by stated quality criteria, only one of them of excellent quality. That one found an increased risk of 12X, but another found nothing. Total, four studies found no increased risk and six found yes increased risk.

Identification of violence in the home: pediatric and parental reports. Bonnie D. Kerker, MPH, Sarah McCue Horwitz, PhD, John M. Leventhal, MD, Stacey Plichta, ScD, Phillip J. Leaf, PhD. Arch Ped Adol med 2000 May; 154(5): 457-462. Pediatricians only picked up DV 0.3% of the time, while mothers self-reported it 4.2% of the time. As far as child physical abuse, pediatricians only picked it up 0.5% of the time, while mothers self-reported hitting their children and leaving a mark 21% of the time. Hence a lot of violence in the home currently goes undetected unless pediatricians directly ask about it.

Sociodemographic risk and child well-being. Kristin Anderson Moore and Sharon Vandivere (Child Trends) and Jennifer Ehrle (The Urban Institute). One of three papers published by the Urban Institute, June, 2000. (See next) Sociodemographic risk is factors of social environment that increase the risk of children having negative outcomes including school problems, behavioral problems. The risk factors are

- (a) family income below the poverty level
- (b) unmarried parents
- (c) household with four or more ch
- (d) parents without high school education

Turbulence and child well-being. Same authors, June 2000. Same negative outcomes. Turbulence defined as moving from one state to another, moving to a different home, moving in with another family, two or more changes in employment by parent, two or more school changes, significant decline in health.

Stressful family lives: child and parent well-being. Same source. Stressful family environment defined as

- (a) Unable to pay the rent
- (b) More than two people per bedroom
- (c) Food money ran out
- (d) Not confident that they can get health care
- (e) Adult in poor mental or physical health
- (f) Child in poor mental or physical health

Had poor outcomes. (low school engagement, behavior problems)

Screening families with young children for child maltreatment potential. Murry SK, Baker AW, Lewin L. Pediatric Nursing 2000 Jan-Feb; 26(1): 47-54. 10% to 20% of chil under 12 experience physical, sexual abuse or neglect, acc to NIS-3 National Incidence Study of Child Abuse & Neglect. Risk indicators have been identified. These authors develop the Parenting Maltreatment Risk and Intervention Protocol to aid a

busy office or clinic practice setting in the prospective ID of families with ch under age 3 who are **at risk**, to guide initial intervention, referral, and followup care.

UK infants born outside marriage have higher 1-year mortality. Reuters Medical News on Medscape, November 17, 2001. Citing government figures. The infant mortality for out-of-wedlock births was 6.6 per 1000 LB and 4.8 for those whose parents were not married (both living together and not living together or unknown.)

A systematic investigation of 16 cases of neonaticide. Spinelli MG. Am J Psychiatry 2001 May; 158: 5-

Are father surrogates a risk factor for child maltreatment? Radhakrishna A, Bou-Saada IE, Hunter WM, Catellier DJ, Kotch JB. Child Maltr 2001; 6: 281-289. Yes. A boyfriend in the home gave a relative risk of 2.0 relative to no male in the home and 2.6 relative to a biological father. Interestingly, having a biological father in the home actually lowered the risk of child maltr, although not statistically significant.

Racial and ethnic disparities in infant mortality rates -- 60 largest US cities, 1995-1998. CDC MMWR 2002 Apr; 51(15): 329-332, 343. (have) Background data. The Black IMR was 13.9 per 1000 LB. White 6.4, hisp 5.9. VLBW accounted for about 2/3 of the black overmortality. Nott accounted for by economic circumstances: poor hisp newborns weighed more than equally poor black newborns. Uneducated white nbs weighed more than equally uneducated black nbs. Black excess mortality was correlated with degree of segregation of the cities in question. Has a graph of IMR by city of residence. Has a table showing the risk factors used broken out by race: birth weight, individual-level factors (maternal age, ed, late PNC), community-level factors (pop, "dissimilarity" (?), median income, degree of childhood poverty).. Discusses the CDC programs in being to reduce infant mortality generally and disparities in mortality.

Household composition and risk of fatal child matreatment. Stiffman MN, Schnitzer PG, Adam P, Kruse RL, Ewigman B. Pediatrics 2002; 109: 615-621. Missouri: child deaths under 5. See Dr Reece's review in *Child Abuse Quarterly* for July 2002 which advises that:

- 2,591 total deaths under 5
 - 1,639 eligible deaths (after excluding SIDS, congenital disease, prematurity, etc.)
 - 1,348 non-trauma deaths
 - 291 trauma deaths
 - 175 were due to maltreatment (60%)
 - 55 inflicted trauma (physical abuse)
 - 39 shaking, dropping, or hitting with hands
 - 11 injured with object
 - 5 unknown mechanism of injury
 - 120 neglect
 - 31% fires
 - 26% drown
 - 14% unsafe sleep
 - 14% MVA
 - 7% choke or strang

Dr Reece further summarizes that "Children residing in hh with adults unrelated to them had the highest risk of maltr, compared to chil residing in hh with two biol parents... Chil in hh with step-, foster-, or adoptive parents also had an increased risk of maltr deaths. This study found that the incr risk of maltr deaths, therefore, occurs primarily in hh which include biol unrelated adult males who are **boyfriends** of the child's mother." NO incr risk in single-parent hh if it was a biol parent and no other adults present; Dr Reece notes that this latter result is in contrast to other studies regarding single-parent hh's. Dr Reece summarizes with the following carefully worded comment: "[I]t adds to an emerging sense that single women with children need to be discerning in their choices of male live-in companions."

Victim, perpetrator, family, and incident characteristics of infant and child homicide in the United States Air Force. Lucas DR, Wezner KC, Milner JS et al. Child Abuse & Neglect 2002; 26: 167-186. Retrospective analysis of 32 homis ages 1 to 15, analyzed for numerous variables.

Poor adolescent expectant mothers: can we assess their potential for child abuse? Zelenko MA et al. J Adol H 2001; 29: 271-278. Fifty pregnant poor girls completed a Child Abuse Potential Inventory. ???

Child abuse and neglect presentations to a pediatric emergency department. Keshavarz R, Kawashima R, Low C. (Sinai) J Emerg Med 2002 Nov; 23(4): 341-345. Retrospective chart review of all the PED admissions that were referred to CPS over a two year period. 106 cases. Mean age 6.4 years. 55% physical, 15% sexual, 30% neglect. The suspect was the mother in 41% and the father in 21%. Bruises the most common injury (25%). Found 69% presented at night. Cases had an average of 6 previous ED visits. Found 89% had medicaid compared with 71% of other PED pts. Of the 106 referrals, CPS found 46% "indicated," 20% unfounded, and 36% undet. The majority of the reported cases did not have any physical findings. (In other words, they were referred based on historical or circumstantial factors.)

Child Abuse: Guidelines for Identification, Assessment, and Case Management. Marilyn Strachan Peterson and Michael Durfee, eds. Volcano, California: Volcano Press, 2003.
<http://www.volcanopress.com/>

Child abuse and the otolaryngologist, parts I and II. Crouse CD, Faust RA. Otolaryngol Head Neck Surg 2003 Mar; 128(3): 305-310, 311-317. Says child abuse incidence in US is about 1 million chil per year, with a greater than 1% mortality.

Are abused babies protected from further abuse? Ellaway BA, Payne EH, Rolfe K, Dunstan FD, Kemp AM, Butler I, Sibert JR. Arch Dis Chil (UK) 2004; 89: 845-846. From the abstract: "A cohort of 69 physically abused babies under 1 year was followed for three years... Of the 49 babies who returned home...15 were further abused, a reabuse rate of 31%, which is grossly higher than the abuse rate in the general population." The profile of risk factors in the reabusing families did not differ from that in the not-reabusing families. This included domestic violence and mental illness, consistent with the previous reports of Browne & Herbert, 1997.

Risk factors for infant maltreatment: a population-based study. Wu SS, Ma C-X, Carter RL, Ariet M, Feaver EA, Resnick MB. Child Abuse & Neglect 2004; 28: 1253-1264. In the year 2002, 900,000 US children were determined to be victims of abuse or neglect. The 0-3 year age group had the highest victimization rate (1.6%) and constituted 9.6% of all victims. These authors prospectively observed a cohort of 189,000 children born in 1996 in Florida with reference to 15 sociodemographic variables. Found that by age 1, 1,602 of the children had suffered maltreatment. Five of the sociodemographic variables showed a relative risk of 2 or greater with respect to these children. These five factors were:

mother smoked during pregnancy	2.8
more than two siblings	2.7
medicaid	2.1
unmarried mother	2.0
low birth weight	2.0

If an infant had four of these risk factors, it had 7X the population average relative risk of maltreatment. The total risk factors looked at were: mother's race, mother's education, mother's age during pregnancy, mother's marital status during pregnancy, previous adverse pregnancy outcome, interpregnancy interval, prenatal care ("inadequate" if <50% of the required visits), smoke, sex, LBW, medicaid, WIC, prenatal risk screen score. Factors in addition to the above five that had elevated relative risks included:

maternal education <HS	1.7
prenatal care inadequate	1.5
prenatal risk score	1.4
race	*

(* = not rated but appears to be 1).

Can these data be used to prevent? Some data shows that risk-targeted home intervention programs have not reduced maltreatment. The authors offer these data as a tool for targeting preventive interventions, but do not make any claims about the expected results. The article contains a condensed review of the literature on risk factors and prevention. My thanks to Tim David, MD for providing me with this article.

Are abused babies protected from further abuse? Ellaway BA, Payne EH, Rolfe K. (Cardiff) Arch Dis chil (UK) 2004; 89: 845-846. In 69 physically abused babies, 49 were returned to their homes. fifteen of these were re-abused, for a reabuse rate of 31% .

Identifying non-accidental injury in children presenting to A&E departments: an overview of the literature. Sanders T, Cobley C. (Manchester). Accident and Emergency Nursing 2005; 13: 130-136. This literature review presents the evidence that child abuse is being underrecognized in emergency rooms in the United Kingdom. The reporting of it to the protective agencies is less than the prevalence of it as indicated by local prevalence statistics. Cites Jenny, 1999, Sundell, 1997, Sidebotham and Pearce, 1997, and Haeringen et al., 1998 for the proposition that many cases remain undetected. Clinicians often accept explanations for minor trauma and nonsevere symptoms. As to severe trauma and severe symptoms, is there an epidemic of underreporting of severe NAI? The reported incidence of SBS in children under 12 months is 21-24 per 100,000 per year, and 36 per 100,000 in the under six months age group, citing Jayawant et al., 1998, and Barlow & Minns, 2000. Most SDH's under 12 months are abusive. They may have mild symptoms. Risk factors for abuse: very young age, bruises on the face, ear, abdomen or buttocks, fractures, esp. spiral fractures, and bathtub drowning over the age of 18 months (which is always due to either epilepsy or abuse, according to Kemp et al, 1994. It is "very rare" for children over 3 to present with NAI. Social risk factors for abuse: very limited research:

High specificity for abuse

spiral fractures
SDH
previous injury

Low specificity for abuse

previous DCFS contact
alcohol abuse
marital conflict
poverty

Accordingly, social risk factors should not be used by themselves as evidence of abuse, just as indicators for further investigation. Why was NAI underreported in the emergency room? 1. Lack of knowledge about risk factors. 2. Lack of availability of specialist consultation. 3. Too difficult to gain access to the local child abuse register, largely because of bureaucratic barriers. In other words, the causes are systemic. Lord Laming's inquiry into the Victoria Climbié case led to some systemic reforms being recommended but not implemented, such as a national database on all children under 16 with a detailed searchable electronic record.

A population-based study of inflicted traumatic brain injury in young children. Keenan HT, Runyan DK, Marshall SW, Nocera MA, Merten DF, Sinal SH. JAMA 2003 Aug; 290(5): 621-626.

1. The authors' purpose was to determine the *incidence* of AHT. They start out by saying that the incidence of AHT is unknown. So they prospectively followed all North Carolina children aged 2 and under who were admitted to a PICU or died of TBI in 2000 and 2001. They found that there were 152 cases of serious or fatal TBI, of which 80 (53%) were inflicted TBI. Out of a state population of 230,000 children under 2, this yielded an incidence of 17 per 100,000 person-years. Relative to the general population, children who incurred an increased risk of inflicted TBI were born to mothers under 21, non-EuroAmerican, or were products of multiple births.
2. The authors defined AHT (inflicted TBI) as a confession or a CPS determination of abuse.
3. Note that the non-EuroAmerican demography did not apply only to inflicted. It also applied to non-inflicted. In other words, the non-EuroAmerican group had a higher risk of both accidental and nonaccidental serious head injury. The incidences were as follows

inflicted

non-inflicted

Euroamerican	9.5	11.5
African-American	27.1	19.2
Other	95.7	62.0

The authors comment that “[R]ace/ethnicity was highly predictive of inflicted injury compared with the general North Carolina population.” BUT: “Race/ethnicity was not a good predictor of inflicted TBI versus noninflicted TBI; rather, minority children were at increased risk of all types of TBI.” There is a great deal of other demographic data in the article.

4. The authors compare their data to those of Barlow & Minns (Lancet, 2000). Those authors obtained an incidence of 24.6, with a 95% confidence interval of 14.9 to 38.5, compared to the present authors’ 17 with 13.3 to 20.7. As the authors state, their incidence finding is within the confidence interval obtained by Barlow & Minns., but may have been artificially lowered relative to Barlow’s because the present authors excluded all children over 2.

This article is cited by Dr Reece in his review of Leestma, 2005 in the *Quarterly* for Winter 2005, for the proposition that more shaking confession cases have been published since 2001 than in all the years before 2001. But note that this article does not say how many confessions and gives 0 clinical details, thus tending to confirm Leestma’s observation that the shaking articles are vague on clinical and historical detail.

Women who kill their children. Rouge-Maillart C et al. Am J Forens Med P 2005 Dec; 26(4): 320-326.

17 child-killing mothers in Angers, France, with 19 autopsied children. The mean age was 29 for the women and 3 for the children. Head trauma, strangulation, suffocation, and drowning were the most common means, but more violent means such as stabbings and shootings did occur. 15 of the 17 women showed mentally disturbed behavior including suicidal tendencies. The incident was frequently precipitated by a dispute.

Child deaths resulting from inflicted injuries: household risk factors and perpetrator characteristics. Schnitzer PG, Ewigman BG. Pediatrics 2005; 116: e687-e693. An epidemiologic study of all the children who died in Missouri over an eight year period. Had 901 deaths including 149 *inflicted* deaths. (Note I said “inflicted,” not necessarily “abuse;” these included stranger-to-stranger assaults.) Found that the risk factors were; Black, young unmarried mothers, Medicaid-eligible mothers, less than a high school education, late or no prenatal care, young siblings in the same household, and prior report to DCFS. The fatal injuries were:

AHT		69
Thoracic, abd, or multiple trauma	39	
Suffoc		20
Burns, poisonings, etc.	13	
Shot with gun		8

The authors compared the inflicted victims with a group of live controls. The inflicted victims had a much lower likelihood of living with both biological parents (37% versus 64% of controls.) According to Dr Reece’s review in the Winter 2006 *Quarterly*, children living in households with an adult unrelated to them were almost 50 times as likely to die of an inflicted injury than children living in household with two biological parents. 82% of the fatal unrelated adults were boyfriends. The perpetrators were 71% male. Also,

fathers	35%
boyfriends	24%
mothers	20%

Nonaccidental head injuries in children: a Sydney experience. Ghahreman A, Bhasin V, Chaseling R, Andrews B, Lang EW. J Neuros 2005; 103: 213-218. According to Dr Ricci's review in the Spring 2006 issue of the *Quarterly*, they had 65 cases in this retrospective review. Found that MRI was clearly superior to CT because CT missed four cases of ischemia, three cases of shearing injury, eight small subdurals, and overall + findings in 49%. As to epidemiology, had:

Perpetrator	
father	37%
boyfriend	31%
mother	15%
other relative	10%
babysitter	7%
Prior DCFS or DVcontact substantiated	24%
Hx alcohol ab, drug ab, or mental illn	14%

The risk of child abuse in infants and toddlers with lower extremity injuries. Coffey C, Haley K, Hayes J, Groner JJ. J Pediatr Surg 2005; 40: 120-123. According to Dr Greeley's review in the Spring 2006 issue of the *Quarterly*, had n = 1,252 patients with lower extremity injuries.

		abuse	not abuse
under 18 mos	(# 66)	44 (67%)	22
over 18 mos	(# 1,186)	16 (1%)	1,170

In the under 18 mos abuse group, the most common fractures were femur (22) and tibia (14). In the under-18 mos group, 74% of all the children admitted with lower extremity fractures were abuse cases.

Child abuse registration, fetal growth, and preterm birth: a population-based study. Spencer N, Wallace A, Sundrum R, Bacchus C. J Epidemiol Community Health (UK) 2006; 60: 337-340. Correlated 119,729 children registered with the child abuse authorities with their birth weight and term. Carla DiScala notes in her review for the Autumn 2006 *Quarterly*, "The authors' conclusion suggests that further studies are needed to explore whether poor fetal growth and preterm birth predispose to child abuse or whether they are indicative of maternal characteristics that increase the risk of poor pregnancy outcome AND child abuse."

BIRTH INJURY

See bibliography prepared by ---- MD on neonatal and intrauterine skull fractures, 2001. In file. See also Kleinman.

Accidental trauma complicating pregnancy and delivery. Dyer et al. Am J Obstet Gynecol 1962 Apr;

Retinal hemorrhage in newborn infants. Sezen F. Br J Ophthalmol 1970; 55: 248

Benign subdural collections of infancy. Robertson WC, Chun RWM, Orrison WW et al. J Pediatr 1979; 94: 382

Rib fractures as a cause of immediate neonatal tachypnoea. Landman L, Homberg R, Sirota L, Dulizky. Eur J Pediatr 1986; 144: 487-488. Reference kindly provided by Dr. Allen R. DeJong, director of the Children At Risk Evaluation Program at the Alfred I. DuPont Hospital for Children.

Tentorial hemorrhage associated with vacuum extraction. Hanigan WC et al. Pediatrics 1990; 85: 534-539.

Birth trauma. Schullinger JN. Pediatr Surg 1993; 40: 1351-1358. Cited in BAT Child Abuse, Check Sample FP 00-5, infra under Abdominal Injury, for the proposition that the most common (abdominal) birth injury is the subcapsular hematoma of the liver, which can rupture up to one week postpartum; and that other abdominal birth injuries occur but are uncommon, including adrenal hematomas, liver lacerations, , K and spl inj.

CT and MRI characteristics of intracranial hemorrhage complicating breech and vacuum delivery. Odita JD and Heibi S. Pediatric Radiology 1996; 26: 782-785

[Posterior rib fxx in LGA infants as birth injury] Arch Path Lab Med 1997 Sep; 151: 947. Two cases of multiple posterior rib fxx detected at birth following vag deliv of LGA

Subdural neomembranes and sudden infant death syndrome. Rogers CB, Itabashi HH, Tomiyasu U, Heuser ET. JFS 1998; 43: 375-376. Cf Chamnanvanakij, 2002, under Shaken, below. See also Ken Feldman, 2001, under Shaken.

Newborn clavicle fractures. McBride MT, Hennrikus WL, Mologne T. Orthopedics 1998; 17: 317-320. 9,106 neonates were prospectively screened by radiology for clavicle fractures. They found an incidence of 0.5%, or one in every 213 live births. Risk factors were LGA, post-dates, shoulder dystocia, and mechanically assisted delivery. Not a single case occurred in caesarean section deliveries.

Subgaleal hemorrhage in a premature infant with congenital diaphragmatic hernia following vacuum-assisted delivery. Posen R, Chan PT, deLemos RA. (USC) J Matern Fet Med 1998 May-Jun; 7(3): 132-136. Case report. This hemorrhage was severe enough to make the newborn ineligible for ECMO.

Effect of mode of delivery in nulliparous women on neonatal intracranial injury. Towner D, Castro MA, Eby-Wilkens E, Gilbert WM. NE J Med 1999 Dec; 341(23): 1709-1714. Intracranial hemorrhage occurs in C-sections done *during* labor, but not in those done *before* labor. It occurs regardless of whether delivery instrumentation was attempted. So the risk factor is abnormal labor, not instrumentation. (have abstract) See editorial in same issue.

Cranial birth injuries: analysis and identification of risk factors. John Pollina MD and Mark S. Dias MD. Unpublished manuscript, 2000. Retrospective review of all birth trauma at Cornell Hosp 1991-98. 32 cases with controls. Avg 39 wks GA (36-42). 28 vag, of which 18 were either vacuums or forceps or both. 47% primips. 50% SDH, of which 58% were interh, 46% tentorial, 21% convx, 8% posterior fossa. All but four were nonoperative. Intraparenchymal hemorrhage in 23%. One death.

Imaging of child abuse. Nimkin K and Kleinman PK. Radiol Clin N Amer 2001 Jul; 39(4): 843-864 at 857. Clavicle most common. "Long-bone fractures have decreased in freq in the modern obstetric era; however, humeral fractures, and less commonly femoral fractures, are still encountered. These fractures are frequently char by exuberant callus formation within the first few weeks of life, and the absence of callus formation in a long-bone, clavicular, or rib fracture at 11 days of age weighs heavily against a birth injury."

Obstetric vacuum extraction: state of the art in the new millennium. Mikovsky P, Watson WJ. Ob Gyn Surv 2001 Nov; 56(11): 736-751. Vacuum extraction is replacing forceps as the preferred method of instrumental delivery.

Classic metaphyseal lesion following external cephalic version and caesarean section. Lysack JR, Soboleski D. Pediatric Radiology 2003; 33: 422-424. From Dr David Merten's review in the Jan 04 *Quarterly*, the left leg had a symptomatic bucket-handle fracture discovered three hours after delivery. It later formed a large callus. Dr Merten comments, "Obstetrical epi-metaphyseal fractures encompass a spectrum of injuries similar to those due to abuse..."

[Subdural haemorrhages at birth] Whitby E et al. (Sheffield) Lancet 2004 Mar 13; 362: 846-851. As per a note in Medscape Medical News by Gary D. Voglin, MD, these British radiologists used an MRI scanner to do two years of followup on subdural hemorrhages present at birth in 9 babies (3 NSVD, 5 forceps after failed vacuum, 1 traumatic vacuum). Out of 111 live deliveries during the initial study period. None required any treatment. All SDH's resolved by 4 weeks, and there were no recurrences or residuals out to two years. Dr Voglin quotes the lead author to the effect that, "These lesions seem to be benign, clinically asymptomatic, and of no long term importance." Therefore, if a SDH is found in an infant more than four weeks of age, it cannot be of birth origin.

The following (in italics) is a bibliography on birth injuries prepared by Dr Christopher Greeley of Vanderbilt University School of Medicine (2007). Dr Greeley has located six published case reports of rib fractures as a birth injury. All were big babies:

Thomas, Rib Fractures in Infancy. European Society of Pediatric Radiology, Stockholm, 1976
2 infants with birth related rib fracture, (1) diagnosed at 3 weeks (not moving arm), forceps delivery, 3 posterior rib fractures noted, (2) OI who died at birth

Rizzolo and Coleman, Journal of Family Practice v29,n5 (Nov 1989): pp561 (3)

Term, (BWt >7#), vacuum used, shoulder dystocia, 9 hours of life developed respiratory distress, crepitus palpated, CXR revealed 5 posteriolateral rib fractures, no other fractures noted, bones appeared normal, abuse investigated, follow-up chest xray at 3 weeks showed normal healing.

Barry and Hocking, Archives of Disease in Childhood 1993; 68: 250

Term (5020 gm), shoulder dystocia, crepitus felt at 9 hours of age, x-ray revealed 5 posterior rib fractures, no other fractures, bones appeared normal

Hartmann, Archives of Pediatric and Adolescent Medicine, Vol 151 (9). September 1997: 947-948

LGA (3912 gm), vacuum extraction, VBAC, immediate concern by OB for clavicle fracture, exam with crepitus, no distress, CXR with 5 posterior rib fractures

Term, LGA (4205Gm), vacuum extraction, exam with crepitus and bruising, no respiratory distress, CXR revealed 3 posterior rib fractures

Bulloch et al., Pediatrics Vol. 105 No. 4, April 2000

Term (3946 gm), vacuum extraction, shoulder dystocia, noted to be grunting, DOL 4 noted to have 3 posterior rib fractures and clavicle fracture, abuse investigated

Other Birth Injuries

Rubin, Birth Injuries: Incidence, mechanism and end results. Obstetrics and Gynecology 1964; 23: 2; 218-221

15,435 birth reviewed (6 year period), 108 with injuries, 43 clavicle fractures, 7 humerus fractures, no rib fractures

Levine et al., Birth Trauma: incidence and predisposing factors. Obstetrics and Gynecology 1984; 63: 729-795

13,870 births reviewed for injuries, no rib fractures noted

Bhat et al., Bone injuries during delivery. Indian Journal of Pediatrics. 61(4):401-5, 1994 Jul-Aug
34,946 newborns evaluated for bone injuries, no rib fractures found

Cumming, Neonatal Skeletal Fractures: birth or child abuse? Journal of the Canadian Association of Radiology. 1979; 30:30-33

233 newborns with fractures from deliveries, no rib fractures

Shoulder Dystocia

Baskett and Allen, Perinatal Implications of Shoulder Dystocia. Obstetrics and Gynecology, Vol 86, NO.1, July 1995; 14-17

10 year retrospective review revealed 254 deliveries complicated by shoulder dystocia, resulted in 13 fractures, no rib fractures, (McRoberts maneuver resulted in 1 clavicle fracture)

McFarland et al., Perinatal Outcome and the Type and Number of Maneuvers in Should Dystocia. American Journal of Gynecology & Obstetrics 55 (1996), 219-224

Retrospective review of 276 deliveries complicated by shoulder dystocia, 8.5% resulted in clavicle fracture, 1.1% resulted in humeral fracture, no rib fractures were noted.

Gherman et al., Obstetric maneuvers for shoulder dystocia and associated fetal Morbidity. Am J Obstet Gynecol 1998;178:1126-30

Retrospective review of 285 deliveries complicated by shoulder dystocia, 27 had clavicle fractures, 12 humeral fractures, no rib fractures noted (McRoberts maneuver alone resulted in 12 clavicle fractures and 1 humeral fracture)

Baskett, Shoulder Dystocia. Best Practice & Research Clinical Obstetrics and Gynaecology. Vol 16, No. 1, 57-68, 2002

(Review Paper)

Fracture of clavicle occurs in ~15%, Fracture of humerus is <1%, rib fractures are not mentioned

FETAL INJURY

Fetal subdural hemorrhages. Fetal bilateral subdural hemorrhages: prenatal diagnosis and spontaneous resolution by time of delivery. Barozzino T, Sgro M, Toi A et al. Prenatal Diagnosis 1998 May; 18(5): 496-503. According to the abstract, this mother had minor abdominal trauma at 30 weeks. She also had Factor XI deficiency, which was considered to be etiologically insignificant. The authors also consider the abdominal trauma etiologically insignificant. The bilateral fetal SDHs were discovered by routine ultrasound. They resolved by the time of delivery, and there were no postnatal sequelae. "The etiologies and outcomes in the few previously reported cases are reviewed and compared with our case."

Fetal head trauma without maternal uterine injury. Watts Et al. Am J Ob Gyn 1967 Sep;

Fetal death secondary to nonpenetrating trauma to the gravid uterus. (letter). Raney EH. Am J Ob Gyn 1970 Jan;

Fetal death secondary to nonpenetrating trauma to the gravid uterus. Poulsen et al. Am J Ob Gyn 1973 Jun;

Fetal skull fracture from an apparently trivial motor vehicle accident. Cumming et al. Am J Ob Gyn 1978 oct;

Profile of a battered fetus. (letter). Morey et al. Lancet 12/5/81

Unexplained intracranial hemorrhage in utero: the battered fetus? Gunn TR and Becroft DM. Aust NZ J Ob Gyn 1984 Feb; 24(1): 17-22. Reports twenty stillborn Pacific Islander fetuses with unexplained intracranial hemorrhage at autopsy. "Prenatal subdural haemorrhage without a history of maternal trauma is extremely rare. In the absence of supporting maternal histories and other fetal or maternal injuries the possibility that these are 'battered' fetuses remains circumstantial."

Physical abuse in pregnancy. Hillard PJA. Ob Gyn 1985; 6: 185-189. Cited in Chapter 25 of Giardino & Alexander for the proposition that battering (DV) during pregnancy can cause miscarriage and stillbirth.

Battered and pregnant: a prevalence study. Helton AS, McFarlane J, Anderson ET. Am J Public Health 1987; 77: 1337-1339. Cited in Chapter 25 of Giardino & Alexander (below) for the proposition that battering during pregnancy can cause miscarriage and stillbirth.

Battering during pregnancy: the tip of an iceberg revealed. McFarlane J. Women's Health 1989; 15: 79-84. Cited in Chapter 25 of Giardino & Alexander (below) for the proposition that battering during pregnancy can cause miscarriage and stillbirth.

Estimating the time of fetal death in stillborn fetuses: I. Histologic evaluation of fetal organs: an autopsy study of 150 stillborns. Genest D, Boyd T. Ob Gyn 1992; 60(4): 575-584

Estimating the time of fetal death in stillborn fetuses: II. Histologic evaluation of the placenta: a study of 71 stillborns. Genest D, Boyd T. Ob Gyn 1992; 80(4): 585-592

Estimating the time of fetal death in stillborn fetuses: III. External fetal examination: a study of 86 stillborns. Genest D, Boyd T. Ob Gyn 1992; 80(4): 593-600

Gail Schauer summarized the above three papers into one table in Chapter 16 of the Tenth Edition of *Anderson's Pathology*.

Blows to the maternal abdomen causing fetal demise: a report of three cases and a review of the literature. Ribe JK, Teggatz JR, Harvey CM. JFS 1993 Sep; 38(5): 1092-1096. Says that if third-trimester gravidas were hit or kicked in the abdomen, 6% of them suffered traumatic abruption (figure derived from a review of the literature). Presents three cases of assaultive traumatic abruption -- two domestic violence and one stranger-to-stranger crime. The medical literature was found to contain 136 cases of blunt force assault on pregnant women, of which nine resulted in abruptio placentae. The article does not specifically discuss falls but the literature review shows that falls are an extremely rare cause of abruption; thus a history of a fall is suspect. The literature shows that gravidas who have been assaulted in a DV setting frequently falsify the history. See Giardino & Alexander chapter 25, below.

Bilateral subdural hematomas in a newborn infant. Stephens RP, Richardson AC, Lewin JS. Pediatrics 1997; 99: 619-621. Cited in Chapter 25 of Giardino & Alexander (below) for the proposition that battering of the abdomen can cause severe direct fetal injury.

Antepartum fetal intracranial hemorrhage: predisposing factors and prenatal sonography: a review. Sherer DM, Anyaegbunam A, Onyeije C. (Montefiore). Am J Perin 1998 Jul; 15(7): 431-441. Antenatal fetal intracranial hemorrhage may occur spontaneously, or occur in association with ITP or ATP, vWD, warfarin, Cocaine, maternal sz, severe abdominal trauma, amniocentesis, twin transfusion.

Fetal death after trauma in pregnancy. Theodorou DA, Velmahos GC, Souter I, et al. Am Surg 2000; 66: 809-812. (Los Angeles) Saw 80 gravidas requiring hospital admission for trauma, without regard to the mechanism or type of trauma. (Injuries not severe enough to require admission were excluded.) Did multiple regression for factors assoc w fetal loss. Found two factors assoc: trauma score greater than 9, and GA less than 23 weeks. Note that this is fetal death, not fetal trauma. Abdominal injury was more common in the cases of fetal death, but not statistically significant. 50% of fetal loss cases had abd trauma, 50% did not. Authors conclude that it was indirect systemic responses to trauma rather than uterine trauma that caused most of the fetal losses. Suggest neurohumeral responses.

See also case report under Menkes' disease, above.

Intrauterine subdural hemorrhage. Akman CI, Cracco J. Dev Med Child Neurol. 2000; 42: 843-846. Cited in Chapter 25 of Giardino & Alexander (below) for the proposition that abdominal battering can cause severe direct fetal injury.

Physical abuse of women before, during, and after pregnancy. Martin SL, Mackie L, Kupper LL, Buescher PA, Moracco KE. JAMA 2001 Mar; 285(12): 1581-1584. A questionnaire survey of 2648 postpartum women. Prevalence of abuse before pregnancy was 6.9%, during was 6.1%, after was 3.2%. 77% of those abused after pregnancy suffered injuries, but only 235 received treatment for their injuries. Since nearly all used well-baby care, well-baby care would be an effective situation for trauma screening of new mothers and injury referral.

Intrauterine subdural hemorrhage. Akman CI, Cracco J. Dev Med Child Neurol 2000; 42: 843-846. Case report of a 34 weeker deliv by C/S with a very large congenital unilateral chronic SDH causing herniation, which was drained. Review of the literature finds seven published cases of apparently spontaneous intrauterine SDH without any evidence of trauma or risk factors for trauma, and 24 cases with risk factors for trauma but no demonstrated trauma. Consider chemical and other coagulopathies.

Children with special needs, Chapter 25 in Giardino AP and Alexander R, *Child Maltreatment: A Clinical Guide and Reference, third edition* (2005). Discussing domestic violence, the authors Paula Jaudes and Linda Shapiro ascribe to Schroedel & Peretz (1994) four ways in which domestic violence adversely affects the fetus:

1. Battered women may be prevented from receiving prenatal care.
2. Battering during pregnancy is statistically correlated with low birth weight.
3. Battering can cause stillbirth and miscarriage (citing articles to be mentioned below).
4. Battering to the abdomen can cause direct fetal trauma (citing articles).

The authors go on to say that direct assault to the gravid abdomen "may be the cause of many of the unexplained prenatal causes of developmental disability... [and] unexplained premature labor." Consider the implications of that. -JKR

As to impact #3 above, the authors cite Hillard, 1985, Helton et al., 1987, Stewart et al, 1993, and Ribe et al., 1993. As to impact #4, they cite Stephens et al., 1997 and Akman et al., 2000.

BATTERED CHILD SYNDROME

See also the bibliography in *Investigation and Prosecution of Child Abuse, 2d ed. (1993)* by the National Center for the Prosecution of Child Abuse. (have)

The battered-child syndrome. Kempe CH, Silverman FN, Steele BF, Droegmueller W, and Silver HK: JAMA 1962 Jul; 181: 17-24 (have). Defining BCS as a specific diagnosis. But they do not in this article restrict it to cases of repetitive trauma, let alone to cases of repetitive skeletal trauma. "The clinical manifestations of the battered-child syndrome vary widely from those cases in which the trauma is very mild ... to those who exhibit the most florid ev of injury to the soft tissues and skeleton." "more often he shows ev of neglect including poor skin hygiene, multiple soft tissue injuries, and malnutrition." And giving the differential diagnosis as: scurvy, syphilis, infantile cortical hyperostosis, OI, paraplegia, congenital indifference to pain. Note that all of these are disorders involving multiple bony lesions, implying that in fact these authors considered BCS to be a radiologic diagnosis involving multiple fractures. "The metaphyseal lesions in particular occur in no other disease of which we are aware."

The battered child syndrome: responsibilities of the pathologist. Curphey TJ, Kade H, Noguchi TT, Moore SM. California Medicine 1965 Feb; 2: 102-104. (have) Pointing out that you need to perform (a) a complete external examination, (b) roentgenograms of the entire skeleton, (c) color photographs of the lesions for presentation in court, (d) microscopic study of the osseous lesions as well as lacerations and contusions and areas of hemorrhage adjacent to osseous lesions. Thanks to Dr. Chadwick for bringing this article to my attention.

Defined: “The battered baby syndrome (in a strict pathologic sense) refers only to those infants and children who have been victims of multiple repeated abuse of one type or another as indicated by convincing objective indicia of recent, healing and healed injuries involving the skin, viscera, or skeleton. The majority of fatally abused children do not meet the above criteria for inclusion in the battered baby syndrome. For example, in our experience approximately 15-20 percent of child abuse fatalities fulfill the foregoing criteria for the battered baby syndrome.” Zumwalt RE and Hirsch CS, Pathology of fatal child abuse and neglect. In: Helfer RE and Kempe RS, *The Battered Child*, 4th ed. Chicago: University of Chicago Press, 1987, p. 251.

Post-mortem radiological examination in infants: evidence of child abuse? Thomsen TK, Elle B, Thomsen JL. FSI 1997; 90(3): 223-230

From a radiologist’s judgment to public policy on child abuse and neglect: what have we wrought? The Neuhauser Lecture (Society for Pediatric Radiology). Pediatric Radiology 2000; 30: 219-228. Includes comments that the CPS system is overwhelmed, underfunded, and understaffed. See the SPR roundtable discussion next below. Says that too many families are charged with neglect just for being poor.

Controversial aspects of child abuse: a roundtable discussion. Society for Pediatric Radiology. Pediatric Radiology 2001; 31: 760-774. (have) Begins with an outcry by Dr. Krugman that the problem was abandoned by the Clinton administration and government generally. Mentions foster care: “Many children are still dying in foster care... CPS and the courts are overloaded with cases...” “no cohesive policy...” Home visits have been shown to prevent abuse (Olds); education has not. Describes a noncriminal approach to intervention used in Belgium and the Netherlands. What is our policy? “Is it adequate to just say we have to make sure that we criminally prosecute all those who violate our laws?” Government only spends \$3.33 per abused child. Advocates a personal-health approach rather than a criminal or social-welfare approach.

Dr. Felman from Florida, who works for the defense (his son is a defense lawyer) discusses the entity of small subdural hematomas with an enlarged subarachnoid space (see below under “Enlarged Subarachnoid Space”) as an example of the mis-prosecution of innocent parents. “It was apparent to me from this case, as well as others, that many of the people prosecuting these cases are not interested in the truth and that there are physicians who will support the efforts to convict innocent parents despite lack of compelling evidence for abuse.” As far as radiology reports, “The stakes are too high for equivocal readings.” (because of the criminalization of borderline cases.) Suggests that CPS agencies are sometimes altering radiology reports in order to obtain a conviction or other intervention. Asks that the SPR help the court acquire expert witnesses. “...there are significant problems in the way we select, evaluate, and dispose of cases of alleged child abuse in this country.”

Dr. Boal reviews her material of 910 cases referred for radiological evaluation of possible abuse at Hershey. The system found 303 of these as definite abuse. 24 abused children, including 14 fatalities, had negative imaging studies. Reviews who the perpetrators were in 201 nonfatal and 102 fatal cases: in fatal cases, it was boyfriend 28 css, mother 26, father 25, BBS 10, other in 13. (Fatal css have a slight preponderance of boyfriend as opposed to nonfatal cases.) Tries to evaluate whether child abuse can be diagnosed by evaluating fractures alone. Had 146 children (abused, nonabused, and unknown) with no ev of any *nonfracture* injury. Of these, The total number of pts with fractures only was similar for all three groups: fractures alone are not specific for abuse. But the number of fractures per pt was much greater in the abused group. As to the value of CNS injury, 189/910 pts had documented CNS injury, of which 70% were abused. “If retinal hemorrhage is present in addition to CNS injury (104 of these pts), then close to 90% of that group turned out to be abused.” And if CNS + RH + FX, (66pts) then 100% were abused. She discusses rib fractures (see below under “rib fractures”), spiral fractures, skull fractures crossing a suture line. All are nonspecific for abuse, but see the individual discussion.

Rejects TBBD.

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 are 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.”

Discusses rebleeds: “Rebleeding within a chronic subdural hematoma does occur, often after minor or no known trauma, but it is venocapillary in origin and occurs at low pressure. It does not cause cerebral edema, mass effect, or rapid clinical deterioration.”

Discusses shaken versus impact: Says that Duhaime’s conclusion “has since been refuted in the literature by several authors (citing Hadley, 1989; Alexander, 1990; Gilliland, 1996; Hymel, 1998; and Block, 1999.)” Concurs with Dr Felman that one should not presume abuse; cites the case of a 4 month old Amish baby with SDH, ICH, RH who was diagnosed as abuse by the coroner with devastating consequences until later it was found to be late-form HDN.

BRUISES

Estimation of the age of cutaneous contusions in child abuse. E.F.Wilson MD, Office of the State Medical Examiner, Lane County, Oregon. Pediatrics 1977 Nov; 60(5): 750-752. Summarizes the older forensic pathology literature on colors in tabular form. Makes some very useful cautions: a bruise may not appear at the site of impact because of fascial planes; it may not become apparent for hours or days after injury. “Since estimation of the age of a contusion is difficult and imprecise at best, the physician should state that the appearance of a contusion is *consistent* with its being so many days old rather than stating categorically that it is exactly so many days old.” 752 Note that this statement is refuted by Larry Ricci, *infra* (1996).

Patterns of skin injury in nonaccidental and accidental injury. Pascoe et al. Pediatrics 1979; 64: 245

Bruises and child abuse (letter). Edward W. Collins. Pediatrics 1981 Oct; 68(4): 614-615. Commenting on an article by Waskerwitz et al. describing hypersensitivity vasculitis as a mimic of bruises. (See above under “DDX -- Specific Disease Entities.”) Dr Collins fears that because of this report, cross examiners will question the diagnosis of bruises if a biopsy has not been done.

The objective interpretation of histopathological data: an application to the aging of ovine bruises. Forensic Sciences International 1986; 31: 225-239 “The model was able to age bruises...only as either 1-20 hours or 24-72 hours old.”

The ageing of bruises: a review and study of the colour changes with time. Langlois NEI, Gresham GA. FSI 1991; 50: 227-238. From the abstract: This work was inspired by a recent case of child abuse where the age of the bruises on the body was raised.... [The literature review] illustrates the paucity of work in this field and the absence of studies of the colour changes in bruises of human skin with time. [Our own original research] consisted of 369 photographs aged 6h to 21 days in 89 subjects aged 10 to 100 yrs. It was found that the development of a yellow colour was the most significant change ... and indicated that bruise was more than 18 hours old (in persons under 65 years old). Cited by Tim David, 2004, for the proposition that attempting to age bruises based on their color is fraught with difficulty. Discussed in *Knight’s Forensic Pathology, Third ed.* (2004) at 145 *et seq.* to the effect that “any green discolouration” was more than 18h old. Cited as concurring with Roberts, 1983. See below.

Patterned abusive bruises of the buttocks and the pinnae. Feldman KW. Pediatrics 1992; 90: 633-636. Cited by Suzanne Starling in the third edition of Giardino & Alexander’s *Child Maltreatment* (2005) for the proposition that injuries to the ears should be looked for in evaluating possible abusive head trauma.

Estimation of the age of bruising. Terence Stephenson and Yvona Bialas. Arch Dis Child 1996; 74: 53-55.

Original research: fifty accidental bruises of known age in 23 living children were photographed by a medical photographer, and shown to a single blinded observer. The observer saw red color in 40% of the bruises that were less than one week old, and yellow in 24% of the bruises that were over one day old. Aging of bruises from photographs was much less precise than textbooks imply. Cited by Tim David, 2004 for the proposition that color is fraught with difficulty.

How accurately can bruises be aged in abused children? Literature review and synthesis. Ari J. Schwartz and Lawrence R. Ricci. Pediatrics 1996 Feb; 97(2): 254-257. "visual aging of bruises remains an inexact science, despite recent composite charts that suggest otherwise." 255. "The available literature does not permit the estimation of a bruise's age with any precision based solely on color. Even for the practitioner to state, as Wilson suggests, that a particular bruise is 'consistent with' a specific age implies a level of certainty not supported by the literature." 256.

Aging of bite marks: a literature review. Dailey JC and Bowers CM. JFS 1997; 42(5): 792-795. The healing dynamics is insufficiently studied and poorly understood.

Greenes DS et al. Ann Emerg Med 1998; 32: 680-686

Bruising in non-accidental head injured children: a retrospective study of the prevalence, distribution and pathological associations in 24 cases. Atwal GS, Ruttly GN, Carter N Green MA. FSI 1998 Sep; 96(2-3): 215-230. The bruising was mainly on the face, followed by the forehead and buttocks. Limb, chest, and abdominal bruising were uncommon. 29% had no fresh bruises. RH present in 96%. Why gripping does not cause bruises (of the chest or limbs).

Postmortem extravasation of blood potentially simulating antemortem bruising. Burke MP, Olumbe AK, Opeskin K. Am J Forens Med Path 1998 Mar; 19(1): 46-49. Presents a case of a 98 y o woman who died of CHF in the hospital. Her grave was entered by vandals who inflicted blunt force injury on the face and then left the body face down. At autopsy it had considerable bruising c/w antemortem trauma.

The prevalence and distribution of bruising in babies. Carpenter RF. (Br) Arch Dis Chil 1999; 80: 363-366. Bruising is very unusual in preambulatory babies. In this study of 177 babies between 6 months and 12 months, there were only 22 who had any bruises. All bruises were on the front of the body. None were larger than 1 cm. Only one baby had as many as four bruises. Head, face, and shins were the most common sites. Bruise color did not correlate with reported bruise age.

Bruises in infants and toddlers: those who don't cruise rarely bruise. Sugar N et al. Arch Ped Adol Med 1999; 153: 399-403 Saw many innocent forehead bruises in babies who were cruising and walking. Rarely did see innocent bruises on the lower face (1-2%) in this study of bruises on nonabused infants and toddlers.

Jenny et al. JAMA 1999; 281: 621-626. Facial and scalp bruises are abnormal in the preambulatory child and clearly point to occult head trauma in the clinical setting.

A scoring system for bruise patterns: a tool for identifying abuse. Dunstan FD, Guildea ZE, Kontos K, Kemp AM, Sibert JR. Arch Dis Child 2002 May; 86(5): 330-333. Studied 133 abused and 189 nonabused ch, recording their bruises by site, maximum dimension, and shape. The differences betw ab and nonab were greatest in the head and neck area. Their scoring system was easy to use and successfully discriminated abuse from nonabuse. But they do not claim that it can replace case-by-case analysis.

When inflicted skin injuries constitute child abuse. AAP Committee on Child Abuse & Neglect. Pediatrics 2002 Sep; 110 (3): 644-645. Any injury beyond temporary reddening of the skin. Why? Two reasons: (1) Counteracting the public backlash against the criminal-justice aspects of child protection, which has resulted in legislation and other administrative movements tending to reduce intervention in child abuse situations. (2) Protecting individual patients against the fact that minor forms of abuse may lead to severe abuse unless interventions are made.

State that “[A]ny inflicted injury that last more than 24 hours constitutes significant injury (e.e. physical abuse.”

They offer the following four features of a skin-injury case that suggest child abuse:

- (a) The pattern of injuries biomechanically fits a particular mechanism.
- (b) The instrumentality indicated by the injuries could not occur through play or natural environmental interactions.

© The history provided is developmentally inappropriate.

- (d) The history is inconsistent with the injury (e.g. deep bruising from what is said to be a spanking).

Citing Helfer & Kempe, *The Battered Child*, 4th ed., 1987, Ludwig & Kornberg, and Reece for the proposition that “if minor forms of abuse are ignored, often this leads to reinjury and to more serious morbidity.

They discuss the legislative-administrative effects of the public backlash. Some states now register an incident of child abuse only if it is considered “serious.” “The stigma of being labelled a child abuser has evoked legislative sympathy in some states. Financial considerations and the legitimate fear of being overwhelmed by the number of abused children has led some child protective services systems to construct a triage system whereby a child has to be in relatively imminent danger or seriously abused before there will be a response. State supreme courts in Iowa and New Hampshire have ruled that bruising is not necessarily considered to be an injury. [See under “Medicolegal,” below.]...The result may be a tendency for some child pr serv dep which are already overloaded, to triage reports on the basis of severity of reported injury...” Arguing against such a standard on two grounds: (1) minor abuse may progress to serious abuse; (2) it discriminates against children, because adult abuse (e.g. in a domestic violence situation) would be managed more aggressively.

This policy statement was RETIRED by the Academy in October 2006. See announcement at page 405 of the February 2007 issue of *Pediatrics*.

Lack of agreement on colour description between clinicians examining childhood bruising. Munang LA, Leonard PA, Mok JYQ. *J clin Forens Med (UK)* 2002; 9: 171-174. Cited by Tim David, 2004, for the proposition that using color is fraught with difficulty.

Dating of bruises in children: an assessment of physician accuracy. Bariciak ED, Plint AC, Gaboury I et al. *Pediatrics* 2003; 112: 804-807. Cited by Tim David, 2004, for the proposition that dating by color is fraught with difficulty.

Knight's Forensic Pathology, Second Edition (2004). pp. 146-148. Bruises often become more prominent with time. There is a sequence of color changes. “The haemoglobin is broken down into compounds including haemosiderin, biliverdin, and bilirubin, which lead the color changes through a spectrum of purple to bluish brown, to greenish brown to green to yellow, before complete fading.” “Langlois and Gresham (1991) reviewed the literature on this subject, indicating the wide variation in opinion... They concluded that the most significant change was the appearance of a yellow colour (in persons less than 65 years of age), which indicated that the bruise could not be less than 18 hours old. Blue, purple, and red did not assist in dating bruises; brown was held to be a mixture of colours and was not considered as useful... It is not practicable to construct an accurate calendar of these colour changes, as was done in older textbooks, as there are too many variables for this to be reliable... Though an absolute date cannot therefore be placed upon a bruise, the following observations are legitimate:

-- If a bruise appears fresh over all its area, with no observable colour change, it is unlikely to have been inflicted more than about 2 days before death, except in old persons.

-- If the bruise has any green discoloration, it was inflicted not later than 18 hours before death.” (citing Roberts, 1983, Langlois & Gresham, 1991). (On p. 463

under “child abuse syndrome,” he repeats this statement, but with the color “yellow” substituted. –JKR)

Avoidable pitfalls when writing medical reports for court proceedings in cases of suspected child abuse. David TJ. Arch Dis Chil (GB) 2004; 89: 799-804.

Don't try to age bruises based on color. But histology has a role.

a. Clinically, color schemes are unreliable. (citing Knight's Forensic Pathology). “Attempts to age bruises based on their colour is fraught with difficulties.” (citing Langlois & Gresham, 1991; Stephenson & Bialas, 1996; Munang et al., 2002; Bariciak et al., 2003). “The time course of the appearances of bruises may vary with the location, depth, extent, and nature of a bruise. The only established fact is that the presence of a yellow colour within a bruise indicates that it is at least 18 hours old.” (citing Langlois, Stephenson.)

b. “Histopathology of bruises is worth performing in some fatal cases. Histology can rule out skin lesions such as blue naevi and can confirm bruising in those with dark skin. Histology of a bruise may enable the pathologist to say it is very fresh (no cellular reaction), recent (infiltration with neutrophils), or more than approximately two days old (presence of haemosiderin).”

Can you age bruises accurately in children? A systematic review. Maguire S, Mann MK, Sibert J, Kemp A. Arch Dis Chil (UK) 2005; 90: 187-189. Conclusion: “A bruise cannot accurately be aged from clinical assessment in vivo or on a photograph. At this point in time the practice of estimating the age of a bruise from its color has no scientific basis and should be avoided in child protection proceedings.”

Are there patterns of bruising in childhood which are diagnostic or suggestive of abuse? A systematic review. Maguire S, Mann MK, Sibert J, Kemp A. Arch Dis Chil (UK) 2005 Feb; 90(2): 182-186. This is another meta-analysis of the literature. These guys analyzed the entire english-language literature published from 1951- 2004. They found 23 studies that fit their criteria. These included 14 studies of abuse and 7 studies of accidental bruising and 2 studies of both kinds of bruising. They found this: In accidental bruising the bruises are

small
over bony prominences
on the front of the body.

The number and location of accidental bruises relates to motor development. Bruising in nonmobile infants is rare. Accidental bruising increases by age:

nonmobile infants	<1%
mobile infants	17% have bruises
walking	53% have bruises
school age	majority have bruises

Abusive bruising is away from bony prominences. Its most common locations, in order of frequency, are

face
head
neck
buttocks
trunk
arms.

Abusive bruises are large, commonly multiple, and occur in clusters. Some carry the imprint of the implement.

Non-accidental injury and the haematologist: the causes and investigation of easy bruising. Liesner R, Hann I, Khair K. (Great Ormond Street) Blood Coagulation and Fibrinolysis 2004 May; 15 Suppl 1: S41-S48. From the abstract online: It is vital that the haematologist confirm or exclude a haemostatic

disorder. Discusses methods, and how the investigation can be “highly problematic.” “For instance, some frequently used tests for the assessment of haemostasis in children are insensitive, inappropriate, or based on values derived from adult populations. Furthermore, artefact is a frequent problem, and many cases present with a negative family history of bleeding. Therefore, the role played by the haematologist in potential child abuse cases is an essential yet challenging one.”

Bruising, coagulation disorder, and physical child abuse. Sibert J. (University of Wales). Blood Coagulation and Fibrinolysis 2004 May; 15 Suppl 1: S33-S39. The interpretation of bruises can be extremely challenging for paediatricians, as the evidence base is limited. The presence of a coagulation disorder does not exclude abuse. A growing body of evidence suggests that the practice of estimating bruise age is unreliable; therefore, a key factor is the pattern of bruise **distribution**, considered in light of the child’s history and level of development. We use a scoring system.

Are there patterns of bruising in childhood which are diagnostic or suggestive of abuse? A systematic review. Maguire S, Mann MK, Sibert J, Kemp A. Arch Dis Chil (UK) 2005; 90: 182-186.

Can you review bruises accurately in children? Maguire S, Mann MK, Sibert J, Kemp A. Arch Dis chil (UK) 2005; 90: 187-189. See Dr David’s 2005 article, Child abuse and paediatrics, next below.

Child abuse and paediatrics. David TJ. J R Soc Med 2005; 98: 229-231. This review of the confused state of diagnosis in child abuse and the resulting effects on the profession. Includes, re bruises, the following conclusion: “Attempts to gauge the age of bruises from their colour are fraught with difficulties. The time course of the appearances may vary with the location, depth, extent, and natures of a bruise. The only established fact is that the presence of a yellow colour within a bruise indicates that it is at least 18 hours old.” (citing the two articles just above.)

How good is the evidence available in child protection? Sibert JR, Maguire SA, Kemp AM. BMJ Jan 30 2007. Refuting a lead article by David Chadwick on 22 July 2006 stating that the published evidence base for child protection is robust. On the contrary, say these authors, who are part of an evidence-based medicine review team. One of the few things they find to be well establish is the fact that you can’t age bruises. See below under “Unclassifiable.”

FRACTURES

See also “DDX” and “DDX -- Specific diseases,” above; Birth Injury, Cervical Spine, TBBD, and OI, and Head Injury -- Accidental vs. Inflicted. See also “Classic articles” and see also Caffey’s papers under “Shaken.” See also the scurvy-vitamin C articles under “Vaccine SBS.”

Etude medico-legale sur les services et mauvais traitements exerces sur des enfants. Tardieu A. (Auguste Ambroise Tardieu) Ann Hyg Publ Med Leg 1860; 13: 361-398. See above under “Classic articles.”

Kleinman PK. *Diagnostic Imaging of Child Abuse, 2d ed.* St. Louis: Mosby, 1998. (Cited as “Kl.”)

Harris VJ, Lorand MA, Fitzpatrick JJ, Soter DK, *Radiographic Atlas of Child Abuse: A Case Studies Approach.* New York: Igaku-Shoin, 1996. Shows films from numerous actual clinical cases of different kinds of physical abuse.

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

Chapter 7, *Skeletal manifestations of child abuse.* In: Reece RM and Ludwig S., eds., *Child Abuse: Medical Diagnosis and Management, 2d ed.*, 2001.

Chapter 13, *Radiographic signs of skeletal trauma*. In: Ludwig S and Kornberg AE, *Child Abuse: A Medical Reference*, 1992.

In General

Multiple fractures associated with subdural hematoma in infancy. Lis EF, Frauenberger GS. *Pediatrics* 1950; 6: 890-892

Roentgen manifestations of unrecognized skeletal trauma in infants. Silverman F. *Am J Roentgenol* 1953; 69: 413-426

Hematome sousdural du nourisson associe a des fractures des membres. Marie A. *Semaine Hospital* 1954; 30: 1757

Significance of skeletal lesions in infants resembling those of traumatic origin. Woolley PV jr, Evans WA. *JAMA* 1955; 158: 539-543.

Patterns of injury in the battered child syndrome. O'Neal JA, Meachum WF, Griffin PP, Sawyers JL. *J Trauma* 1973; 13: 332-339

Unsuspected costovertebral fractures demonstrated by bone scanning in the child abuse syndrome. Smith FW, Gilday DL, Ash JM, Green MD. *Pediatric Radiology* 1980; 10: 103-106

Chapter 9: Birth and Pediatric Fractures. Robert B. Salter, MD. In: *Fracture Treatment and Healing*. Edited by R. Bruce Heppenstall, MD, FACS. Philadelphia: W. B. Saunders, 1980.

Patterns of fracture in accidental and non-accidental injury in children: a comparative study. Worlock P, Stower M, Barbor P. *BMJ* [cited by Knight as "Br Med J Clin Res Ed"] 1986; 293: 100-102. Quoted in Chapter 22 of *Knight's Forensic Pathology, Third Edition* (2004) as having compared fractures in 35 abused and 286 control children and found that the abused were all under 5 years old, while 85% of the accidentals were over 5 years old. Also that the abused infants were more likely to have multiple fractures. Also that rib fractures were almost confined to the abused "when major chest trauma was excluded." Also that spiral fractures of the humeral shaft were common.

Analysis of 429 fractures in 189 battered children. King J, Diefendorf D, Aphorpe KJ, Negrete VF, Carlson M. *J Ped Orthop* 1988; 8: 585-569.

Fractures in premature infants. Amir J et al. *J Ped Orthop* 1988; 8: 41-44. Fracs in 1.2% of all prematures, 2.1% of VLBW. Predisposition to fracturing ends at birth age 4 mos. See Dahlenburg, *infra*.

Are preterm infants at risk for subsequent fractures? Dahlenburg SL, Bishop NJ, Lucas A. *Arch Dis Chil* 1989; 64: 1384-1385

Radiologic contributions to the investigation and prosecution of fatal infant abuse. Kleinman PK, Blackburne BD, Marks SC Jr, Karellas A, Belanger PL. *NE J Med* 1989; 320: 507-511.

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

Long-bone fractures in young children: distinguishing accidental injuries from child abuse. Thomas SA, Rosenfield NS, Leventhal JM, Markowitz RI. *Pediatrics* 1991 Sep; 88(3): 471-476. [See also Fractures in young children ..., by the same authors, 1993, below]. There is lack of knowledge of a specific mechanism for many injuries. We focused on the humerus and femur. We reviewed medical records of

all children younger than 3 treated for fx: excluded patients with any disorder likely to cause bone weakening. Found 215 children treated for 252 fractures of all types. Of these, 39 had fx of the humerus or femur. Of these 39, 18 were under 1 year of age. Of the 39 humerus or femur patients, 20 were found to be abuse (51%).

Conclusions:

1. Nonsupracondylar fractures of the humerus are strongly associated with abuse.
2. Supracondylar fractures of the humerus are typical of a fall.
3. Femur fractures under one year of age are likely due to abuse.
4. Children old enough to run can fall and fracture their femurs.

Humerus: n=14

Age	0-11m	12-23m	24-35m	Tot
Abuse	7	2	2	11
Accident	1	0	2	3

All the abusive humeral fractures were either midshaft or distal, or proximal CML.

All the accidental humeral fractures were distal supracondylar. All resulted from falling on their elbows.

All nonsupracondylar fractures were abusive.

Femur: n=25

Abuse	9
Acc	14
Undet	2

60% of all femur fractures under one year were abusive, but in the older groups acc was more common (only 20% were abusive.)

There was no distinctive radiological pattern for abusive versus accidental femur fractures.

Children who are old enough to run can trip and fracture a femur, contrary to what has been said.

Table 1 lists "criteria used by the radiologists to distinguish abuse from accidents." (I have reworded some for clarity)

Definite abuse:

- The history is not consistent with the amount of force involved
- Delayed reporting of serious trauma
- Multiple fractures present, not explained by the history
- Healing fractures present, not explained by the history

Likely abuse

- The history is not consistent with the severity of the injury

Questionable abuse

- Suspicious fracture, check history for explanation
- Suspicious delay in reporting

Unknown cause

- Fracture not suspicious but the story is incomplete

Questionable accident

- Fracture not suspicious but history lacks details

Likely accident

- Fracture not suspicious, history consistent with the fracture

Definite accident

- Fracture not suspicious, story consistent and thorough, witnesses

Fractures in young children: distinguishing child abuse from unintentional injuries. Leventhal JM, Thomas SA, Rosenfield NS, Markowitz RI. Am J Dis Child 1993; 147(1): 87-92. (Yale) Retrospective review of 215 Yale ER admissions under 3 years of age with a fracture over a five year period 1979-1983 (253 fractures) were retrospectively rated for likelihood of abuse by two clinicians and two pediatric radiologists. They created a seven-level scale of likelihood (now known as the Thomas scale, see Banaskiewicz et al, 2002 under "Fractures -- In General"):

Definite abuse	+ skel survey or eyewitn
Likely abuse	severe unexpl inj, alleged mech not expl, +delayed pres or changing hx or prev CPS hx
Suspicious for abuse	hx not suff to expl and either changing hx, delayed pres or prev hx CPS
Unknown	not enough information to reach a consensus
Likely accident	hx consistent w fx, no suspicious fxx
Definite accident	multiple eyewitnesses

Found:

Abuse	24%
Unknown	8%
Accident	67%

The fractures found likely to be abusive were:

1. Humeral fractures -- midshaft or metaphyseal
2. Radius, ulna, tib, fib in ch under 1 year
3. Any fx where the caregiver did not report any accidental event or where the history was of a minor fall but the fracture was more severe than expected

See also Thomas, 1991, above.

In Kleinman's 1995 article (excluding skull fxx) on a population of 31 infants of average age 3 mo, out of 165 inflicted fxx, 51% were rib fxx, 39% were CML's (classic metaphyseal lesion), only 5% long bone shaft fxx.

Inflicted skeletal injury: a postmortem radiologic-histopathologic study in 31 infants. Kleinman PK, Marks SC Jr, Richmond JM, Blackbourne BD. AJR 1995; 165: 647-650.

Radiographic Atlas of Child Abuse: a case studies approach. Harris VJ, Lorand MA, Fitzpatrick JJ, Soter DK. New York: Igaku-Shoin, 1996. ISBN 0-89640-258-4. Gives radiographs and clinical correlation in 72 cases from all areas of the body. In other words, 72 short annotated case reports. The strongest feature of this approach is that it allows us to actually see the films that were used as the basis of diagnosis in real cases, so we know what these lesions actually look like. See below under "Shaken" and "Head injury -- fall vs. inflicted."

The coverage is comprehensive, including both bony and soft tissue injuries from all body regions. Here are the items covered: Shaken babies in various forms, including alleged falls. Shearing tears. Aging of SDH's. EDH. Limb fractures. Rib fractures. Skull fractures. Spinal fractures. Scapular fractures. Femur fractures. Metaphyseal fractures. Bruises, Osteogenesis imperfecta (gives radiographs of fractures, bowing, Wormian bones, rib deformities). Duodenal perf, duodenal hematoma, traumatic pancreatitis, marasmus, live birth (air in the stomach), Repeated falls. Cigarette burns, retinal hemorrhages, Gives twenty "red flags of child abuse," which are worth quoting. [I have edited the wording a bit. Note also the similarity to elder abuse risk factors in some of these, particularly no's 6,7,8,9,12,13,19. -JKR]. The individual case reports mention those red flags that were present in the cases presented.

1. The history involves an unlikely mechanism of trauma, such as a fall from a bed or a fall down stairs
2. No history of trauma
3. Changing history
4. Delay in seeking medical attention
5. Injuries attributed to another child
6. History of domestic violence
7. History of abuse as a child in the parent
8. Drug or alcohol abuse in the caregiver
9. Parental isolation
10. History of previous unusual injuries or hospitalizations
11. History of unexplained child deaths in the family
12. Lack of bonding
13. Developmental or behavior problems in the child
14. Skin injuries in a location that could not have been self-inflicted
15. Multiple injuries in different stages of healing
16. Fractures in an infant
17. Multiple fractures of different ages
18. Fractures specific for abuse
19. Nutritional deprivation or emaciation
20. Lack of any reasonable medical explanation for the total clinical picture

But the authors caution that "The presence of one or several of these red flags alone cannot determine whether child abuse or neglect has occurred..."

The book brings out many diagnostic points, including the correlation of CT and MRI, the use of T1 and T2 MRI, the fact that MRI is more sensitive than CT for detecting small SDH's, aging of SDH's by MRI (the change from oxyhemoglobin to methemoglobin), discussion of fall histories, reversal sign, As to the reversal sign, see cases 11, 13, 15, 16 18,

"The reversal sign is recognizable because of the striking difference in the density of the cerebral cortex and white matter; which become blacker, and the increase in density of the thalamus, brain stem, and cerebellum, which becomes white. this is a sequela of severe brain injury and has an ominous prognosis..." shown on pp. 64 and 73.

One disadvantage of this book is that the images were made on older equipment and are not as detailed as modern scanners.

Our thanks to Donald C. Boger, MD, for donating this book to the Department of Coroner for our use.

Skeletal evidence for child abuse: a physical anthropological perspective. Walker PL, Cook DC, Lambert PM. JFS 1997 May; 42(2): 196-207

A general comment by Kleinman, from Child Abuse Quarterly, v, 4, Oct. 1998:

"Prior studies have shown that a meticulously performed skeletal survey employing high-detail imaging systems will yield fractures which are not suspected on clinical grounds or detectable on conventional imaging systems. Specimen radiography and histologic studies will reveal additional injuries and may provide further information regarding the age and extent of the pathology. In the current climate where the concept of the "shaken baby syndrome" has come under increasing scrutiny, the presence of skeletal injuries carrying a high specificity for abuse may provide compelling evidence of inflicted injury."

Brogdon B.G., Child abuse. In: *Forensic Radiology*. B.G. Brogdon, M.D. Boca Raton: CRC Press, 1998. ISBN 0-8493-8105-3. Also includes radiology of visceral injuries. Includes a photograph of the abstract of Tardieu's 1860 paper (see above) and a portrait of Dr. John Caffey (1895-1978), described as "the father of pediatric radiology." As to fractures, Brogdon discusses:

The metaphyseal lesion, first described by Caffey, "is virtually pathognomonic." Ordinarily seen in children who don't yet walk. Described by Caffey as sometimes causing "traumatic bowing of the ends of the diaphyses due to metaphyseal infraction" (?infarction). Brogdon's fig. 15-8 demonstrates this

“bowing” lesion in the tibia of what appears to be a toddler, showing marked enlargement of the distal tibial metaphysis surrounding a chronic metaphyseal fracture.

Periosteal new bone (several radiographs shown, including some of Caffey’s original ones, where Caffey referred to it as the “involucrum.”)

Spiral fractures, “highly suggestive of abuse, particularly in the nonambulatory child.” (P. 294) See also p. 40, fig. 3-6, where Dr. Brogdon places side by side two spiral fractures of the distal tibia, one in a toddler classed as a typical toddler’s fracture, and one in a nonambulatory infant, where this fracture is “impossible to acquire naturally in the course of infantile movement. Rather, this fracture was caused by a twisting force or torsion at the hands of an adult caregiver.” (cf. Boal in “Controversial aspects of child abuse: a roundtable discussion,” *Pediatric Radiology* 2001; 31: 760-774 at 768, noting, “Beware the spiral or blique fracture, particularly if it is solitary. For some, particularly the Child Protective Services, this fracture is synonymous with abuse... However, spiral fractures do occur in a child that is ambulatory, and we now know that they may occur accidentally in younger infants...”

Transverse long bone fractures (p. 294). High specificity for child abuse in the nonambulatory child.

Dislocations (p. 295).

Rib fractures (p. 295-296). Discussion follows Kleinman. “Rib fractures are practically never seen after resuscitative efforts in children.”

Hand (p. 296). Quite rare and highly suspicious.

Clavicle (p. 298). Commonly perinatal, rare in abuse, except that fx of the lateral end can be seen in shaking.

Scapula (p. 298). Highly suspicious. In the acromion, “care must be taken to differentiate a true fracture from an ununited apophysis,” giving radiographs.

Rare fractures (p. 299). High specificity for abuse: sternum, spinous processes, vertebral subluxation from slamming (gives an example, fig. 15-19).

DDX: OI, syph, rickets, Caffey’s disease, leukemia, PGE therapy for congenital heart disease, Menkes’ neuroblastoma, nmets, hypervitaminosis A, scurvy, osteomyelitis, methotrexate, myelodysplastic syndrome, congenital indifference to pain, metaphyseal osteochondrodysplasia, Dilantin, birth injury, normal variant.

Orthopedic aspects of child abuse. Kocher MS. (Children’s Hospital of Boston). *J Am Acad Orthop Surg* 2000 Jan; 8(1): 10-20. About 1,200 deaths per year. Fractures are the second most common presentation after skin lesions. There is no pathognomonic fracture pattern for abuse; all factors must be considered in every case, including age, injury pattern, history of mechanism, psychosocial factors. Suggestive injury patterns include: (a) certain metaphyseal lesions in young children, multiple fractures in various stages of healing, posterior rib fractures, and long bone fractures in children less than 2 years old. The DDX includes OI and metabolic bone disease, as well as true accident.

Development and duration of radiographic signs of bone healing in children. Islam O, Soboleski D, Symons S, Davidson LK, Ashworth MA, Babyn P. *AJR* 2000 Jul; 175(1): 75-78. (have) This is accidental fractures of the forearm in 141 children ranging in age from 1 to 17 years by a single-blind retrospective study where the actual time of injury was known. Objective: “Few articles report the evaluation of pediatric fracture healing and dating ... We established a timetable for expected radiographic changes visible during bone healing in otherwise healthy children.” Conclusion: “A wide variation exists in the appearance and duration of the radiographic signs of bone healing. Marginal sclerosis should be an

expected radiographic sign of normal bone healing.” In the early phase of healing, visibility was limited by the cast. Things observed:

Blunting of the fracture margins at 1 week	60%
Periosteal reaction before 2 weeks	0%
Periosteal reaction seen at 4 weeks	100%
Fracture gap widening at 2 weeks	9%
Fracture gap widening at 4-6 weeks	56%
Fracture gap widening at 7 weeks	0%
Marginal sclerosis at 2 weeks	6%
Marginal sclerosis peaking at 4-6 weeks	85%
Marginal sclerosis disappearing by 11 weeks	100%
Calcific callus seen at 2 weeks	15%
Calcific callus seen at 4 weeks	100%
Callus density less than cortex at 8-10 weeks	50%
Callus density greater than or equal to cortex after 10 weeks	90%
Partial bridging first noted at 3 weeks	yes
Partial bridging at 8 weeks	50%
Complete bridging (obliteration of the fracture defect) after 10 weeks	40%
Remodelling after 8 weeks	95%

The authors state that the most comprehensive review of radiographic dating is by O'Connor & Cohen in the first addition of Kleinman; it says that a lack of data has left dating up to the individual radiologist's experience. They note that "the time ranges found in our study differ somewhat from those described in the literature." [I would note that their patients are a lot older than Kleinman's material, which could account for the considerably longer healing times they found. The authors agree with this. --JKR] They give a bar graph correlating Kleinman's histologic stages with their radiographic changes:

Histologic stage + Kl dating	Their stage	Time
Stage I stage of induction 0-3 wks	fracture gap widening	3 - 7 weeks
Stage II stage of inflammation 0-3 wks	“ “ “	“ “
Stage III soft callus 2-6 wks	marginal sclerosis	3-11 wks
	Periosteal reac	2-14 wks
	Calcific callus	2-14 wks
Stage IV hard callus 2-13 wks	callus denser than ctx	5-14 wks
	Bridging	5-14 wks
	Periost incorporation	7-14 wks
Stage V remod 3mos +	remod	4 wks +

The authors note, concerning abused children, that most abused children are much younger than their material, and this limits their study for that purpose. Not only that, but abusive fractures are not immobilized and may be malnourished (Vit D, Vit C, Vit A, Vit B) or chronic illness. The reviewer for the *Quarterly* (Jan 2001), David F. Merten MD, makes a good statement concerning the factors that limit the applicability of this study to non-accidental injuries of infants: "...repair of fractures may be more rapid in

infants, while repetitive injury and lack of mobilization typically associated with non-accidental injuries also alter radiographic patterns of bone healing.”

Diagnostic imaging of child abuse. AAP, Section on Radiology. Pediatrics 2000; 105: 1345-1348. Cited by the Section on Child Abuse & Neglect (2001a) for the proposition that healing does not become radiologically apparent for 7 – 10 days.

Imaging of child abuse. Nimkin K and Kleinman PK. Radiol Clin N Amer 2001 Jul; 39(4): 843-864. (have) A comprehensive review with pictures. Actual case illustrations of a differential diagnosis including:

- Rickets
- Bone disease of prematurity
- OI
- Osteomyelitis
- Syph
- Accident (including a CML of proximal humerus in a 6 month old)
- Obstetrical injury (oblique femur fx shown)
- Sickle cell anemia (dactylitis)
- Spondylometaphyseal dysplasia (looks like a corner fracture) (See book)
- Normal variants, particularly of the metaphysis, the acromion, the ulna

States that Caffey originally described the CML in 1957. (Br J Radiol 30: 225).

In infants the most common pattern is rib fxx, skull fxx, and metaphyseal injuries. After 1 year, long bone fxx are the most common.

“Skeletal injuries can be grouped according to their relative specificity for abuse:”

High specificity

- CML
- Rib fxx, especially posterior
- Scapular
- Spinous process
- Sternal

Moderate specificity

- Multiple fxx, especially if bilateral
- Fxx of different ages
- Epiphyseal separations
- Vertebral body fx and sublux
- Digits
- Complex skull

Low specificity

- SPNBF
- Clavicle
- Long bone shaft (the most common abusive fx in older chil)
- Linear skull

[For a slightly different table which should also be consulted, see table 7.3 in Reece & Ludwig, second edition, 2001, at 153, below]

In non-infants, the most common abusive fractures, in order:

1. Femur
2. Humerus
3. Tibia
4. Forearm

In infants, the most common abusive fractures:

Femur
Humerus other than supracondylar
Toddler's fracture if not walking or cruising

Discusses rib fractures: see under ribs, below. Discusses various specific bones. Discusses natural diseases that are in the DDX (see above under "DDX" and below under "OI.")

As to accidental fractures, states that "Determination as to whether the biomechanical forces occurring with the claimed accidental event are consistent with the observed fracture is one of the most challenging aspects of this complex field. ...Stairway injuries, injuries in infant walkers, and falls in the arms of a caretaker are important sources of accidental long-bone fractures." 857

As to SPNBF and tibias, Caffey's disease is a rare disorder of widespread SPNBF. As a normal variant, less than 2 mm of SPNBF in the tibia and femur can be seen between 1 month and 6 months. Watch out for normal variants of the metaphysis. See paragraph on p. 467 of *Knight's Forensic Pathology, Third Edition* (2004) as to tibial periosteal thickening due to handling during breech delivery.

Child Abuse: Medical Diagnosis and Management, Second Edition. Reece RM and Ludwig S, eds. Philadelphia: Lipincott Williams & Wilkins, 2001, table 7.3, p. 153:

Specific for child abuse

Metaphyseal (if under 2 years of age)
Rib
Sternum
Scapula
Clavicle if medial end or lateral end
Spine if anterior compression or spinous process fx

Highly suggestive

Multiple, bilateral, symmetric
Repetitive/ different ages
Hands and feet
Complex skull fracture
Associated with visceral injury
Associated with intracranial injury

Nonspecific

Long bone diaphyseal
Midclavicular
Linear skull

As to the pathognomonic fractures, the authors comment that "The mechanical forces required to produce these fractures are not generated by simple accidental falls and normal handling in an otherwise healthy infant and child."

Compare to the classification given by Nimkin & Kleinman, 2001, above.

Accidents and resulting injuries in premobile infants: data from the ALSPAC study. Warrington SA et al. Arch Dis Chil 2001; 85: 104-107. 3357 falls in 2554 infants.

Subtle fractures in kids: how not to miss them. Leonard E. Swischuk, MD (Galveston). Appl Radiol 2002 Nov; 31(11): 15-19. Subtle fractures of the long bones: be sure to examine the soft tissues for edema and the fatpads for joint fluid and edema. Gives illustrative radiographs. This short article is high-priority reading.

There are five basic types of fractures in infants and children:

- Epiphyseal-metaphyseal (Salter-Harris)
- Buckle (torus)
- Hairline
- Plastic bending
- Avulsion

Epiphyseal-metaph: shows widening of the epiphyseal line

Buckle result from axial loading of an extremity (e.g. falling on outstr arm): show a slight buckling inward or outward of the cortex. If severe forces, it can coexist with a Salter-Harris II. One commonly missed buckle fracture is that of the base of the first metatarsal called a bunkbed fracture. Upper tibia can be considered another form of toddler's fracture.

Hairline: toddler's fracture is a typical hairline fx. A spiral fx fo the tibia, often subtle. The one he shows is not that subtle. Also a transverse hairline of the proximal ulna from a direct blow, a spiral hairline of the proximal ulna from torsion, and very subtle one of the proximal ulna from hyperextension and twisting.

Plastic bending occurs usually in the forearm.

Avulsion: elbow including condylar; quite subtle, just a sliver off.

Spontaneous fractures in the differential diagnosis of fractures in children. Torwalt CR, Balachandra AT, Youngson C, DeNanassy J. JFS 2002 Nov; 47(6): 1340-1344. (Winnipeg) Case report of a four year old spastic who was in institutional care and was found to have multiple limb fractures in various stages of healing, which the authors determined to be due to spasticity combined with osteopenia. The terms often used for these include spontaneous fractures, stress fractures, pathologic fractures, handling fractures, and insufficiency fractures. They are fairly common in bedridden patients. A 1966 study of CP patients showed that these resulted from normal handling, getting a limb caught in crib, also falls and blows. Often multiple in the lower limbs. Thought to result from osteoporosis, lack of protective sensation, vitamin C deficiency, vitamin D deficiency, and a side-effect of anticonvulsant medication, which interferes with bone metabolism. The biomechanics include a long, fragile lever arm. The article does not mention anything about fractures of the axial skeleton.

Fractures in children younger than age 1 year: importance of collaboration with child protection services. Banaszkiwicz PA, Scotland TR, Myerscough EJ. J Ped Orthop 2002; 22: 740-744. (UK) Retrospective chart review of 74 children with fractures in the ER. 46 skull, 28 long bone. In many of them, NAI was not considered or worked up. The authors felt that 46% were at least suspicious for abuse. See Kunen et al., next below.

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

Underdiagnosis of child abuse in emergency departments. Kunen S, Hume P, Perret JN, Mandry CV, Patterson TR. Acad Emerg Med 2003 May; 10(5): 546. It is likely that more than 75% of all child abuse cases presenting to EDs are being missed, and many of these missed cases involve fractures and dislocations in infants. In this chart review of 7,827 infants under two years old seen in the ER, 127 had

fractures or dislocations that were *not* diagnosed as abuse in the ER. We randomly selected 50 of these charts and rated them for likelihood of abuse: found that 30% of them were highly suspicious for abuse. See next above.

Injuries when children reportedly fall from a bed or couch. Hennrikus WL, Shaw BA, Gerardi JA. Clin Orthop 2003 Feb; 407: 148-151. Had 115 pts seen for orthopedic injuries following such a history. 113 fractures, 2 impalements. Overall, found that 95% of the injuries were explained by the history and 5% were not. But of the four children under 1 year of age, 50% were abusive. Conclusion: Orthopedic injuries attributed to a child falling from a bed or couch are usually accidental **unless the child is younger than 1 year.**

Child Abuse: Radiologic-Pathologic Correlation. From the Archives of the AFIP, pp 811-845. RadioGraphics 2003; 23: 811-845. Gael J. Lonergan, LTC USAF MC, Andrew M. Baker, MD, Mitchel K. Morey, MD, Steven c. Boos, LTC USAF MC. Covering bony and soft-tissue injuries.

Fractures in biliary atresia misinterpreted as child abuse. DeRusso PA, Spevak MR, Schwarz KB. Pediatrics 2003 Jul; 112(1 Pt 1): 185-188. Biliary atresia is associated with metabolic bone disease. There are numerous reports of rickets, osteopenia, and fractures.

Fractures in young children: are physicians in the emergency department and orthopedic clinics adequately screening for child abuse? Oral R, Blum K, Johnson CF. Pediatric Emergency Care 2003; 19: 148-153. Chart review of 653 patients ranging in age from 5 weeks to 3 years (average = 18 months). Numerous patients had more than one fracture:

one fracture	653
two fractures	141
three fractures	31
four fractures	14

Dr Reece summarizes the findings in the *Quarterly* for October 2003, stating that the authors divided the patients into four categories based on the chart review, noting that 39% of the charts contained inadequate information to make a categorization:

agreed with the original dx of accidental in	63% of the patients
agreed with the original dx of inflicted in	13% of the patients
possibly missed inflicted in	23% of the patients

Complementary use of radiological skeletal survey and bone scintigraphy in detection of bony injuries in suspected child abuse. Mandelstamm SA, Cook D, Fitzgerald M, Ditchfield MR. Arch Dis Chil 2003; 88: 387-389. Argues that you should use both modalities during the initial evaluation. Retrospectively reviewed the written reports of the skeletal surveys and bone scans (not the scans themselves) on 30 abused children with 124 fractures. Found that if you leave aside rib fractures, x-ray missed 25% and bone scan missed 56%. 20% of the patients had a normal skeletal survey but an abnormal bone scan. 10% had normal bone scans but abnormal skeletal surveys. X-ray missed 50% of solitary rib fractures. Bone scans missed 65% of CML's. Therefore you should do both. Paul Kleinman, reviewing the article for the Spring 2004 issue of the *Quarterly*, says that a two-week followup skeletal survey is a cheaper alternative, but agrees that initially doing both modalities is reasonable rather than discharging the child back into an unsafe home.

Postmortem radiography in the evaluation of unexpected death in children less than 2 years of age whose death is suspicious for fatal abuse. Joint Statement of the Society for Pediatric Radiology and the NAME. Pediatric Radiology 2004; 34: 675-677. Says that a minimum standard would be certified radiographic technicians performing the long bones at least. That's a minimum. Says that local pediatric radiologists have a civic duty.

Evaluating long bone fractures in children: a biomechanical approach with illustrative cases. Pierce MC, Bertocci GE, Vogeley E, Moreland MS. Child Abuse & Neglect 2004; 28: 505-524. According to Betty

Spivack's review in the *Quarterly* for Autumn 2004, this analyzes the mechanisms of five types of long bone fractures:

spiral fractures
buckle fractures
transverse fractures
oblique fractures
CML's

Spiral fractures, which are due to twisting, are often abuse but can come from trip & fall while running

Buckle fractures, which are axial-compressive, occur at the metaphysis, are frequently accidental but can be abusive in children under nine months

Transverse fractures, due to bending loads, can be caused by direct impact or blow to the extremity, result from high-energy impacts such as fall from a height or traffic accident.

Oblique fractures, due to combined loads

CML's, due to shearing injury through the metaphysis -- shaking, yanking.

Betty adds that "The authors identify several features that indicate a high-energy event. These include transverse, displaced, comminuted, and femoral neck fractures. such fractures are highly unlikely to be associated with a simple fall or other lowenergy event."

Chapter 22 "Fatal Child Abuse" in: Saukko P and Knight B, *Knight's Forensic Pathology, Third Edition*. New York: Oxford University Press, 2004. See chapter summary under "Unclassifiable," below.

Diagnostic errors by radiology residents in interpreting pediatric radiographs in an emergency setting. Halsted MJ, Kumar H, Pacquin JJ et al. *Pediatric Radiology* 2004; 34: 331-336. From Dr Merten's review in the *Quarterly* for Autumn 2004, they reviewed 23,273 resident dictations, found that 77 cases contained errors (total of 80 errors). For an error rate of 80/ 23,273 or 0.3%. 90% of the errors were missed fractures, mostly buckle, Salter II, avulsion, and transevers fractures.

Skull radiograph interpretation of children younger than two years: how good are pediatric emergency physicians? Chung S, Schamban N, Wypij D, Cleveland R, Schutzman SA. *Ann Emerg Med* 2004; 43: 718-722. According to Dr Strouse's review in the *Quarterly* for Autumn 2004, the miss rate for skull fractures was 20%. He writes that "Pediatric skull films are challenging to interpret...." Therefore, CT should be done if there are any signs of intracranial pathology. He says that it is controversial whether skull plain films are indicated in the emergency setting. They are important as part of a skeletal survey. In this particular study, the participating ER physicians had a SENS of 76% and a SPEC of 84% for skull fxx in infants. Thus, you should consider the expertise and experience of the physician who will be interpreting the films before you order skull films in this setting.

Skeletal injuries associated with sexual abuse. Johnson K, Chapman S, Hall CM. (UK) *Pediatric Radiology* 2004 Aug; 34(8): 620-623. From the abstract: Sexual abuse is often associated with physical abuse. Usually soft tissue injuries, but fractures occur in 5% of sexually abused children. We report three sexually abused children who had fractures:

A 3 year old girl with extensive soft-tissue injuries to the arms, legs, and perineum sustained fractures of both pubic rami and the right sacroiliac joint.

A 5 month old girl with an introital tear had an undisplaced left femoral shaft fracture.

A 5 year old girl presented with an acute abdomen and pneumoperitoneum due to a ruptured

rectum due to sexual abuse. She had old healed fractures of both pubic rami with disruption of the symphysis pubis.

Non-accidental injury. Chapman S. (Birmingham) Imaging 2004; 16(2): 161-173. A monograph on the radiology of physical abuse. The author provides a box summary:

1. Requires meticulous detail
2. Normal variants are pitfalls
3. Correlation with the history is mandatory, bc many fxx are not diagnostic of abuse
4. Many fxx are **asymptomatic**, including rib fxx and CML
5. The DDX includes accident, birth injury, prematurity, dysplasia, rickets

Describes what a skeletal survey should consist of. Notes that followup study may increase the number of fractures and assist with dating.

Normal variants include the fact that SPNBF on the shaft of long bones of very young infants is a normal variant out to four months, due to radial growth. (gives an example) Commonly is unilateral. If it (the band of mineralization) exceeds 2mm in thickness, further investigation is warranted. Also, it should be smooth and regular, and blend in with the cortex near the ends of the bone.

Healing and dating. Recopies Kleinman's table of peak times:

resolution of ST changes	4-10	days
SPNBF appears	10-14	"
loss of fracture line	14-21	"
hard callus appears (lamellar bone)	21-42	"

Cites Matthew et al., BMJ 1998 for the proposition that the majority of fractures are not associated with bruising. Notes in particular that this is true of rib fractures. "Surprisingly ... rib fractures are not associated with chest wall bruising, although fingertip bruising (thumb marks anteriorly over the upper part of the chest, finger marks posteriorly) may be an accompanying clinical sign." (p. 164)

CPR. Rarely causes rib fractures, citing Feldman & Brewer, 1984; Spevak, Kleinman et al., JAMA 1994, who found no rib fxx in CPR infants. Betz & Liebhardt, 1994 found rib fxx in 1/94. Bush, Cohle 1996 found 1/211. But note that CPR fractures are **not posterior**, because there is no levering on a flat table or bed.

Rib fractures and birth injury. "Exceptionally rare." Gives an example showing three fresh displaced posterior rib fxx in a macrosomic newborn. Citing Barry, Hocking, Arch Dis Chil 1993.

Long bone fractures. Spiral fx is a result of twisting. Oblique fx is a result of levering. Transverse fx may be from a direct impact. Greenstick or buckle fx is from compression, such as a fall. **The correlation between the type of fracture and the likelihood of abuse is poor.**

Unusual fractures raise the suspicion of abuse. For example; spine, scapula, acromion, sternum, pelvis, fingers & toes in a preambulatory child.

Differential diagnosis:

Birth trauma. Usually clavicle, femur, humerus. **Metaphyseal fractures can happen.**

Accidental injury. This explanation is tested by comparing the fracture morphology to the following considerations:

mechanism
dating

developmental stage versus the history given
commonness of the injury at a given age

As to mechanism, he cites Joffe & Ludwig, 1988, to the effect that, aside from falling in the arms of a caregiver or in a walker, a stair fall is one moderate-height fall followed by multiple low-height falls. Therefore, "when multiple, severe, truncal, or proximal extremity injuries are noted in patient who reportedly fell down stairs, a different mechanism of injury should be suspected." "However, falling down stairs in the arms of a carer or while constrained in a baby walker is far more dangerous." (p. 170)

Osteogenesis imperfecta. Can be a problem if the family history is poor. Type IV is most often a problem because of the variable severity of its bone fragility and the fact that the sclerae are white. Wormian bones are only significant when they are greater than 6mm in size, more than 10 in number, and arranged in a mosaic. Note that metaphyseal fractures are "quite rare" in OI.

Other bone diseases. Osteopenia of prematurity. Rickets. Neurogenic bone disease (cerebral palsy, myelomeningocele). Congenital syph. Osteomyelitis. Copper deficiency. Rare skeletal dysplasias. Pre-term infants are NOT at increased risk for fractures.

My thanks to Tim David, MD for providing this article.

Identifying non-accidental injury in children presenting to A&E departments: an overview of the literature. Sanders T, Cobley C. Accident and Emergency Nursing (UK) 2005; 13: 130-136. "Bone fractures may not manifest outward signs and can be easily missed by the treating physician, especially in A&E where clinicians may not have the same expertise in identifying signs of abuse as in paediatrics." p. 133.

Subperiosteal new bone formation. (see also Scurvy, under "DDX -- Specific diseases," above)

Dating of healing rib fractures in fatal child abuse. Zumwalt RE and Fanizza-Orphanos AM. Adv Pathol 1990; 3: 193-205

Physiologic SPNBF occurs in young infants as a symmetrical radiographic finding, mostly on the legs. SPNBF can also occur in infectious, accidental, and metabolic conditions (Kleinman ch. 11).

The mechanism is torsion or shearing stress, most often from shaking. Usually does not involve direct impact to the limb or even grasping of the limb. (Kl. p.12)

Imaging of child abuse. Nimkin K and Kleinman PK. Radiol Clin N Amer 2001 Jul; 39(4): 843-864 at 857. SPNBF can be caused by syph, Caffey's disease, sickle cell anemia (including dactylitis). As to SPNBF and tibias, Caffey's disease is a rare disorder of widespread SPNBF. As a normal variant, less than 2 mm of SPNBF in the tibia and femur can be seen between 1 month and 6 months. Watch out for normal variants of the metaphysis.

Utility of followup skeletal surveys in suspected child physical abuse evaluations. Zimmerman S, Makaroff K, Care M, Thomas A, Shapiro R. Child Abuse & Neglect 2005; 29(10): 1075-1083. Followup skeletal surveys at two weeks are an accepted part of the workup. This study validated the technique, yielding additional information in 46% of patients. In three patients the followup study changed the diagnosis by either ruling out child abuse (one case) or ruling it in (two cases). In three other cases, the followup study refuted earlier tentative radiological findings.

The risk of child abuse in infants and toddlers with lower extremity injuries. Coffey C, Haley K, Hayes J, Groner JJ. J Pediatr Surg 2005; 40: 120-123. According to Dr Greeley's review in the Spring 2006 issue of the *Quarterly*, had n = 1,252 patients with lower extremity injuries.

abuse not abuse

under 18 mos	(# 66)	44 (67%)	22
over 18 mos	(# 1,186)	16 (1%)	1,170

In the under 18 mos abuse group, the most common fractures were femur (22) and tibia (14). In the under-18 mos group, 74% of all the chil admitted with lower extremity fractures were abuse cases. See also below under "Femur."

Sensitivity of autopsy and radiological examination in detecting bone fractures in an animal model: implications for the assessment of fatal child physical abuse. Cattaneo C, Marinelli E, DiGiancamillo A, DiGiancamillo M, Travetti O, Vigano L, Poppa P, Porta D, Gentilomo A, Grandi M. FSI 2006; 164: 131-137. These Italian researchers took four dead piglets and beat them unmercifully. They then performed four types of examinations of their bodies with respect to detecting the resulting postmortem fractures:

1. Traditional radiography
2. Whole-body CT scanning
3. Autopsy
4. Complete defleshing with osteological analysis of the skeleton

The purpose was to find out how sensitive the various examination methods are in the different regions of the body. Found that if you use defleshing with osteological analysis as the gold standard, the sensitivities by region were:

Cranium

Traditional radiography	35%
CT	100%
Autopsy	31%

Rib cage

Traditional radiography	47%
CT	34%
Autopsy	65%

How good is the evidence available in child protection? Sibert JR, Maguire SA, Kemp AM. BMJ Jan 30 2007. Refuting a lead article by David Chadwick on 22 July 2006 stating that the published evidence base for child protection is robust. On the contrary, say these authors, who are part of an evidence-based medicine review team. One of the few things they find to be well established is the fact that you can only age fractures within broad limits. See below under "Unclassifiable."

Fracture rate in children with cerebral palsy. Stevenson RD, Conaway M, Barrington JW et al. Pediatric Rehabilitation 2006; 9: 396-403. Found a 45 fracture rate, correlated with obesity and gastrostomy and with history of a previous fracture.

Evaluating infants and young children with multiple fractures. Carole Jenny and the AAP Committee on Child Abuse & Neglect. Pediatrics 2006 Sep; 118(3): 1299-1303. The differential diagnosis of fractures is:

1. OI
2. Prematurity
3. Rickets
4. Osteomyelitis (!)
5. Copper deficiency
6. Immobility
7. Rare diseases

Menckes syndrome, scurvy, osteopetrosis, hypophosphatasia, congenital syph, leukemia, vitamin A toxicity, metabolic disease, kidney conditions that cause calcium wasting, prolonged adm of prostaglandins, glucocorticoids, methotrexate

8. Child abuse

Discusses how to make the dx of child abuse in a (living) patient with fractures, including coordinating with other specialties and keeping an open mind.

Unexplained fractures in infancy: looking for fragile bones. Bishop N, Sprigg A, Dalton A. Arch Dis Chil (UK) 2007; 92: 251-256. This is a nice literature review on pathologic fractures of infants. Four risk factors (other than prematurity and primary bone disease):

Cholestatic jaundice
TPN
BPD
Lasix

Lack of exercise/ physical activity is clearly a factor in fracturing in these sick infants. Intravenous feeding provides a poor supply of mineral. Lasix promotes calcium loss in the urine. As far as prematurity itself, it ends as a risk factor by age 2, because by that time the child has been on an oral diet long enough to backfill the skeletal envelope. Has discussions of the following diseases:

osteogenesis imperfecta
rickets
Cole-Carpenter syndrome
Bruck syndrome
familial osteoporosis
rare bone diseases

panostotic fibrous dysplasia/ McCune-Albright syndrome
osteopetrosis
infantile severe hypophosphatasia
congenital insensitivity to pain
congenital rickets
CMV

Now as to these:

Osteogenesis imperfecta is caused in 90% of cases by mutations in the genes for type I collagen. There are now seven types; types V, VI, and VII or "rhizomelic" OI are the ones that do not involve type I collagen. Types II and III (the bad ones) are readily diagnosed on clinical and radiographic grounds. Types I and IV can be difficult to recognize.

Cole-Carpenter syndrome is a rare congenital disorder of unknown cause with proptosis, hydrocephalus, and early fracturing.

Bruck syndrome is congenital joint contractures. [From the description, it sounds a lot like arthrogryposis. -JKR]

Familial osteoporosis is a few kindreds with a mutation of a gene that regulates bone formation. If they're homozygous, they get the osteoporosis pseudoglioma syndrome, of which less than 50 cases have been reported worldwide. They get vertebral crush fractures, eye disease, metaphyseal fractures (!)

Pain. Rib fractures are splinted by the surrounding structures, and so may be less painful (or nonpainful?). Here is what he says: "All fractures are painful, whether in children with normal bones or in those with

bone disorders. Fracture-related pain is likely to recur when the affected site is disturbed in any way, and will probably be more intense and persist longer when the affected area is not splinted by surrounding structures. Thus, rib fractures in particular may go unsuspected by both parents and clinical staff, but fractures of the midshaft of a long bone will be associated with protective disuse."

He then discusses the following topics: plain-film radiography, osteopenia, dating, metaphyseal fractures, biochemical markers, postmortem testing, molecular diagnosis, and when should genetic testing be requested?

As to metaphyseal fractures and OI, says, "Fractures can be found in almost any site or combination of sites in OI. However, metaphyseal fractures are rare in both OI and premature infants, and multiple fractures in infants with OI are usually accompanied by some degree of bony deformity."

As to postmortem testing, says you can take bone tissue for specialized analysis, or you can take DNA samples and skin samples. Discusses the technical details of testing these.

Classic Metaphyseal Lesion (CML)

As per Kleinman, 2d ed: Always involves severe indirect force. Highly specific for abuse. Most common in the proximal humerus. (Kl. 22) Radiographic appearance can be strikingly altered by radiologic technique. Also by the age of the fx. The break is across the proximal portion of the metaphyseal primary spongiosa, (distal to) the area of the chondrocyte columns.

Epiphyseal/physeal separations:

These are mostly accidental. (Kl. 23). They are more severe force, possibly direct force. You can have both physeal and metaphyseal lesion in one injury, as in the Salter-Harris types II, IV.

Some traumatic lesions in growing bones other than fractures and dislocations: clinical and radiological features. Caffey J. Br J Radiol 1957; 30: 225-238. The original description of bucket-handle or corner fractures.

Atlas of the Battered Child Syndrome. Cameron JM and Rae LJ. Edinburgh: Churchill Livingstone, 1975. Cited by Knight's Forensic Pathology, Third Edition (2004) as possibly the origin of the view that metaphyseal fragments are virtually pathognomonic of child abuse.

Analysis of 429 fractures in 189 battered children. King J. J Ped Orth 1988; 8: 585-589. Fractures of the femur, humerus, and skull were the most common in battered children. Only 16% were metaphyseal. Rib fractures tended to be posterior and multiple.

Long-bone fractures in young children: distinguishing accidental injuries from child abuse. Thomas SA., Rosenfield NS, Leventhal JM, Markowitz RI. Pediatrics 1991; 88(3): 471-476. 215 children under 3 years of age. 39 children under 3 with humerus or femur fxx. Of 14 humerus fxx, 11 were considered to be abuse and 3 accidental (these were supracondylar elbow fxx.) Of 25 femur fxx, 9 were abuse, 14 acc, 2 unk. In infants under 1 yr, 60% of femur fxx were abusive. Femur fxx are often accidental in running children. 3 femurs were caused by being dropped.

Fractures in young children. Leventhal JM. Am J Dis Child 1993; 147: 87-92. Presents 215 children under 3 y o with 253 fractures. 24% overall were abuse. Abuse was more likely if (A) the caregivers did not give a history of injury; (B) the child was under 1 year and it was an arm or leg fracture; (C) humerus other than supracondylar. Abuse by age groups: under 1 = 39%; under 2 = 14%; under 3 = 8%. Note that femur fractures were seen from running and exercise in toddlers.

Child abuse or osteogenesis imperfecta: how can we tell? Gahagan S and Rimsza ME.

Pediatrics 1991; 88: 987-992. Used collagen type I biosynthesis studies in 3 infants suspected for abuse. All three were OI. (But note that according to later articles, collagen studies should rarely be needed; the diagnosis is made by clinical, historical, anatomic, and radiologic means).

(Syphilitic periostitis versus fracture) Horodniceanu M, Volovitz B, Nitzan M. *Pediatr Radiol* 1978 Dec; 7(4): 232-234 Abstract: congenital syph is re-emerging. A case of fx of the olecranon and facial bone lesions is described; the condition had to be differentiated from the battered child syndrome. Lilien LD, Harris VJ, Pildes RS. *Pediatr Radiol* 1977 Oct; 6(3): 183-185. See also under DDX, above.

Non-accidental injury: review of the radiology. Rao P, Carty H (UK). *Clin Radiol* 1999 Jan; 54(1): 11-24

Controversial aspects of child abuse: a roundtable discussion. Society for Pediatric Radiology. *Pediatric Radiology* 2001 Nov; 31(11): 760-774. Dr. Boal makes some comments on the specificity of fractures.

1. "Not long ago, skull fractures crossing the suture line, involving more than one bone, were thought to have a high specificity for abuse. We now know that this is frequently not the case."
2. Spiral fractures can happen accidentally in ambulatory children and occasionally in infants (citing the famous 5-month videotaped rollover spiral fx of the arm).
3. Presents data on rib fractures. See below under "rib fxx."
4. Comments on TBBD. See below under TBBD.

Complementary use of radiological skeletal survey and bone scintigraphy in detection of bony injuries in suspected child abuse. Mandelstamm SA, Cook D, Fitzgerald M, Ditchfield MR. *Arch Dis chil* 2003; 88: 387-389. According to Dr Kleinman's review of this in the Spring 2004 *Quarterly*, bone scans miss 65% of CML's. See above under "In general."

Whole body STIR MR imaging in suspected child abuse: an alternative to skeletal survey radiography? Stranzinger E, Kellenberger CJ, Braunschweig S et al. (Zurich) *Eur J Radiol* 2007 May-June (in press). Whole-body STIR-MR imaging permits eval of the entire skel and all viscera with a single examination while avoiding ionizing radiation. We report a 2 month-old suspected victim of child abuse in whom whole-body STIR MR imaging revealed multiple rib fractures sugg of child abuse that were only partially recognized by conventional radiography.

Skull

Anatomy of the shaken baby syndrome. Lancon JA, Haines DE, Parent AD. *New Anatomist* 1998; 253: 13-18. Cited by Cattaneo et al. in their 2006 art on experimental piglet fracture imaging for the proposition that "The infant skull consists of extremely thin bones that lack the rigidity and strength of adult ones. In addition, the bones of the infant skull are separated by loose cranial sutures. Consequently, the immature skull is fairly resistant to fracturing except in the setting of significant trauma." I have never heard of this journal.

See also Kleinman's chapter.

Femur

Intertrochanteric fractures: see case 8 in Woolley, *JAMA* 1955; 158: 539. Cases 1 and 2 in Silverman, *AJR* 1953; 69: 413. Ogden, *J Pediatr Orthop* 1984; 4: 285. Blockey, *JBJS* 1969; 51: 106.

The significance of femoral fractures in children. Anderson WA. Ann Emerg Med 1982; 11: 174-177. Presents 117 femoral fracs. 24 were abuse (20% overall). However, in the under-1-yr age group, 15/18 (83%) abuse. In the under-2-yr group 19/24 (79%).

Causative factors responsible for femoral fractures in infants. Gross R. J Ped Orth 1983; 3: 341-343. Presents 74 cases under 5 yrs. Overall 34 were abuse. In the under-1-yr group, 17/26 = 65%. There were 5 traffics, one other trauma, 1 pathologic fracture.

Fractured femur in infancy. Beals, J Ped Orth 1983; 3: 583-586. Discusses 80 cases of fractured femurs in children under 4. 30% were considered to be due to abuse. 46% accidental. 12% pathologic fractures. 8% other trauma. In the birth-to-six months age group, 65% were abusive.

Fractures of the femur in childhood. Wellington P. Injury 1987; 18: 103-104. Presents 142 cases under age 5. Overall 26% were abuse, but 39% in the under 1 year age group.

Quasi-static and dynamic bending stress of the pediatric femur for producing a femoral fracture. Ztschr f Rechtsmed. (ger) 1989; 102: 535-544. Summary provided by Dr. Feldman: 28 cadavers age 1 day to 6 yrs. 18 had static transverse loading applied at mid-femur (loading rate 50mm bend per minute). Fracturing force was found to be 470 N in a 6 day old infant, increasing to 2920 N in a 6 y o. One newborn required 2720 N and one 15 mo req 5700 N. Fractures occurred at from 16 to 60 mm of bending. 10 children between 2 mo and 27 mos were given sustained dynamic transverse loading using a falling weight or a horizontal impactor. Only one transverse fx was caused. Forces in this series ranged from 320 to 660 N with falling weights or up to 2370 N with the horizontal impactor. These forces would suggest that falls from adults' arms or from changing tables are inadequate to cause these fractures.

Undiagnosed abuse in children younger than 3 years with femoral fracture. Dalton HJ, Slovis T, Helfer R, et al. AJDC 1990 Aug; 144: 875-878. Evaluated 138 femur fxx. 31 % were abusive, 22% accidental, 8% bone disease, 10% uncertain etiology, of which 35% later became abuse. Dr. Feldman points out that once you exclude the obvious automobile trauma and obvious pathologic fractures, 44% of these cases were abuse.

Role of intentional abuse in children 1 to 5 years old with isolated femoral shaft fractures Blakemore LC, Loder RT, Hensinger RN.. J Pediatric Orthop 1996 Sep; 16(5): 585-588. We reviewed 42 children with isolated femoral shaft fxx. Only one fx was documented to have been inflicted. The likelihood of intentional injury to the femoral shaft appears to be low.

A regional approach to the classic metaphyseal lesion in abused infants: the distal femur. Kleinman PK and Marks SC jr. Am J Roentg 1998; 170: 43-47. Fourth in a series of four papers. The previous ones described the radiol and hist f of CML in prox tib, dist tib, prox hum. Here 15 CML's of the distal femur in 11 infants 1-5 mos. The distal femur is known to be a common site of abusive injury. These particular CML's are c/w shaking. These papers called "superb articles" by reviewer Deborah Ablin MD.

Pediatric femoral shaft fracture. Hakala BE and Blanco JS. 2000 Feb. Published on Medscape at <http://www.medscape.com/Medscape/OrthoSportsMed/journal/2000> (have). Cite as Medscape Orthopaedics & Sports Medicine 4(1), 2000. "As many as 30% of femoral shaft fractures in children younger than 4 years may be the result of child abuse, and the most common cause of femur fractures in the nonambulatory infant is nonaccidental trauma." (Citing Leventhal, 1993 and Thomas, 1991, *supra*, under "In General."). "Factors suggestive of child abuse include bruises, burns, multiple fractures in various stages of healing, and late presentation. Osteogenesis imperfecta, stress fractures, and pathologic fractures may also be encountered." This article gives a detailed discussion of the management of femoral shaft fractures both abusive and accidental in both the very young and the older age groups. The younger patients are generally managed with casting (spica cast), while the older children are managed with internal fixation requiring general anesthesia and rehabilitation. Discusses differential leg length.

Femoral shaft fractures in children: incidence, mechanisms and sociodemographic risk factors. In: program and abstracts of the 67th annual meeting of the American Academy of Orthopaedic Surgeons; March 15-19, 2000; Orlando, FL. Scientific Exhibition Paper No. 224. A copyrighted summary by Colin Moseley MD CM appears on Medscape, from which the following is summarized: Showed a bimodal incidence – small children caused by falls, and teenagers caused by automobile accidents. A separate peak for teenage black males caused by gunshots. No cases of child abuse are represented in the database, which was the Maryland Health Services Cost Review Commission showing 1,485 cases of femoral shaft fracture in patients younger than 18. Dr. Moseley comments that the absence of child abuse in the youngest age group probably underrepresents this etiology.

Femur shaft fractures in toddlers and small children: rarely from child abuse. Schwend RM, Werth C, Johnston A. J Pediatr Orthop 2000; 20: 475-481. Retrospective review of the hospital records of 139 children. Note that most were not infants. 24 were infants; of these, 10 were due to abuse. (40%) Three toddlers were abused. Conclude that CPS need not be involved in the management of femur fractures over one year of age unless there is (a) other indication of abuse, such as bruises, (b) a history of previous fractures, or (c) history not consistent with the fracture.

Accidental and nonaccidental femur fractures in children. Scherl SA, Miller L, Lively N et al. Clin Orthop 2000; 376: 96-105.

Computer simulation of stair falls to investigate scenarios in child abuse. Bertocci GE, Pierce MC, Deemer E, Aguel F. Arch Ped Adol Med 2001 Sep; 155: 1008-1014. Children's Hospital of Pittsburgh. The purpose was to model stair falls as a possible mechanism of femur fractures in ambulatory children, by modelling a 3 year old child. Begins with the statement that "Unfortunately, it is often difficult to determine the validity of caregiver-stated scenarios, since so little is known regarding pediatric biomechanics and injury risk associated with these relatively common falls. A better understanding of the influence that specific fall-environment factors have on injury biomechanics in children is needed." "Although a limited number of studies describing relationships between the biophysics of a particular fall and associated injuries in children exist, additional studies are greatly needed to advance the forensic science to detect child abuse." Reviews Lyons & Oates, 1993; Warner & Demling on free falls, 1986; Greenberg on falls from heights, 1978; Lallier on falls from heights in children, 1999; Joffe & Ludwig, 1998; several other articles on femur fractures. Their model considered number of steps, stair friction, stair elasticity, and slope. Concluded that stair characteristics such as surface material and slope can play an important role in the risk of femur fracture. Therefore, you need to know these characteristics in order to determine the likelihood that a given stair fall could or could not have been the mechanism of a femur fracture. Consult Kleinman's textbook 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 (?) The Exersaucer is a nonrolling type of walker that spins instead of walking; see below under "Walkers."

Analysis of the cause, classification, and associated injuries of 166 consecutive pediatric pelvic fractures. J Pediatr Orthop 2001 Jul-Aug; 21(4): 446-450. Had 136 children.

Doubt, difficulties, and practicalities in the diagnosis of non-accidental injury -- a personal view. Kenney JJ. Imaging 2001 Dec; 13(4): 295-301.

Racial differences in the evaluation of pediatric fractures for physical abuse. Lane WG, Rubin DM, Monteith R, Christian CW. JAMA 2002 Oct; 288(13): 1603-1609.

Supracondylar femoral fracture in an infant. Swischuk LE. Pediatric Emergency Care 2003; 19: 104-107. Case report of a toddler or infant with a distal femur fracture attributed to a fall from a baby swing, who was also found to have rib fractures and retinal hemorrhages.

Skeletal injuries associated with sexual abuse. Johnson K, Chapman S, Hall CM. (UK) Pediatric Radiology 2004 Aug; 34(8): 620-623. See above under “In general.”

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 cannot adequately present this article by a summary format; it has to be studied in detail, in which it is highly instructive. Used quantitative scoring to retrospectively evaluate the plausibility of the history in 29 femur fractures in children aged under 5.

The authors created four categories of information to construct an Injury Plausibility Score, assigning a score of 0 to 3 for each category, so that the most plausible score would be 0 and the least plausible score would be 12. The four categories were:

- I. Fall components per history: richness of detail, fall dynamics, and final position
- II. Type of fracture
- III. Time of rescue (immediate to severely delayed)
- IV. Other injuries (from none to many)

The four suspicious elements which decreased plausibility were:

- I. Lack of detail in the mechanical history or changing history or history of no trauma
- II. CML or high-energy fracture
- III. Delayed rescue
- IV. Additional injuries (the more the worse)

Elements supporting plausibility included eyewitnessed fall, biomechanically detailed fall history including landing position, and biomechanical compatibility between the fracture type and the alleged fall mechanism; in other words, “if the fracture load type was not accounted for by the history and the fracture type was classified as a high-risk fracture for abusive trauma (CML or a high-energy fracture, meaning open or comminuted). The “plausible” fractures for solitary child falls included spiral fractures, buckle fractures, and oblique fractures. Yes, that’s right: transverse fractures take 10X more energy to cause than do these other types of fractures. In three of the four suspicious cases in this article, it was a transverse fracture. Gives a chart of types of fractures, entitled, “Criteria for determining biomechanical and fracture type compatibility (biomechanical match)”:

<u>Biomech conditions</u>	<u>Fracture types</u>	<u>History examples</u>
Torsional loading	spiral/ long oblique	twisting as child slips
Bending load	transverse/ short oblique	perpendicular impact, such as caught under a falling caretaker
Compressive loading	Buckle/ impaction	knee impact
Tension or shear loading	CML	pulling or yanking leg
High-energy event	open or comminuted	auto vs. pedestrian

They created an “injury plausibility score” comprised of the above items, ranging from 0 for a completely plausible history to 12 for the most implausible history. The innocent cases were in the 0 to 3 range, while the four probable or obvious abuse cases were 6 to 12.

Walkers are a known risk for severe injury. One of these cases was a walker-stairs fall, and it received an injury plausibility score of 3.

Childhood femur fractures, associated injuries, and sociodemographic risk factors: a population-based study. Rewers A, Hedegaard H, Lezotte D et al. Pediatrics 2005; 115: e543-552. Studied all femur fractures in the Colorado Pediatric Trauma Registry for four years.

The risk of child abuse in infants and toddlers with lower extremity injuries. Coffey C, Haley K, Hayes J, Groner JJ. J Pediatr Surg 2005; 40: 120-123. See above under "In general" for the findings that in the under-18 mos age group, 67% of all lower extremity fractures were abusive. The reviewer in the Spring 2006 issue of the *Quarterly*, Dr Greeley, lists the following references:

Anderson, Signif of femoral fxx in chil. Ann Emerg Med 1982; 11: 4
 Skellern et al., Non-acc fxx in chil. J Paed Ch Health 2000; 36: 590
 Banaszkiwicz et al., Fxx in chil under 1. J Ped Orthop 2002; 22: 740-744
 Hoskote et al., Fxx in inf: 1 in 4 is nonacc. Child Ab Review 2003; 12: 384
 Rewers et al., Child femur fxx. Pediatrics 2005; 115: e543

Epidemiology and mechanism of femur fractures in children. Loder Rt, O'Donnell PW, Feinberg JR. J Pediatr Orthop 2006; 26: 561-566. Had 1076 femur fractures in the under-two age group. 35% falls, 33% MVA, 15% abuse. Cerebral palsy and OI were well represented in the overall total children group of 9960 chil. (181 and 135, respectively).

Ribs

For rib fractures as a birth injury (it happens), see above under "Birth injury."

Multiple fractures associated with subdural hematoma in infancy. Lis EF, Frauenberger GS, . Pediatrics 1950; 6: 890-892. Cited by Cattaneo et al. in their 2006 paper on experimental imaging of fractures in dead piglets, along with Smith et al, 1980, Woolley et al., 1955, Marie, 1954, and Silverman, 1953 for the proposition that the importance of rib fractures to the diagnosis of child abuse has been appreciated for a long time.

Roentgen manifestations of unrecognized skeletal trauma in infants. Silverman F. Am J Roentgenol 1953; 69: 413-426. Cited by Cattaneo et al. in their 2006 paper on experimental imaging of fractures in dead piglets, along with Smith et al, 1980, Woolley et al., 1955, Marie, 1954 for the proposition that the importance of rib fractures to the diagnosis of child abuse has been appreciated for a long time.

Hematome sousdural du nourisson associe a des fractures des membres. Marie A. Semaine Hospitaliere 1954; 30: 1757. Cited by Cattaneo et al. in their 2006 paper on experimental imaging of fractures in dead piglets, along with Smith et al, 1980, Woolley et al., 1955, and Silverman, 1953 for the proposition that the importance of rib fractures to the diagnosis of child abuse has been appreciated for a long time.

Significance of skeletal lesions in infants resembling those of traumatic origin. Woolley PV jr, Evans WA. JAMA 1955; 158: 539-543. Cited by Cattaneo et al. in their 2006 paper on experimental imaging of fractures in dead piglets, along with Smith et al, 1980, Marie, 1954, and Silverman, 1953 for the proposition that the importance of rib fractures to the diagnosis of child abuse has been appreciated for a long time.

Cameron JM and Rae L, The radiological diagnosis. In: JM Cameron and L Rae, eds., Atlas of the Battered Child Syndrome. London: Churchill Livingstone, 1975., pp. 20-50. Cited by Cattaneo et al., 2006, for the proposition that "rib fractures are lesions which are highly specific for infant abuse."

Rib fractures in infancy. Thomas PS. Ann Radiol 1977; 20: 115-122. Cited by Cattaneo et al., 2006, for the proposition that "rib fractures are lesions which are highly specific for infant abuse." But it is also cited

by Dr Allen R. DeJong, Director of the Children At Risk Evaluation Program at the Alfred I. DuPont Hospital For Children (SIGCA-MD-L, 2007) as reporting a case of rib fractures as a birth injury.

Unsuspected costovertebral fractures demonstrated by bone scanning in the child abuse syndrome. Smith FW, Gilday DL, Ash JM, Green MD. Pediatric Radiology 1980; 10: 103-106. Cited by Cattaneo et al. in their 2006 paper on experimental imaging of fractures in dead piglets, along with Lis et al. , 1950, Woolley et al., 1955, Marie, 1954, and Silverman, 1953 for the proposition that the importance of rib fractures to the diagnosis of child abuse has been appreciated for a long time.

Fractures in the first year of life: a diagnostic dilemma? McClelland CQ, Heiple KG. Am J Dis Child 1982; 136: 26. Rib fxx from CPPD in the NICU.

The abused child: a radiological reappraisal. Merten D, Radkowski M. Radiology 1983; 146: 377-383. Cited by Dr Reece (2002) for the proposition that the vast majority of abusive rib fxx are seen in infants under 2. Also for the proposition that more than 80% of abusive rib fxx are posterior. Also Kleinman, Marks, 1995. Gives a table showing the frequency of different fractures at different ages.

Child abuse, CPR, and rib fractures. Feldman KW and Brewer DK. Pediatrics 1984; 73: 339-342. Showing very few rib fxx in a large retrospective series of in-hospital CPR. 61 abusive rib fxx. 18 were anterior. 19 were lateral. 24 were posterior. 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.

See Zumwalt, above, under "In general.

Rib fractures as a cause of immediate neonatal tachypnoea. Landman L, Homberg R, Sirota L, Dulizky. Eur J Pediatr 1986; 144: 487-488. Reference kindly provided by Dr. Allen R. DeJong, director of the Children At Risk Evaluation Program at the Alfred I. DuPont Hospital for Children.

Radiologic and histopathologic correlates of posterior rib fractures in abused infants: an alternate mechanism of injury. Kleinman PK. Pediatric Radiology 1987; 17: 83-91. Positing the lever-arm mechanism involving the transverse process.

Factors affecting visualization of posterior rib fractures in abused infants. Kleinman PK, Marks SC, Adams VI, Blackbourne BD. AJR 1988; 150(3): 635-638.

Injuries to children younger than one year of age. Rivara et al. Pediatrics 1988; 81: 93-97. 191 injuries studied: 146 accidental and 45 abusive. Accidental injuries were overwhelmingly nonsevere; abusive injuries were commonly severe and multiple injuries. There were 8 cases of abusive rib fxx, 0 cases of accidental rib fxx

Neonatal rib fracture: birth trauma or non-accidental injury? Rizzolo PJ, Coleman PR. J Fam Prac 1989; 29: 561-563.

Rib fractures in children: a marker of severe trauma. Garcia VF, Gotschall CS, Eichelberger MR, Bowman LM. J Trauma 1990 Jun; 30(6): 695-700 (have). Retrospective review of the charts of 2,080 children aged 0-14 years admitted to a trauma center. 33 children had rib fractures. Of these 14 died (42% mortality.) Children with rib fractures were significantly more severely injured. The risk of mortality increases with the number of ribs fractured. Rib fractures are a rare injury in childhood. Dr Reece (SBS Quarterly, Fall 2002) cites this art for the proposition that major forces are required to fracture ribs.

Fractures of the rib head in abused infants. Kleinman PK, Marks SC, Spevack Mr, Richmond JM. Radiology 1992 Oct; 185(1): 119-123. From the abstract: "Fractures of the posterior ribs are well-recognized sequelae of infant abuse. Previous reports have indicated a predilection for fracture near the costotransverse process. This study expands the spectrum to include fractures involving the rib head. The radiologic and histologic features are described, and the mechanism of injury of this lesion is examined. In situ and specimen radiography, followed by histopathologic examination, was performed in

78 ribs removed from seven abused infants who died with posterior rib fractures. Computed tomography of the intact posterior thorax was performed in two of these infants. Fifty posterior rib fractures were identified; 29 involved the rib head. Frontal radiography was insensitive in identifying these fractures, clearly revealing injury only when periosteal reaction was present (four of 29 cases.) Axial specimen radiography delineated the fractures in all cases. In the two infants studied, CT depicted five of 19 fractures visible only with axial specimen radiography. The morphologic features of these fractures further support the concept that most fractures in abused infants occur by means of indirect forces and are consistent with anteroposterior manual thoracic compression during assaults." Notes by JKR: 1. Obviously there were no controls. 2. In these cases, the finding of rib head fractures was merely cumulative; there were already obvious rib arc fractures. 3. It would be interesting to know whether these rib head fractures can exist in the absence of rib arc fractures. 4. This work suggests that we should be doing specimen axial radiography routinely in child abuse autopsies, and that it is better than other methods.

Infant rib fracture -- birth trauma or non-accidental injury. (letter). Barry PW, Hocking MD. Arch Dis Chil 1993

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. Cited 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.

Fractures of the first rib in child abuse. Strouse PJ, Owings CL. Radiology 1995 Dec; 197(3): 763-765. Retrospectively they identified 35 children under 2 seen in the radiology department with rib fractures over a two-year period. Only four children had a first-rib fracture. Of these, one was OI, three were abuse. Concluded that first-rib fxx were likely to be abusive.

Rib fractures in 31 abused infants: postmortem radiologic-histopathologic study. Kleinman PK, Marks SC jr, Nimkin K, Rayder SM, Kessler SC. Radiology 1996; 200: 807-810. Posterior rib fxx as char of abuse. Cited by Reece, 2002, for the proposition that more than 80% of abusive rib fxx are posterior. Also Merten, Radkowski, 1983.

Pediatric injuries from CPR. Bush CM, Jones SJ, Cohle SD, Johnson H. Ann Emerg Med 1996 Jul; 28(1): 40-44. 211 living children who had CPR: one had CCJ fractures.

Skeletal evidence for child abuse: a physical anthropological perspective. Walker PL, Cook DC, Lambert PM. JFS 1997; 42(2): 196-207. Posterior rib fxx as char of abuse

Radiological case of the month: rib fractures produced by birth trauma. Arch Ped Adol Med 1997 Sep; 151(9): 947-948

[Posterior rib fxx in LGA infants as birth injury] Hartmann. Arch Path Lab Med 1997 Sep; 151: 947. Two cases of multiple posterior rib fxx detected at birth following vag deliv of LGA.

Quantitative analysis of trabecular morphogenesis in the human costochondral junction during the postnatal period in normal subjects. Fassalari NL, Moore AJ, Byers S, Byard RW. Anat Rec 1997 May; 248(1): 1-12. Morphometry used to measure the rate at which the trabeculae thickened and the interchondrocyte distance decreased in the primary and secondary spongiosa during postnatal life. This relates to the study of achondroplastic and other dwarfisms.

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. This article cited by Nimkin & Kleinman, 2001, for the proposition that “Fractures near the CCJ are particularly hard to see and are probably more common than reviews suggest.” Cited by Reece, 2002, for the proposition that “CCJ fxx are rare ... difficult to visualize [until] after callus formation has occurred.”

Characteristics of child abuse by anteroposterior manual compression versus CPR: case reports. Gunther WM, Symes SA, Berryman HE. Am J Forens Med Path 2000 Jan; 21(1): 5-10. Presents three autopsies of infants (7 mos, 7 mos, and 13 mos) who had multiple healed and unhealed anterior and lateral rib fxx due to inflicted chest compression. Includes several refractures.

Case 1 had multiple fresh and healing lateral rib fxx but no CCJ or CVA fxx. There were refractures. “Some of the fresh fractures had broken through healing callus from old fractures.” Case died of abdominal trauma with mesenteric scarring also. Also had hemosiderin macrophages in the lungs. Fingertip bruises on the anterior rib cage area bilaterally convinced the authors that this child was gripped and compressed from the rear. No confession.

Case 2 survived; it was the twin brother of case 1, found at hospital to have multiple lateral rib fxx.

Case 3 had fresh lateral fxx. These were fxx of the inner cortex, limited to the inner curvature. Died of abdominal trauma. Similar anterior fingertip marks to case 1. Confession to squeezing the infant.

Discussion compares these cases to Bush and Steve Cohle’s series of CPR infants in which there was one CCJ fx. Emphasizes the finding of breaks of the inner curvature cortex only in some ribs, and posits that sustained compression produces this failure mode, while the brief-intermittent compression of CPR cannot produce it.

Cause and clinical characteristics of rib fractures in infants. (electronic article.) Bulloch B, Schubert CJ, Brophy PD, Johnson N, Reed MH, Shapiro RA. Pediatrics 2000 Apr; 105(4): e48. From Children’s Hospital of Cincinnati and Children’s Hospital of Winnipeg. These pediatric radiologists and pediatricians from two referral centers retrospectively identified all infants under 12 months with rib fractures, amounting to 39 infants. They state that this is the largest study ever of rib fractures in infants. (As distinguished from studies including large numbers of older children.) They found the causes to be:

Definitely accidental	1 pt (auto collision w severe inj)
Possibly accidental (they diagnose accidental)*	2 pts*
Birth injury	1 pt (40 wks shoulder dystocia)
VLBW	1 pt (birth wt 645 gm)
OI	1 pt
Rickets	1 pt (malnourished Native American)
Total nonabuse	7 pts
Abuse	32 pts

- The two infants considered to be accidental, abuse was not conclusively ruled out but after CPS investigation the parents’ histories, albeit unwitnessed, were felt to be plausible. Case 1 mother brought her infant (age not given) to private physician’s office with a history that the 5-year-old sibling had fallen on the infant while rollerblading and that the infant’s chest felt “crackly” and he was very fussy. No x-rays obtained at that time. One month later she brought the infant to the ER for wheezing. CXR showed five healing rib fxx (L midposterior 4,5,6,7,8). (The authors differentiate “posterior” from “midposterior,” with the latter term apparently meaning somewhat more lateral than the former term.) Case 2 the father brought infant immediately to the hospital after an accident in which he stated that he fell down stairs while carrying the infant. One acute fx anterior L 7th rib. Lac spleen, transverse fx L femur, 3 linear skull fxx (R occ, R temp, L par-occ). Fxx apparently all acute.

The fractures in the study were located as follows:

Nonabuse	Posterior	71% of the pts
Abuse	(119 fractures in 32 pts, or 3.7 fxx per pt)	
	Posterior	35% of the fxx
	Midposterior	25% of the fxx
	Lateral	34% of the fxx
	Anterior	6%

Two of their 39 infants received CPR. Both were abused infants. One of these (a death) had healing rib fxx. The other had only bruises. Neither had any acute rib fxx. No rib fxx could be attributed to CPR.

By statistical criteria ($P=.05$) the location of fractures did not differ between the abused and nonabused groups. All cases were reviewed by senior specialists at the respective teaching hospitals. The authors acknowledge limitations of the study due to its retrospective design and short followup interval.

The authors review the literature on infantile rib fractures from all causes. Conclusion: rib fractures in infants are usually caused by abuse.

Posterior rib fractures in neonates and infants due to CPR. Patrick E. Lantz, MD, Associate Professor of Pathology, Wake Forest Univ Sch of Med, Winston-Salem, NC. Presented at the 1999 AAFS Conference. (have abstract). Four cases of infants, three of whom had spent their entire lives in the hospital and the forth child abuse was ruled out. They had posterior rib fxx, acute except in one case of a remodelling fx of posterior L 6th rib in an 8 mo BPD w nutritional osteopenia. Fxx as fol:

- Neonate w AVM, acute fxx l posterior 3-6
- 2 wk w aortic atresia, emergency L thoracotomy, L 1-6
- 4 mo trisomy 21 VSD CHF L 1-6
- 8 mo BPD remod fx L 6

Rib fractures in infants: red alert! The clinical features, investigations, and child protection outcomes. Cadzow SP, Armstrong KL. J Paediatr Child Health (Aus) 2000; 36: 322-326. Report 18 pts of which 15 were abuse and 3 MVA. The abuse group had 92 fxx -- 39 postr, 12 lat, 41 antr. The MVA group had 9 fxx. The average age of the abuse group was 4 mos, the MVA group was 1 year. Paul Kleinman comments (Child Abuse Quarterly Apr 2001) that rib fxx in infancy are strong indicators of physical abuse. Cited by Reece (2002) for the proposition that cutaneous bruises were not seen overlying abusive posterior rib fxx.

Restrictive asphyxia: a recognizable form of fatal child abuse. Boos SC. Child Abuse & Neglect 2000; 24: 1503-1507. A father confessed to squeezing his infant's chest multiple occasions to make him stop crying. Also jamming his legs up onto his chest. Also wrapping him up tightly at the time of death. Had old and fresh rib fxx, also face and scalp bruises. Argues that this is a diagnosable entity. But without a confession or eyew?

Non-accidental rib fractures. Michelle Shouldice and Dirk Huyer. In: T. J. David, ed., Recent Advances in Paediatrics 18. Edinburgh: Churchill Livingstone, 2000, pp. 63-76. (have) From the Hospital for Sick Children, Toronto. Rib fxx in as many as 35% of abused infants. "It is well recognized that injuries occurring during typical childhood play and falls from low heights do not cause rib fractures." Often missed on CXR. "In fact, infants with rib fractures may display few signs related directly to the fracture. Clinical signs, if any, are nonspecific, such as irritability or mild respiratory distress." "[A]busive rib fractures are usually caused by indirect forces, with no resultant overlying bruising or soft tissue swelling." Accidental rib fxx are caused by MVA or falls from a significant height, usually in ch over 2, and involve violent forces which are associated with a high mortality in accidentally injured children having rib fractures (42% versus 18% in those not having rib fxx) (citing Garcia, Gotschall, Eichelberger, 1990;

Schweich, 1985.) Non-acc rib fxx usually in infants under 1. "Different stages of healing may be evident, reflecting several episodes of injury." Rib fxx typically result from thoracic compression, often during shaking. Posterior rib fxx result from a lever mechanism from forceful squeezing of the thorax. Lateral fxx "are similarly thought to result from indirect trauma applied during compression of the chest. Anterior fractures at the CCJ likely also result from external compression. (Citing Kleinman book at 113) They are likely to be more common than described in the literature, because they are difficult to detect both by imaging and by pathology (citing Kleinman, Blackbourne, 1995.) Dating rib fxx: radiographic dating is extrapolated from features known in long bones: visible fracture line 0-14 d, periosteal rxn 2-14 d, callus 10d-3mo, with Kleinman saying that callus appears at 7-10d. "It appears that the dating of rib fxx radiologically can be an approximation only. Infants who have compressive injury to the bony thorax may experience more than one abusive episode, leading to acute injury superimposed on chronic fractures. This could lead to persistence of the visibility of a fracture line, a feature commonly used to date acute fractures, within an extensive callus, and contribute to difficulties in dating." 71 As to CPR: "In summary, there is no biomechanical or clinical ev to suggest that CPR causes posterior rib fxx in infants without bone disease. Caution shd be used in attributing rib fxx in any location to CPR in infants." 72 As to birth trauma: it is a rare occurrence; when it does occur, it is likely related to large size and traumatic delivery. As to metabolic bone disease, particularly in the setting of prematurity: Amir reported 12 pts with rib fxx among a series of 973 NICU pts, all with demonstrable osteopenia due to LBW and cholestasis, lung disease, TPN, or diuretics. "There is no ev in the literature to suggest that prematurity or LBW result in fxx with normal handling after discharge from hospital." As to OI types I and IV: generalized osteoporosis and bowing should be present; fxx are mostly long bone. "Rib fxx are rare in OI. Kleinman reports that rib fxx from minor trauma in OI are seen in cases where the ribs are obviously thin or osteopenic. Therefore, OI alone should not be accepted as an expl for rib fxx in infants." 75

Imaging of child abuse. Nimkin K and Kleinman PK. Radiol Clin N Amer 2001 Jul; 39(4): 843-864. (have) As to rib fxx, says "Rib fractures are central to a radiologic diagnosis of infant abuse. These injuries are quite unusual even in the setting of severe accidental trauma in infants and rarely if ever result from vigorous cardiopulmonary resuscitation. ...typically result from excessive anteroposterior compression of the chest during shaking or with impact. ...are often symmetric and most frequently involve the middle ribs." "Acute posterior rib fxx in infants are often invisible radiographically. ...fxx near the CCJ are particularly hard to see and are probably more common than reviews suggest." (citing book and Ng & Hall, 1998.) CCJ fractures resemble bucket-handle fxx of long bones. "seem to be associated with abdominal injuries in toddlers and older children"

Controversial aspects of child abuse: a roundtable discussion. Society for Pediatric Radiology. Pediatric Radiology 2001 Nov; 31(11): 760-774. Dr. Boal makes some comments on the specificity of fractures.

"There is excellent scientific evidence that posterior rib fractures at the costovertebral junction have a high specificity for abuse; there is no argument. However, I was surprised to find, at least in our patient population, that the majority of identified rib fractures in abuse were other than CVJ in location... Rib fxx occurred in all three of our pt populations."

1608 rib fractures

1463 were in the abuse group

145 were in the nonabuse or unknown groups

Divides rib fractures by location:

	Ab	Nonab	Unk	Tot
CVJ	479	23	22	524
Postr	257	10	22	289
Lat	301	17	3	321
Ant	251	14	22	287
CCJ	175	0	12	187
Tot	1463	64	81	1608

[Interesting that the nonabuse group never had CCJ fractures, while they did have posterior and CVJ fractures. That argues that it is CCJ fractures, not posterior fractures,

that are specific for abuse. I wonder what the statistical workup of these figures would show as to SENS and SPEC, PVP and PVN. –JKR]

Pediatric ribs: a spectrum of abnormalities. Glass RBF, Norton KI, Mitre SA, Kang E. Radiographics 2002; 22: 87-104. Complete review of the radiology and abnormalities of the ribs.

Cause and clinical characteristics of rib fractures in infants. Bulloch B, Schubert C, Brophy P et al. Pediatrics 2002; 105: e48. Cited by Dr Reece (2002) as finding that abuse was the etiology in 32 out of 39 infants with rib fxx. (82%)

Chest compressions in an infant with osteogenesis imperfecta type II: no new rib fractures. Sewell RD, Steinberg MA. Pediatrics 2000; 106: 1-6. A 38 week newborn with two episodes of CPR. Short, flexed limbs. Osteopenia, multiple rib fxx, pulmonary hypoplasia, short, broad, crumpled multiply fractures humeri and femora. This severe malignant form of OI. But note that acute rib fxx can be hard to see radiographically, see Kleinman, Marks, 1988.

What the literature tells us about rib fractures in infancy. Reece RM. SBS Quarterly, Fall 2002, pp. 2,3,6. (have) A very current literature review as of the end of 2002. Salient points:

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).
3. No cutaneous bruises are seen over posterior rib fxx. (citing Cadzow, Armstrong, 2000).
4. Costochondral junction fractures are rare. [??? -- not consistent with my own observations. see also Kleinman’s comment, 2001. -- JKR] They are difficult to visualize radiographically, particularly before callus forms. (citing Ng, Hall, 1998).
5. Acute rib fxx will not show up radiographically unless there is displacement. (citing Kleinman, 2d ed.)
6. The differential dx of rib fxx in under-2 age group would be:
 - a. Birth injury rarely if ever, and not unless dystocia, very large fetus, breech delivery.
 - b. Accidental severe compression: MVA, heavy object.
 - c. OI will be diagnosable by other means.
 - d. TBBD forget it.
7. “Rib fractures in infants and young children, especially those located in the posterior arcs of the ribs, are very close to being diagnostic of inflicted injury. Their specificity is based on their location, the abundance of the literature failing to ascribe them to other causes, and the biomechanics required to produce them.”

Rib fractures after chest physiotherapy for bronchiolitis or pneumonia in infants. Chalumeau M et al. (Paris) Pediatric Radiology 2002 Sep; 32(9): 644-647. The reported causes of rib fxx in infants are:

Child abuse

Accident
CPR
Bone fragility
Birth trauma
Severe cough

Five infants median age 3 months who had CPT and where all other potential causes were ruled out. The median number of rib fxx was four (range 1 – 5). The Fracs were lateral in four pts and posterior in one pt. Unilateral in four and bilateral in one. Estimates that 1/1000 infants receiving CPT will get rib fxx. CPT shd be considered a potential but very rare cause of rib fxx in infants.

The positive predictive value of rib fractures as an indicator of nonaccidental trauma in children. Barsness KA, Char E-S, Bensard DD. J Trauma 2003; 54: 1107-1110. The PPV was 95%.

Knight's Forensic Pathology, 3d ed., 2004. Saukko P, Knight B. New York: Oxford University Press, 2004. If rib fxx are less than 10 days old, no callus is formed. "With fresh fractures found at autopsy, the possibility of chest compression during cardiopulmonary resuscitation must always be considered, even though some paediatricians and radiologists will strenuously deny the possibility of this happening. The literature has different opinions on this matter, though it is admittedly very uncommon... It must be conceded that pliable infant ribs are unlikely to be broken by proper cardiac massage, which in infants whould b e performed with finger pressure; however, lay persons, especially in the panic of apprent collapse and death, may forcibly pump the small chest using techniques intended for adult resuscitation." pp. 467-468. "It is said by paediatricians and radiologists that anterior rib fractures are rare in infancy other than from child abuse; though this is probably gen erally correct, care must be taken to exclude bony injury fromt the now almost universal attempts at resuscitation (even thoug infant ribs are very pliable)." p. 224.

In children undergoing chest radiography what is the specificity of rib fracture for non-accidental injury? Williams RL, Connolly PT. Arch Dis Chil 2004; 89: 490-492. (UK) According to Dr Kleinman's review in the *Quarterly* for Winter 2005, this is a meta-analysis of the literature. Concluded from the literature that rib fractures in children under three years of age are highly predictive for NAI.

Hypocalciuric hypercalcemia presenting as neonatal rib fractures: a newly described mutation of the calcium-sensing receptor gene. Nyweide K, Feldman K, Gunther DF et al. Pediatric Emergency Care 2006; 22: 722-724. A seventeen day old infant with multiple healing rib fractures. Neonatal hyperparathyroidism.

How good is the evidence available in child protection? Sibert JR, Maguire SA, Kemp AM. BMJ Jan 30 2007. Refuting a lead article by David Chadwick on 22 July 2006 stating that the published evidence base for child protection is robust. On the contrary, say these authors, who are part of an evidence-based medicine review team. One of the few things they find to be well established is the fact that rib fractures are highly specific for abuse. See below under "Unclassifiable."

Flail

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.

Tibia / lower leg

Infantile cortical hyperostosis. Caffey J. J Ped 1946; 29: 541-559. Describing what is now called Caffey's disease. (not fractures)

Spiral tibial fractures of children: a commonly accidental spiral long bone fracture. Mellick LB and Reesor K. Am J Emerg Med 1990; 8: 234-237. Toddler's fracture.

Fracture-dislocation of the lumbar spine in an abused child. Gabos PG, Tuten HR, Leet A, Stanton Rp. Pediatrics 1998 Mar; 101(3 pt 1): 473-477 Case report and review of the literature. A 15 month old child with flaccid paralysis below the umbilicus and neurogenic bladder, L1-L2 subluxation, boyfriend confessed. A rare injury outside the context of vehicular trauma. They mention also SCIWORA, and advocate detailed vertebral column x-rays in a bone survey.

Case report: multiple neonatal fractures -- dietary or deliberate? Duncan RA and Chandy J. Clin Radiol 1993; 48: 137-139 Case of neonatal rickets with SUDS at 5 mos. Baby born at 28 weeks. Brought in at 1 month bc of reluctance to move her right leg. Found to hv a 1 wk old distal femur fx, 2 wk old fxx R tib fib, 2-3 wk fxx of both radii. , R 6th rib, healed L 5th rib. The metaphyses were splayed and frayed. Bruises seen. Diagnosed rickets with rule-out child abuse, undet. Three years later, a second daughter of the same parents presented with a spiral fx of L femur and inconsistent hx. Metaphyseal splaying and fraying, and osteopenia and diffuse generalized periosteal reaction rather than localized distinguish rickets from child abuse. OI was also in the differential.

Toddler's fracture: presumptive diagnosis and treatment. Halsey MF, Finzel KC, Carrion WV et al. J Pediatr Orthop 2001; 21: 152-156. Spiral-oblique fx of the distal tibia in children under six years who had witnessed trauma resulting in limping or non-weight-bearing. This article reviews the management of 59 such children with long-leg casts. 39 of them initially had normal x-rays, with the fx only showing up as healing fxx on followup x-rays. Recommends that children with this history be casted regardless of whether a fx is definitely demonstrated, bc as long as there is no inflammation the cast is well-tolerated. (as reviewed by Daniel R. Cooperman, MD in Child Abuse Quarterly July 2001.)

Imaging of child abuse. Nimkin K and Kleinman PK. Radiol Clin N Amer 2001 Jul; 39(4): 843-864 at 857. "Caffey's disease is a rare condition characterized by widespread SPNBF. Its onset in early infancy corresponds with the peak incidence of serious inflicted injuries. Because bone density is preserved and there is no history of trauma, child abuse may be considered initially."

Pelvis

Non-accidental pediatric pelvic fracture: a case report. Nancy C Pendergast, Stephen J DeRoux, N Volkan Adsay. Pediatr Radiol 1998; 28: 344-346. Four year old girl w multiple abusive trauma including vaginal tear, lac ileum. Autopsy found healing fxx of the left acetabulum and the left pubic ramus.

Pelvic fractures in infants as a sign of physical abuse. Starling SP, Heller RM, Jenny C. Child Abuse & Neglect 2002; 26(5): 475-480. Case report of two infants aged 3m and 3m with unexplained pelvic fractures. Case 1 had 30 fxx of different ages. Case 2 had multiple fxx areas. Literature review found 11 cases. Concluded that "Pelvic fractures occurring in the absence of serious, well documented accidents should be considered highly suspicious for child physical abuse." The most common fractures seen in abused children are metaphyseal, rib, skull, and long bone fractures.

Skeletal injuries associated with sexual abuse. Johnson K, Chapman S, Hall CM. (UK) Pediatric Radiology 2004 Aug; 34(8): 620-623. See above under "In general."

Clavicle

Newborn clavicle fractures. McBride MT, Hennrikus WL, Mologne T. Orthopedics 1998; 17: 317-320
Original prospective study of 9,106 newborns for clavicle fractures. 7,317 vag, 1,789 C/S. Found 0.5% incidence overall, but .6% if only vag deliveries are considered. Risk factors were high birth weight, post-dates, forceps, shoulder dystocia. Not a single case happened in C/S delivery.

Humerus

Humeral fractures without obvious etiologies in children less than 3 years of age: when is it abuse? Strait RT, Siegel RM, Shapiro RA. Pediatrics 1995 Oct; 96(10): 667-671.

Etiology of supracondylar humerus fractures. Farnsworth CL, Silva PD, Mubarak SJ. J Pediatr Orthop 1998; 1: 38-42. Studied records on 388 children. Girls more often. The nondominant arm was injured twice as often. Ages 4-7 mainly. Usually playground falls in this age group. Found that only 2 of these fxx were known to be caused by abuse. But the only screen for abuse used was if the consulting physician suspected abuse and called for a social service consult; this happened in only four cases. If you look at the under-2 age group, this was 40 patients, and of these, 23% had no known etiology, 48% had short falls from furniture.

Long-bone fractures in young children: distinguishing accidental injuries from child abuse. Thomas SA., Rosenfield NS, Leventhal JM, Markowitz RI. Pediatrics 1991; 88(3): 471-476. "humerus fractures were found in 14 children; 11 were due to abuse and 3 were due to accidental injuries. All the abusive humeral fractures were either midshaft or distal or proximal metaphyseal... All three of the children with accidental humeral fractures had fallen on their elbows... All three had supracondylar fractures of the distal humerus.

Kleinman, p. 216: "Supracondylar fractures [of the humerus] are usually accidental injuries beyond infancy and can occur when children fall while running or playing."

Best evidence topic report: humeral fractures and non-accidental injury in children. Williams R, Hardcastle N. Emerg med J 2005 Feb; 22(2): 124-125.

Back

Thoracolumbar fracture with listhesis -- an uncommon manifestation of child abuse. Levin TL, Berdon WE, Cassell I, Blitman NM. (Montefiore). Pediatric Radiology 2003 May; 33(5): 305-310. Only six prior reports in the literature. Reporting seven new cases. Findings varied from subtle listhesis of one vertebra on another to frank vertebral dislocation., most commonly at L1-L2. All but one case had paravertebral calcification.

Mandible

Infant mandibular fractures: are you considering child abuse? Schlievert RM. Pediatric Emergency Care 2006; 22: 181-183. Case report of a six month-old with bilateral mandibular fractures, history of a short fall, which was not compatible with the findings. Advises a face CT plus a full workup for child abuse in any infant that presents with any facial injury. Reviews the literature, which refers to mandibular fxx from alleged short falls that were not worked up. (From Scott Krugman's review in the Spring 07 Quarterly)

OI AND RICKETS

Signs

- Family history of fracturing
- Fractures usually spiral or transverse but can be metaphyseal*
- Abnormally large or small callus
- Blue sclerae
- Hearing impairment in adolescence
- Unusual skull shape
- Bell-shaped rib cage
- Dentinogenesis imperfecta**
 - W/wo slowed dental development
 - Opalescent dentin
 - Broken, discolored, or eroded teeth
 - Yellow or gray-blue transparent teeth
 - Short roots
 - Bell-shaped crowns
 - Wide pulp chambers
- Hypermobility of joints
- Pseudarthrosis
- Bruisability
- Short stature
- Abnormally short bones
- Wormian bones
- Osteopenia
- Thin cortex
- Bowing of lower limbs
- Scoliosis
- Overtabulation (abnormally narrow diaphysis)
- Metatarsus varus
- Codfish vertebrae
- Crush fractures of vertebrae
- Aortic dilatation

* Jan Bays, *Conditions mistaken for child abuse*. In: Reece & Ludwig, 2d edition (2001), p. 199.

** Jan Bays advises get a dental consult if OI is suspected.

General discussion (combined from *Nelson's Pediatrics*, 15th ed. and Jan Bays).

Generally the recessive forms (II and III) are the more severe. They are rare.

We are now (2004) up to VIII types. The newer types are not dependent on the gene for collagen I. Watch out for mosaicism and co-occurrence with other diseases.

Type I, the most common type, is an amino acid substitution in collagen alpha I. 1/30,000 live births. Autosomal dominant. Variable expression. The sclerae are deep blue-black. 10% have fractures at birth. Usually live well into adulthood. Have osteopenia and normal callus formation. Variable hearing loss. Unlikely to be mistaken for abuse bc of the obvious blue sclerae, abnormally short stature, abnormal teeth, and wormian bones.

Type II is lethal congenital type (osteogenesis imperfecta congenita). Crumpled bones and beaded ribs at birth. 1/60,000 LB. About 50% are stillborn, the remainder die in neonatal life owing to respiratory insufficiency from chest deformity. Autosomal recessive.

Type III is a disease of the newborn or young infant. Autosomal recessive. Severe bone fragility and multiple fractures leading to progressive skeletal deformity (in infancy). The sclerae may be blue at birth and get less blue with time. Fractures are usually present at birth. Skull deformity in childhood is severe, with temporal bulging and triangular head. Many die in infancy and childhood; very few reach adulthood. Generalized osteopenia, codfish vertebrae; skull shows multiple wormian bones.

Type IV is autosomal dominant but is rare. This is the one that can be confused with abuse. Has variable bone fragility without the other features of type I. May have blue sclerae at birth or not have blue sclerae. Variable age of onset of fractures, varying from birth to adulthood. Bowing of the lower limbs at birth may be the only feature. There is generalized osteopenia. Most have short stature.

Type V described by Glorieux, Ward et al., looks like type IV but has hyperplastic callus.

Type VI described by Glorieux, Ward et al. in 2002, looks like type IV also. It has fractures diagnosed between 4 and 18 months of age, more frequent fractures, white or only faintly blue sclerae, never has dentinogenesis imperfecta. Has vertebral compression fractures. Lumbar spine areal bone density is low and serum alkaline phosphatase is higher than type IV. No mutations of the type I collagen gene, and collagen protein assays are normal. Bone biopsy histology shows a fish-scale pattern under polarized light. Accumulation of osteoid due to a mineralization defect. The underlying genetic defect is unknown. Glorieux FH, Ward LM, Rauch F et al., (McGill Univ), Osteogenesis imperfecta type VI: a form of brittle bone disease with a mineralization defect. J Bone Miner Res 2002 Jan; 17(1): 30-38.

Type VII described by Glorieux, Ward et al. (McGill Univ) in 2002 in four children and four adults among an isolated Indian kindred in northern Quebec province, is an autosomal recessive. According to their abstract, the phenotype is moderate to severe, characterized by fractures at birth, bluish sclerae, early deformity of the lower extremities, coxa vara, osteopenia, and rhizomelia. Bone biopsy resembles type I. Chromosome 3p22, outside the loci for type I collagen. Ward LM, Rauch F, Travers R et al., Osteogenesis imperfecta type VII: an autosomal recessive form of brittle bone disease. Bone 2002 Jul; 31(1): 12-18. Labuda M, Morissette J, Ward M et al., Osteogenesis imperfecta type VII maps to the short arm of chromosome 3. Bone 2002 Jul; 31(1): 19-25.

Type VIII

The battered child syndrome: pitfalls in radiological diagnosis. Radkowski MA. Pediatric Annals 1983; 12: 894-903

Brittle or battered? Carty H. Arch Dis Chil 1988; 63: 350-352

OI in the differential diagnosis of child abuse. Paterson CR, McAllion SJ. BMJ 1989; 299: 1451-1454

Subdural hematoma as a rare complication of OI. Tokoro et al., Neuros 1988; 22: 595

Intracranial hemorrhage is not part of the picture. See Kleinman's book.

Look out for dentinogenesis imperfecta.

OI type I has vasculopathy, i.e. enlargement of the aortic root, myxoid degeneration of the valves, and cystic medial necrosis. Types III and IV do not have vasculopathy. Thanks to Karen Kelly of the Edwards Registry of Cardiovascular Disease in St. Paul.

Differentiation of child abuse from osteogenesis imperfecta. Ablin DS, Reinhart M, Grix A. Am J Roentg 1990 May; 1035-1046. Has side-by-side tables

Child abuse or osteogenesis imperfecta: how can we tell? Gahagan S and Rimsza ME.

Pediatrics 1991; 88: 987-992. Used collagen type I biosynthesis studies in 3 infants suspected for abuse. All three were OI. (But note that according to later articles, collagen studies should rarely be needed; the diagnosis is made by clinical, historical, anatomic, and radiologic means).

An autopsy case of osteogenesis imperfecta initially suspected as child abuse. Ojima K, Matsumoto H, Hayase T, Fukui Y. FSI 1994; 65(2): 97-104

Fractures of the first rib in child abuse. Strouse PJ, Owings CL. Radiology 1995 Dec; 197(3): 763-765. Includes one case of OI causing a congenital first-rib fx.

Studies of collagen synthesis and structure in the differentiation of child abuse from osteogenesis imperfecta. Steiner RD, Pepin M and Byers PH. J Peds 1996 Apr; 128(4): 542-547 Child abuse is diagnosed by clinical assessment, not by collagen studies. Collagen studies are useful only where diagnostic uncertainty remains after clinical assessment by experienced practitioners. "Routine biopsy of children suspected to have been abused is unwarranted."

Conditions mistaken for child abuse: part I. Stewart GM, Rosenberg NM. Pediatric Emergency Care 1996; 12: 116-121

Non-accidental injury: confusion with temporary brittle bone disease and mild osteogenesis imperfecta. Ablin DS, Sane SM (UCD). Pediatr Radiol 1997 Feb; 27(2): 111-113. Careful review of the scientific literature clearly indicates that collagen analysis to exclude mild forms of OI, especially type IV, is recommended only in rare cases in which dx of NAI remains in doubt even after thorough eval by experienced radiologists and/or other physicians. Cited by APRI Update, 2004 (below) for the proposition that "The likelihood of a clinician seeing a child with mild type IV OI and with white sclera, normal hearing, normal dentition, negative family history, and no wormian bones is exceedingly rare."

Osteogenesis imperfecta: a review. Deborah S. Ablin. Can Assoc Radiol J 1998; 49: 110-123. This article mentions a hypothetical disease entity, not (yet) medically accepted, which would be called "Temporary Brittle Bone Disease." It would be restricted to the under six-months age group, not existing in children older than six months. Cited by APRI Update, 2004 (below) for the proposition that "It is vitally important that all multidisciplinary teams know that TBBD is not a recognized disease." In commenting on this point in Child Abuse Quarterly for October 1998, David F. Merten terms this "a medical hypothesis without supporting scientific data. Until such data are forthcoming, TBBD cannot be considered a medically or legally acceptable diagnosis."

Images in clinical medicine: blue sclerae in osteogenesis imperfecta. Leidig-Bruckner G, Grauer A (Heidelberg). N E J Med 1998 Oct; 339(14): 966

Osteogenesis imperfecta. Kocher MS, Shapiro F. (Children's Hospital of Boston). J Am Acad Orthop Surg 1998 Jul-Aug; 6(4): 225-236.

Osteogenesis imperfecta: a review of basic science and diagnosis. Minch CM, Kruse RW. Orthopedics 1998 May; 21(5): 558-567. Literature review with 81 refs.

Orthopedic aspects of child abuse. Kocher MS. (Children's Hospital of Boston). J Am Acad Orthop Surg 2000 Jan; 8(1): 10-20. See above under "Fractures -- In General."

Rickets re-emerging in US. Journal of Pediatrics/ Medscape Wire, Aug. 18, 2000. August 2000 issue of the Journal of Pediatrics is reported to contain two arts which show 30 cases of vitamin D-dependent rickets over a ten year period in exclusively breast-fed Negro infants from Cleveland. If only breast fed and no vitamin D supplement given, Negro infants are at risk because apparently their skin does not form much vitamin D due to heavy pigmentation. See below.

Chest compressions in an infant with osteogenesis imperfecta type II: no new rib fractures. Sewell RD, Steinberg MA. Pediatrics 2000 Nov; 106(5): E71. A 38 week newborn with OI type II underwent in-

hospital CPR. The authors compared before and after chest x-rays and found “no new radiographically visible rib fractures.” [But note that acute rib fxx can be hard to see radiographically; notice how carefully worded their finding is, and that it doesn’t exclude refractures. --JKR] Includes a literature review of 4 previously published studies of rib fractures and CPR. From the abstract: “This unique case supports previous articles that have concluded that rib fractures rarely, if ever, result from CPR in pediatrics, even in children with a lethal underlying bone disease.”

Commentary: nutritional rickets in Georgia. Tomashek KM, Nesby S, Scanlon K et al. Pediatrics electronic article 2001 Apr; 107(4): e45. (have) 5 breastfed Black male children who were weaned late, had no VitD suppl during nursing, had inadequate sun exposure. At 8 mos 2 had hypocalcemic tetany and sz, one had ARDS and osteopenia on CXR, one had a febrile sz. Two others at 21 months had CXR, fever, failure to bear weight on tibia. Pathognomonic findings on radiography were:

- Osteopenia
- Frayed and cuffed metaphyses
- Widened epiphyseal plates
- Pathologic fracture

Physical findings were:

- Genu varum
- Rachitic rosary
- Frontal bossing
- Large fontanelle
- Lumbar kyphosis

Severe nutritional deficiencies in toddlers resulting from health food milk alternatives. 2001. See under “Folk customs.”

Imaging of child abuse. Nimkin K and Kleinman PK. Radiol Clin N Amer 2001 Jul; 39(4): 843-864 (have) OI is 1:20,000 LB. Gives Table 3 listing the current “Classification of OI modified for the DDX of nonaccidental trauma.” (Noting, however, that this classification is going to be replaced by a molecular-genetic reclassification.)

Notes that Types II and III are too obvious and severe to usually be a problem in the DDX of inflicted trauma. Type I can usually be diagnosed or ruled out radiographically without the need to resort to collagen biochemical testing or genetic testing. Type IV is more of a problem, although it is less than 5% of all cases of OI. But type IV is a rare disease.

Most OI fractures are associated with *demineralization* and *bowing*.

Type I (most cases)

Mild phenotype

- Normal or close-to-normal stature
- Hearing loss in about 50%
- Mild but significant osteoporosis
- Wormian bones

Group IA

- Blue sclera
- Normal teeth

Group IB

- Dentinogenesis imperfecta

Type II always lethal in the prenatal period

Type III

Moderately severe early

- Short stature at birth with bowed legs
- Blue-gray sclera at birth normalizing later

DI
 Many fxx early and continuing
 Progressive deformation
 Severe osteoporosis
 Wormian bones with skull deformation
 Thin angulated long bones
 "Popcorn" metaphyseal calcifications
 codfish vertebrae
 exuberant callus

Type IV similar to type I

Short stature early
 Triangular face with temporal bulge
 Often prenatal fracturing
 Normal sclera
 No hearing loss usually
 Group IV-A without DI
 Group IV-B with DI

Testing for osteogenesis imperfecta in cases of suspected non-accidental injury. Marlowe A, Pepin MG, Byers PH. J Med Genet 2002 Jun; 39(6): 382-386. Tried to determine whether laboratory testing identifies OI children unrecognized by clinical examination in situations where NAI is suspected. From the abstract: The authors reviewed the medical records and biochemical test results on 262 pediatric patients, including cultured fibroblasts. Eleven (11) of the samples had alterations in the amount or structure of type I collagen synthesized consistent with the diagnosis of OI, and in 11 others they could not exclude OI. Referring physicians correctly identified OI in 6 of the 11 positive cases, **missed it in 3**, and in two other cases the authors could not tell whether the referring physicians had made the diagnosis or not. In the eleven inconclusive cases, none of the referring clinicians suspected OI. Four children believed by referring physicians to have OI ended up testing negative. "Given the inability to identify all children with OI by clinical examination in situations of suspected NAI, laboratory testing for OI (and other genetic predispositions for fractures) is a valuable adjunct in discerning the basis of fractures, and may identify a small group of children with previously undiagnosed OI.

Double valve replacement in a patient with osteogenesis imperfecta. Chryssant GS, Cassivi SD, Carey CF, Sundt TM. J Heart Valve Dis 2002 Sep; 11(5): 751-754. Aortic and mitral valve disease is well documented in OI, and valve replacement has had a high mortality. These surgeons report better results.

For genetic testing (\$2,100)
 Send EDTA blood to
 Matrix DNA Diagnostics
 MCP Hahnemann University
 10310 NCB, Mail Stop 421
 245 North 15th Street
 Philadelphia, PA 19102-1192
 215-762-7234

For fibroblast culture
 Contact
 Dr. Peter Byers
 University of Washington
 206-543-4206

A local expert on OI and OI genetics:
 William Wilcox, MD, PhD
 Associate Professor of Pediatrics
 Cedars-Sinai Medical Center
 8700 Beverly Blvd, SSB-3
 Beverly Hills, CA 90048
 310-423-6673

See also "Temporary Brittle Bone Disease," below. See also scurvy and Barlow's disease (infantile scurvy) under "Differential diagnosis -- specific disease entities," above.

Retinal hemorrhages in type I osteogenesis imperfecta after minor trauma. Ganesh A, Jenny C, Geyer J, Shouldice M, Levin AV. Ophth 2004; 111: 1428-1431. Three patients culled from SIGCA-MD-L. All had short-fall histories and two presented with seizures. Two had blue sclerae. Pt 1 had flame hemorrhages in the posterior pole. Pt 2 had intraretinal hemorrhages and vitreous hemorrhages. Pt 3 had small scattered intraretinal hemorrhages in the posterior pole. The authors postulate that the RH were caused by softness of the globe and capillary fragility. Not clear what if any the intracranial injuries were.

Classical osteogenesis imperfecta and allegations of nonaccidental injury. Paterson CR and McAllion SJ. Clinical Orthopaedics and Related Research (UK) 2006; 452: 290-294. They report 12 children with classical OI who were diagnosed as NAI and seven of whom were removed from their families, with criminal proceedings instituted against the parents of some. Even though in six of them there was a positive family history, nine of them had blue sclerae, four had abnormally large fontanelles. Five infants had more than ten Wormian bones. (Should take a Towne's view to see these.) The reason they were diagnosed as NAI was that the parents were unable to explain the numerous fractures. (Case 1 had a neonatal X-ray taken which showed conclusively that many of his fractures had occurred *in utero*.) Cites Popich & Smith, 1972 for the size of normal fontanelle up to age 12 months. (Popich GA, Smith DW, Fontanels: range of normal size. J Paediatr (UK) 1972 May; 80(5): 749-752.) [I have that paper, thanks to Dr Andrews. It shows that the maximum normal size, at least in a British population, was 5 cm; that is two S.D. above the mean of 3 cm. -JKR]

Unexplained fractures in infancy: looking for fragile bones. Bishop N, Sprigg A, Dalton A. Arch Dis Chil (UK) 2007; 92: 251-256. See above under "Fractures -- In General." Includes a discussion of OI as well as prematurity and all metabolic bone diseases of infancy.

TEMPORARY BRITTLE BONE DISEASE (TBBD)

Osteogenesis imperfecta in the differential diagnosis of child abuse. Paterson CR. Child Abuse & Neglect 1977; 1: 499.

Osteogenesis imperfecta in the differential diagnosis of child abuse. Paterson CR and McAllion SJ. BMJ 1989; 299: 1451-1454. Cited by APRI Update, 2004 (below) as having found that NAI had been suspected in 113 out of 804 patients with OI while in fact none of them had suffered from abuse. APRI Update goes on to argue that, this study has been criticized by Jan Bays, (Chapter 9 in Reece), concerning "the lack of diagnostic criteria, and the fact that the metaphyseal fractures, vomiting, diarrhea, breathing abnormalities, liver enlargement, rib fractures, and other abnormalities which are listed as symptoms of TBBD are also all classic signs of child abuse and neglect." (Citing also Kleinman, Marks & Blackbourne on the metaphyseal lesion, 1986).

Osteogenesis imperfecta and other bone disorders in the differential diagnosis of unexplained fractures. Paterson CR. J Royal Soc Med 1990; 83: 72.

Temporary brittle bone disease: association with decreased fetal movement and osteopenia. Miller ME, Hangartner TN. Calcif Tissue Int 1999; 64(2): 137-143. Asserts the existence (which is controversial) of this entity by reviewing the medical records of 33 infants whose cases were referred because of multiple unexplained fractures and no history of trauma or findings of abuse and normal laboratory studies for metabolic bone disease. 26 of the 33 were given a diagnosis of TBBD. 17 of the 26 had normal collagen tests. All 26 had normal bone density by plain films, but 8 of them had low bone density by CT and/or radiographic absorptiometry; one had normal bone density by CT/RA. 25 of the 26 had a history of decreased fetal movement or "intrauterine confinement." From the abstract: "These findings implicate decreased fetal movement and intrauterine confinement as contributing factors to temporary brittle bone disease and suggest that normal, unconstrained fetal movement during pregnancy is important for normal fetal bone formation.... The results also demonstrate the usefulness of bone density measurements in

evaluating the infant with multiple unexplained fractures to help distinguish nonaccidental injury from intrinsic bone disease.” The diagnostic criteria given for TBBD are:

- 1) No history of abuse
- 2) No history of trauma
- 3) No external injury
- 4) No other findings of abuse
- 5) No radiographic evidence of metabolic bone disease
- 6) No laboratory evidence of metabolic bone disease

Problem is that his cases are self-selected (some referred by attorneys) and the parental denials of abuse are taken at face value. Also lack of documentation of controls. Also no baseline exists on the normal degree of mineralization of fetal or neonatal skeleton. See criticism letter by Ralph Hicks, 2001, *infra*.

Temporary brittle bone disease: a true entity? Miller ME. Semin Perin 1999 Apr; 23: 174-182. Advocating the existence of the entity.

Non-accidental injury: confusion with temporary brittle bone disease and mild osteogenesis imperfecta. Ablin DS, Sane SM (UCD). Pediatr Radiol 1997 Feb; 27(2): 111-113. Until clinical research scientifically establishes the existence of temporary brittle bone disease, it should remain strictly a hypothetical entity and not an acceptable medical diagnosis.

Child abuse -- controversies and impostors. Robert W. Block. Curr Prob Pediatr 1999; 29: 253-272. Discusses TBBD along with SBS, RH, OI. Says that TBBD is lacking scientific data to support its existence.

Review by Betty Spivack in Child Abuse Quarterly 2000 Jul; vii(3): 21-23. (have)

Another perspective as to the cause of bone fractures in potential child abuse. (letter). Miller M. Pediatric Radiology 2000; 30: 495. Comment on Rooks, Sisler & Burton, 1998. (See under “Cervical spine.”) See Betty Spivack’s commentary in *Child Abuse Quarterly*, April 2001. Dr. Miller contends that the two patients of Rooks et al. with subluxed neck fractures and other fxx were TBBD due to confined pregnancy and prematurity and inappropriate formula leading to calcium and phosphorus deficiency (shd hv bn fed premie formula, which has more of these,) and that their neck fractures were probably caused by positioning for an LP. Noting that they had no external injuries at any time. The authors reply that in one pt the cervical fx was present *before* the LP. Also that both had cervical subluxations, which rule in sever force. Also one pt had CPR and got no new fxx from that. Dr. Spivack agrees that this arg for TBBD is specious as the previous ones. As to Dr. Miller’s arg that the absence of external injury shows that fxx were spontaneous, see Paul Kleinman’s review of Cadzow et al., 2000, in *Child Abuse Quarterly*, April 2001 at 18, where he refers to “the preposterous notion put forth by some authors that the absence of bruising in some way indicates that a child’s rib fractures are due to metabolic bone disease.”

See also the discussion by Jan Bays in “*Conditions mistaken for child abuse*,” in Reece & Ludwig, 2d edition (2001), p. 200.

Imaging of child abuse. Nimkin K and Kleinman PK. Radiol Clin N Amer 2001 Jul; 39(4): 843-864 (have) Comments on TBBD as follows: “Several authors have suggested the existence of a ‘self-limiting variant of OI’ and have coined the term TBBD. Support for this hypothesis is lacking elsewhere in the medical literature, and several reviews have been highly critical of the methodology used in these authors’ studies.” 855

Relating to the methodological shortcomings and the concept of temporary brittle bone disease (letter). Ralph Hicks. Calcif Tissue Int 2001; 68: 316-319. Criticizing Miller’s 1999 study as to (a) lack of social history, and (b) lack of detail about fracture types.

Controversial aspects of child abuse: a roundtable discussion. Society for Pediatric Radiology. Pediatric Radiology 2001 Nov; 31(11): 760-774. (have) (See above under “Battered Child Syndrome.”) Dr. Boal makes some comments on the specificity of fractures. TBBD was proposed as a variant of OI by Paterson

in 1989. His hypothesis has been refuted; Paterson himself has admitted on the witness stand that his patient group included several abused children. Dr. Boal rejects Miller's diagnostic category because it perfectly overlaps the diagnostic features of abuse.

Infants at higher risk to fracture than the general population. Miller M. (letter). *Pediatric Radiology* 2003 Oct; 33(10): 733-734. Part of a debate. See *Pediatric Radiology* 2002 Sep; 32(9): 644-647.

A courtroom diagnosis: countering the defense of temporary brittle bone disease and mild OI. by Joelle Anne Moreno, J.D. *APRI Update*, vol. 16, no. 8, 2004. OI is the most frequent medical/ legal defense in cases of child abuse (citing Reece.) This article looks toward a *Daubert* hearing on whether the defense will be allowed to offer evidence of OI or TBBD/ mild OI. "It is vitally important that all multidisciplinary teams know that TBBD is not a recognized disease." (citing Ablin, 1998).

What is OI? OI is a genetic disorder characterized by bone fragility and frequent fractures. Incidence 1:15,000 to 1:60,000 births.

Type I]	
Type II]	together = 80%. Obvious manif. Type II usually lethal in utero
Type III	-	wormian bones and osteoporosis, easily detected radiographically
Type IV	-	only type that might be confused with child abuse

Citing Jan Bays, *Conditions mistaken for child abuse* (Chapter 9 in Reece & Ludwig, 2001) for the proposition that "One recent study calculated the probability of encountering a child under one year of age with OI and no other features or family findings of the disease as between 1 in 1,000,000 and one in 3,000,000, or an annual incidence of one case every 100 to 300 years in a city of half a million people." Says, "Judges deciding whether to admit a defense diagnosis of a variant form of OI or TBBD must assess the likelihood that child would suffer from this disorder, but have no signs or symptoms of the disease beyond multiple fractures." Citing Ablin & Sane, 1997, above, for the proposition that "The likelihood of a clinician seeing a child with mild type IV OI and with white sclera, normal hearing, normal dentition, negative family history, and no wormian bones is exceedingly rare." Citing Merten in Reece & Ludwig for the fact that the likelihood of diagnostic confusion between OI and child abuse is minimal. [But see Marlowe, Pepin & Byers, 2002, above, showing that clinicians incorrectly failed to suspect OI in three out of eleven children with suspected NAI who ended up testing positive for OI on biochemical tests. -JKR] Citing U.S. Department of Health and Human Services Children's bureau (online), Child Maltreatment, 2001: reports from states to the National Child Abuse and Neglect Data System. <http://nccanch.acf.hhs.gov> that the incidence of child abuse in the 0-3 year age group is 2 to 3 per thousand [which would be 2,000 to 3,000 times the incidence of OI. -JKR]

As far as TBBD, says the dx originated at the 1990 Fourth Annual Conference of OI (citing Ablin & Sane, 1997). "A short-lived developmental bone disease that results in easy bone fracturability in very young children for a limited period of time." (citing Ralph Hicks, 2001; Miller & Hangartner, 1999; Paterson & McAllison, 1989). Bones will break with routine handling. "The assumption that this disease is transient cannot adequately explain how all fractures stop once the child is placed in protective custody, when routine handling obviously continues." TBBD is not a recognized clinical entity. **However**, the Arizona Supreme Court recently held that it was an abuse of discretion for the trial court to exclude defense expert testimony of Dr. Colin Paterson on TBBD -- "arguably the world's preeminent TBBD expert," in the words of the court. *State v. Talmadge*, 999 P.2d 192 (Ariz. 2000). The author goes on to discuss this point. In their 1989 article (see above) Colin Paterson and Susan J. McAllison found that 113 out of 804 OI patients had been incorrectly suspected to suffer from child abuse. This article criticized by Jan Bays and others on the ground that the symptoms displayed by those patients are exactly the symptoms of child abuse, many of them not bone-related, and that many of these patients had metaphyseal fractures and rib fractures. Judge Peter Singer of the Royal Courts of Justice, Family Division, has recently ruled that TBBD testimony is inadmissible and not scientifically valid, citing "the subjectivity, the unreliability, the unscientific and unproved nature of Dr. Paterson's speculations that TBBD exists as a clinical entity." *Re X (Non-Accidental Injury: Expert Evidence)*, 2 F.L.R. 1, 27 (Royal Courts of Justice, Family Div. 2001).

Critical review of “temporary brittle bone disease.” Mendelson KL. Pediatric Radiology 2005; 35(10): 1036-1040. This review is composed by the Society for Pediatric Radiology and the European Society of Paediatric Radiology. They dismiss TBBB as a complete fraud. Its advocates, who are few, propose two etiologies: self-limited copper deficiency and disuse osteopenia from lack of intrauterine movement.

Evaluating infants and young children with multiple fractures. Carole Jenny and the AAP Committee on Child Abuse & Neglect. Pediatrics 2006 Sep; 118(3): 1299-1303. TBBB does not exist

FOLK CUSTOMS

"Fallen fontanelle" (caida de mollera): a variant of the battered child syndrome. Guarnaschelli J, Lee J, and Pitts FW. JAMA 1994; 118: 168-171 or is it JAMA 1972; 222: 1545? A 2 mo male with bilateral SDH, sz, tense fontanelle, F&D, was shaken upside-down by parents to relieve a fallen fontanelle.

Folk remedies and child abuse: a review with emphasis on caida de mollera and its relationship to shaken baby syndrome. Karen Kirhofere Hansen. Child Abuse Negl 1997; 22: 117-127. (have abstract)
Treatment for caida de mollera consists of a number of gentle, nonviolent maneuvers quite different from shaking.

Infant head molding: a cultural practice. Ellen Fitzsimmons, Jack H Prost, Sharon Peniston. Arch Fam Med 1998; 7: 88-90. Binding the cranial bones. This practice is ancient and very common throughout the world; see chapter 1 in Helfer & Kempe's 1968 book, "The Battered Child Syndrome." It is nonpathologic except where it serves as a prelude to human sacrifice, as in the Inca.

Skin scraping, cupping, and moxibustion that may mimic physical abuse. Look KM and Look RM. JFS 1997 Jan; 42(1): 103-105.

Severe nutritional deficiencies in toddlers resulting from health food milk alternatives. Carvalho NF, Kenney RD, Carrington PH, Hall DE. (Atlanta) Pediatrics 2001 Apr; 107(4): e46. (have) Health food beverages. Case 1: kwashiorkor in a 22 mo toddler who was breastfed until 13 mos and weaned to a so-called “rice milk” because of a perceived cow milk allergy. This stuff is extremely low in protein. Case 2 rickets in a 17 mo Black male breastfed until 10 mos, weaned to a soy health food beverage which was not fortified with either vitamin D or calcium. Had complete growth arrest at 9 mos. Was unable to crawl or roll over at 22 mos.

Chapters 24 and 25 of Giordano AP and Alexander R, *Child Maltreatment: A Clinical Guide and Reference*, 3d ed. St. Louis: G.W. Medical Publishing, 2005. In Chap. 24 “Cultural Aspects,” it discusses *cao giao* as “rubbing the edge of a heated coin vigorously in downward strokes against the skin on the trunk from the midline laterally,” producing linear ecchymoses over bony surfaces of the trunk. Cupping as burning small amounts of material on the skin beneath a cup or glass, resulting in round red areas on the skin with or without burns. Moxibustion as the moxa herb is rolled into a ball, placed on the body part, and burned; may lead to circular or targetoid burns. *Mollera caida* as bouncing or dropping an infant or holding him upside down and striking his feet, or yanking the nipple out of his mouth, so as to treat the condition of “fallen fontanelle.” Alternative medicine, meaning Christian nonmedical therapy/ neglect. CAPTA provides the legal foundation.

RITUAL ABUSE

Religion and child abuse (letter). James L. Fletcher. Pediatrics 1988 Oct; 82(4): 673-674. Comments briefly on the religious exemption existing in certain state child abuse statutes.

Child abuse by drowning. Griest KJ and Zumwalt RE. Pediatrics 1989 Jan; 83(1): 41-46. See below under "Drown." Their case 2 was a 3 y o girl whose religiously fanatical mother cleansed the devil by forcibly pouring water down her throat.

The differential diagnosis of ritual abuse allegations. Bernet W and Chang DK. (Vanderbilt) JFS 1997 Jan; 42(1): 32-38. The DDX includes:

1. Cult-based ritual abuse
2. Pseudoritualistic abuse
3. Activities by satanic groups
4. Repetitive psychopathologic abuse
5. Pedophilic abuse
6. Child porn portraying ritual abuse
7. Distorted memory
8. False memory
9. False report due to severe mental disorder
10. Pseudologia phantastica
11. Adolescent behavior simulating ritual abuse
12. Epidemic hysteria
13. Deliberate lying
14. Hoax

BURNS

Child abuse by burning. Stone NH, Rinaldo L, Humphrey CR et al., Surg Clin N Amer 1970; 50: 1419-1424. Cited by Kim Collins (2003 Check Sample) for the proposition that risk factors for abusive burns include male, age under 4, youngest of siblings, and Black. Perpetrator is lower-class.

Child abuse by burning: an index of suspicion. Purdue GF, Hunt JL, Prescott PR. J Trauma 1988; 28: 121-124. Cited by Kim Collins (2003 Check Sample) for the proposition that an estimated 10% to 25% of pediatric burns are inflicted, most often contact burns. The mortality of inflicted burns is 30% - 50%, while that of accidental burns is only 2%. Perpetrator is often a female.

Burn abuse: a four year study. Schowers J, Garrison K. J Trauma 1988; 28: 1581-1583. Cited by Kim Collins (2003 Check Sample) for the proposition that Inflicted burns often occur in children under 4, with 2 ½ being the average age. The most frequent burn is the contact burn. And that burns are present in "up to" 25% of all child abuse cases???

Child abuse by burning -- an index of suspicion. Purdue GF, Hunt JL, Prescott PR. J Trauma 1988; 28(2): 121-124. Cited by Kim Collins, 2006, as an original source for pattern scalds.

Etiology and outcome of pediatric burns. Morrow SE, Smith DL, Cairns BA. J Pediatr Surg 1996; 31: 329-333.

[Risk factors for abusive burns] Clark KD, Tepper D, Jenny C. Pediatr Emerg Care 1997; 13: 259-261. Used a 13-point ER questionnaire to improve MD recognition of suspicious burns.

Non-accidental burns in children. Andronicus M, Oates RK, Peat J, Spalding S and Martin H. Burns 1998; 24: 552-558. (Australia). Reports 507 consecutive pediatric burn admissions. 498 were accidental. 69 were abuse or possible abuse. Abuse burns were more likely to involve both hands or both feet. The inflicted ones tended to be more serious. But there were no diagnostic discriminant features.

Profile of an abused burned child. Bennett B and Gamelli RL. J Burn Care Rehab 1998; 19: 88-94. Found 79 abuse cases among 321 consecutive pediatric admissions to a burn center. Socioeconomic and

demographic data are included. Abuse was found in 44% of the welfare cases and 11% of the insured cases. 50% of the single-parent homes versus 13% of two-parent homes. 41% AA, 40% White, 15% H. Reece, in reviewing this paper, says that, "The authors allude to the progressive nature of abusive injuries and state that, 'many of the children admitted to our unit showed signs of parental previous abuse, such as old burn injuries in various stages of healing, old fractures, closed head injuries, retinal hemorrhages, patterns of welts and bruises,' but they give no figures to support these statements."

Burns of children caused by electric stoves. Still J, Craft-Coffman B, Law E et al. J Burn Care Rehab 1998; 19: 364-365. Reports eight patients who were burned from tipping over stoves, caused by the child climbing on the stove. Reece comments that, "Once again we see that one of the distinguishing characteristics of accidental burns is the 'splattering effect.' Abusive burns are usually sharply demarcated with no associated splash burns."

Cutaneous burns caused by sulfuric acid drain cleaner. Bond S, Schnier G, Sundine M et al. J Trauma 1998; 44: 523-526. Skin burns, eye injuries, and ingestions.

Child burn: accident, neglect, or abuse -- a case report. Leonardi WF, Vedovato JW, Werlang PM, et al., Burns 1999; 25: 69-71.

A decade of pediatric homicide: a retrospective study at the Medical university of South Carolina. Am J Forens Med P 1999; 20: 169-172. Burns were up to 25% of all child abuse cases.

Thermal injury and child abuse: the medical evidence dilemma. Dressler DP, Hozid JL (Harvard Medical School). J Burn Care Rehab 2001 Mar-Apr; 22(2): 180-185, discussion 179. From the abstract: This art reviews nine cases defended by the public defender in which the authors were involved. Illustrate some of the pitfalls in making the dx of child abuse caused by thermal injury. Biologic, engineering, and socioeconomic factors in eval. The devastating and long-lasting label of a false accusation or false imprisonment. Professionals with thermal injury expertise must become involved in the judicial process if justice is to prevail. This art is reviewed very well by Dr Reece in the January 2003 issue of *Child Abuse Quarterly*, including a chart of the features of all nine cases, pointing out that several of the "accidents" were due to children being left unsupervised (child neglect.) It is also cited with approval by Dr Sheridan in his review of Peck & Priolo-Kapel's art in the *Quarterly* for July 2003, see below.

Burns and scalds in pre-school children attending accident and emergency: accident or abuse? Bengner JR, McCabe SE, Emerg Med J 2001; 18: 172-174. (UK) Had ER residents use a four-item questionnaire to try and improve recognition of suspicious burns needing referral to an expert.

Curling iron-related injuries presenting to US emergency departments. Qazi K et al. Acad Emerg Med 2001; 8: 395-397. Found 82,000 injuries over five years from NEISS. 56% were from grabbing. Very few if any were dx'd as abuse. Ken Feldman (Child Abuse Quarterly, fall 2001) cautions that such a history in an infant under one year is suspicious, because reaching and grabbing is developmentally inappropriate.

Burn injuries caused by a hair-dryer: an unusual case of child abuse. Darok M, Reischle S. FSI 2001; 115(1,2): 143-146.

Scald abuse. Stratman E, Melski J. Arch Derm 2002 Mar; 138(3): 318-320.

Genital and perineal burns in children: 10 years of experience at a major burn center. Angel C, Shu T, French D, et al., J Pediatr Surg 2002; 37: 99-103.

Child abuse by burning: a review of the literature and an algorithm for medical investigations. Peck MD, Priolo-Kapel D. (UNC) J Trauma 2002 Nov; 53(5): 1013-1022. Algorithm for investigation to prevent false-positive and false-negative reporting was developed based on the authors' review of the literature and their own experience. Dr. Sheridan reviews this in the *Quarterly* and observes that the authors find that even in situations where the injury pattern does not match the parents' story, only 40% of the victims "are later found" [adjudicated?] to have been abused. He says the authors bring out the value of photography

and x-rays, and also a scene investigation. He says the authors “spend several paragraphs describing the importance of avoiding false-negative and false-positive accusations,” pointing out that such errors cause major problems. [Interesting how the burn situation parallels the CSA situation with respect to the need for historical information and the costs of error. JKR]

ASCP Check Sample No. FP 03-4 (FP-285). Kim Collins MD, S Erin Presnell MD, 2003. Case report of a 2 y o girl with a sharply demarcated 3d degree scald of the lower extremities up to the waist with groin sparing, Dxd as homicide because well demarcated and no splash burns. The warning signs of abusive burns are:

1. Burns in varying stages of healing
2. Other injuries
3. Ev of neglect or malnutrition
4. Prior hospitalizations, ER visits, or “accidents”
5. Delayed presentation
6. Burns appear older than stated history
7. Location incompatible with history (e.g. a burn on the back said to have come from a spill)
8. Someone other than the caretaker brings the victim to the hospital
9. A sibling is blamed
10. The victim is withdrawn and submissive
11. Signs of immersion (hands and feet or posterior buttocks)
12. Burns isolated to the buttocks and/or perineum

Approximately 1/3 of all abusive burns are contact burns. Cigarette, stove, or iron. Consider the developmental stage of the child in comparison to the history given. Scalds are often during the toilet training years. In adults, a scald can occur if the water temperature is over 127. Times to scald:

130	30 sec
140	3 sec
150	1 sec

Children’s skin will get a full-thickness scald in ¼ the time that adult skin takes. On exam, watch for

immersion line
spared areas (grasping areas)
satellite splashes

Limb withdrawal by the child can result in groin and popliteal sparing. Possible central buttock sparing if resting against the bottom of the tub. Glove and stocking patterns, possibly with palmar sparing due to clenching. Inflicted immersion burns are of uniform depth, while accidental immersion burns are of varying depth. Splash marks do not rule out inflicted, because a hot liquid could have been thrown at the child. With spills, the burn pattern goes from more severe to less severe downward as the liquid cools. Clothing may alter the burn pattern. Material may hold the hot liquid against the skin for longer.

Unusual inflicted hot oil burns in a 7-year-old. Mukadam S, Gilles E. Burns 2003; 29: 83-86. From the review by Dr Reece in the *Quarterly* for July 03: Grease burns are usually accidental. But this girl had 50 bite marks, plus 30 circular wounds inflicted with a metal spatula that was heated in hot oil and applied to the skin. “The inflicted nature of these lesions was suggested by the distribution of the lesions on the inner protected surfaces of the arms and legs, their clear demarcation, their extension onto different body surface planes [contours], their different ages of healing...” These are actually contact burns.

Accidental scald burns in sinks. Titus MO, Baxter AL, Starling SP. Pediatrics 2003; 111: e191-194. From Ken Feldman’s review in the *Quarterly* for July 03: Three 1 ½ year old toddlers with leg burns. They climbed into the sink and turned on the hot water. 140 degrees to 150 degrees. The burns were asymmetric with irregular depth and margins. There were no stocking patterns. Bilateral leg burns are

associated with abuse, but here abuse was ruled out. Required multidisciplinary investigation. Newer water heaters are supposed to be controlled at lower temperatures.

Into hot water head first. Distribution of intentional and unintentional immersion burns. Daria S, Sugar NF, Feldman KW, Boos SC, Denton SA, Ornstein A. *Pediatric Emergency Care* 2004; 20: 302-310. From the review by Dr Reece in the Autumn 2004 issue of the *Quarterly*: Review of the records of 195 scalded children under age 5, with detailed review of six who went in head or face first. Of the six head cases, five were facial, while one was back of head and upper body. Four had their face forced into hot water, one was forced into a faucet. As far as the others, only 5% of the 195 cases were found to be inflicted. Of the accidentals, 90% were caused by something other than tap water -- usually stovetop liquids. But there were some tap water immersions among the accidentals. The inflicted were more likely to have two patterns: (a) simultaneous burns of both legs, or (b) buttock burns.

Kitchen scalds and thermal burns in children five years and younger. Drago DA. *Pediatrics* 2005; 115: 10-16. Collected 704 cases from 100 hospitals. Most of the injuries occurred in one year-olds. Toddlers appear to understand that hot things are dangerous. Scalds were much more severe than thermal burns -- hospitalization rate of 11.7% versus 0.1%. Found that 74% of the scalds happened from child either pulling the pot off the stove, grabbing the pot, or splashing. Overall, there were 17,237 cases nationwide in a seven year period.

Children may be able to climb into bathtubs earlier than previously thought. Allasio D and Fischer H. *Pediatrics* 2005 May; 115: 1419-1421. These guys were actually studying the issue of scalds. They set up an (empty) bathtub with toy boats in it, and placed 176 toddlers aged 10 months to 18 months outside the tub. 35% successfully climbed into the bathtub, including one ten month-old who couldn't even walk.

Pediatric homicides related to burn injury: a retrospective review at the Medical University of South Carolina. Zaloga WF and Collins K. *JFS* 2006 Mar; 51(2): 396-399. Had 124 cases -- 121 fire and 3 scalds. Of these 124, had 108 accidents and 12 homicides and 4 undet. "Classifying the manner of death in burn fatalities can be extremely challenging." Both the accidents and the homicides were mostly house fires. "Inflicted burns usually have a characteristic pattern, which must be correlated with the given history. The inflicted lower body immersion scald type of burn has a distribution usually simultaneously of the buttocks and distal limbs, perineum and both feet with some sparing of the areas of flexion. Accidental immersion scald injuries are more common and do not have this pattern. (citing Purdue et al., *J Trauma* 1988). Abusive burns tend to be more severe, deeper and larger than accidental burns. Our review showed the classic pattern of immersion burn of the lower body in all three scald deaths." Discrepant history in scalds: the caregiver says the child burned himself, but it is a clear immersion burn. But in vehicle fires and house fires, there were no discrepant histories. As to groups at risk for homicide burns, it was 1-8 years, with a peak at 2. For scalds, it was the youngest ages -- 1 to 2 yrs. All fatal scalds were homicides. The majority of the perpetrators were female.

How good is the evidence available in child protection? Sibert JR, Maguire SA, Kemp AM. *BMJ* Jan 30 2007. Refuting a lead article by David Chadwick on 22 July 2006 stating that the published evidence base for child protection is robust. On the contrary, say these authors, who are part of an evidence-based medicine review team. One of the few things they find to be well established is the definition of inflicted scalds by pattern. See below under "Unclassifiable."

Non-accidental burns in children -- are we neglecting neglect? Chester DL, Jose RM, Aldiyami A, King H, Moiemmen NS. *Burns* 2006; 32: 222-228. (UK) Neglect apparently meant lack of supervision in these 440 children admitted to a burn unit. Had 4 inflicted burns and 41 due to neglect.

MSBP

(see also "SIDS v. Suffocation" and "ALTE")

See also the bibliography given in *Investigation and Prosecution of Child Abuse, 2d ed. (1993)* by the National Center for the Prosecution of Child Abuse.

Munchhausen syndrome by proxy: the hinterland of child abuse. Roy Meadow. *Lancet* 1977; 2: 343-345
The original article. Referred to later on in connection with the judicial and press treatment of Prof Meadow's evidence in the Angela Cannings and Sally Clark cases before the Criminal Case Review Commission and the Supreme Court of Judicature. See Lord Justice Kay's full opinion in *R v. Sally Clark*, Neutral Citation No. [2003] EWCA Crim 1020.

Pathology of fatal child abuse and neglect. Ross Zumwalt and Charles S. Hirsch. Chapter 13 In: Helfer RE and Kempe RS, eds, *The Battered Child, 4th Ed.* Chicago: University of Chicago Press, 1987, pp. 275-276. Defining MSBP as "when a parent or guardian falsifies a child's medical history or alters a child's laboratory test or actually causes an illness or injury in a child in order to gain medical attention for the child which may result in innumerable harmful hospital procedures. Examples of MBP include untruthful descriptions of symptoms, alteration of body fluids before laboratory testing, or by actually causing an illness or injury by poisoning or injuring the child."

Parental administration of chemical agents: a cause of apparent life-threatening events. Hickson G, Altemeier WA, Martin ED, Campbell PW. *Pediatrics* 1989; 83: 772-776.

Levin AV and Sheridan MS (eds.), *Munchhausen Syndrome by Proxy: Issues in Diagnosis and Treatment.* New York: Lexington Books, 1995.

Munchhausen syndrome by proxy: child abuse in the medical system. Donald T, Jureidini J. *Arch Ped Adol Med* 1996 Jul; 150(7): 753-758.

MSBP abuse perpetrated by men. Roy Meadow. *Arch Dis Child* 1998; 78: 210-216.
From Robert Reece's precis in *Child Abuse Quarterly*: Fifteen cases. The male perpetrators were engaged in acting out the need to assume a sick role by proxy or were engaged in another form of attention-getting behavior. In nine families, there were false seizures or apnea with smothering. ...In four homes there had been unusual fires and in four homes dogs or cats had died by poisoning.

Procedures, placement, and risks of further abuse after MSBP, nonaccidental poisoning, and non-accidental suffocation. Davis P, McClure RJ, Rolfe K, Chessman N, Pearson S, Sibert JR, Meadow R. *Arch Dis Child* 1998 Mar; 78(3): 217-221. A British study by questionnaires on 119 victims w 24 mos mean followup. 17% of the ones allowed to go home were reabused. (The more serious cases were usually not allowed to go home.) Siblings were abused: in 50% of the families with suffocation and in 40% of the families with poisoning, siblings were subsequently abused.

Practical Aspects of Munchhausen by Proxy and Munchhausen Syndrome Investigation. Kathryn Artingstall. Boca Raton: CRC Press, 1999.

Manifestations of MSBP in pediatric gastroenterology. De Ridder L and Hekstra JH. *J Pediatr Gastroe Nutr* 2000; 31: 208-211. Taken from Randy Alexander's review note in *Quarterly* for Jan 2001. Chronic diarrh, FTT, vom, abd pain hematem gastric erosions, Mallory-Weiss, colitis hematoch constip CF FB ingestion, cigarette burns of the mouth seen as aphthous ulcers.

Munchhausen syndrome by proxy. Donna Andrea Rosenberg. Chapter 14 in: Reece RM and Ludwig S, eds., *Child Abuse: Medical Diagnosis and Management, 2d ed.* Philadelphia: Lipincott Williams & Wilkins, 2001, pp. 363-383

Munchhausen syndrome by proxy: the ultimate betrayal. Dawn Doran Wilsey, JD. *APRI Update* 2001 Aug; 14(8). (have) Noting that as an adult psychiatric disorder DSM IV does not recognize "factitious disorder by proxy" as a recognized mental disorder, but states that it is a "category in need of further study." There is no objective psychological test that detects it. But as a *pediatric* diagnosis and as a crime

it is “generally accepted,” as cases have held. Proving the corpus delicti requires expert T bc it is not within the common knowledge of jurors.

Fabricated or induced illness In children (editorial) Wilson RG. BMJ 2001 Aug; 323: 296-297.

Munchausen syndrome by proxy: tall tales and real hurts. David Paulk, MS, PA-C. Clinician Reviews 2001 Sep; 11(8): 51-56. (have) A review article. “The crucial factor is intent to deceive.” The DSM-IV criteria are:

Intentional production or feigning of physical or psychological signs or sx in a person under the perpetrator’s care

Intent of the perpetrator to assume the sick role by proxy

Absence of external incentives for the behavior (e.g. economic gain)

Absence of another mental disorder to account for the behavior

Article gives Table 1 (classic characteristics of MSBP) and Table 2 (the most commonly seen child abuses in MSBP), which I will not reproduce here.

Bleeding ears: a case of MSBP. Griffiths H, Cuddihy PJ, Marnane C. Int J Pediatr 2001; 57: 245-247. (UK) Blood and pus repeatedly from both ears, also anemia and FTT.

Fabricated or induced illness in children. (editorial) Wilson RG. BMJ 2001; 323: 296-297. Recent literature update.

Considering suffocatory abuse and Munchhausen syndrome by proxy in the evaluation of children experiencing apparent life-threatening events and sudden infant death syndrome. Truman TL, Ayoub CC. Child Maltreat 2002; 7: 138-148. See Dr Reece’s review in *Child Abuse Quarterly* for July 2002. Dr Reece summarizes that this retrospective chart review of 2,755 pts over 2 days and under 4 yrs with *multiple admissions* for SIDS, ALTE, bradycardia, cyanosis, apnea, suffoc, etc. Excl pts with known diseases and those with only a single admission. Remained 138 pts. Avg 7 mos. 9% of the 138 records documented “significant social problems” such as DV, marital strife, and criminal activity in the home. Maternal psychiatry in 9%.

103 survived; 35 expired.

35 pts who expired (25%): 5 of the deaths (14%) had inflicted injuries. In 32% of the autopsies there was a specific cause of death found (i.e. SIDS was ruled out); that leaves 68% of the deaths in which no cause of death was found. But the authors considered that 37% of the deaths were suspicious for non-accidental injury.

Abuse: total 14. 5 died, 9 survived, of which eight abuse survivors were in the high-risk group for abuse (see below.)

The 103 survivors were stratified into high, moderate, and low risk groups for child abuse based on [?]. [I have inserted this “?” here because I am concerned about whether the risk criteria were known (i.e. independently established from the literature) risk factors or risk factors purely from within the data of this study, in which case I am concerned that the reasoning could be circular. –JKR]

34 high risk were older (avg 12 mos, some over 22 mos). Had an average of 24 ALTEs, in 76 pts with only by the mother. 38% had siblings with ALTE or SIDS. 26% had blood from nose or mouth. 8 abuses.

31 moderate risk avg 7 mos had up to 30 ALTE or SIDS, 26% with only one parent. Mostly no bleeding. Only one abuse.

37 low risk avg 3 mos only 1 ALTE, no bleeding. No definite abuses.

Dr Reece summarizes that the authors identified five major factors to be considered by clinicians in evaluating a child with multiple ALTE’s:

1. Recurrent, poorly explained ALTE's
2. The same caregiver is the witness to most of the ALTE's
3. Blood in the mouth or nose
4. Bruising inconsistent with resuscitation
5. Siblings with other "medical problems." (Especially SIDS or ALTE).

Dr Reece comments that the dx was often missed because it wasn't considered.

Different interpretations of MBP. Roy Meadow. Child Abuse & Neglect 2002; 26: 501-508. From the summary by Randy Alexander in the *Quarterly*: The different interpretations are those of defining it by

The characteristics of the perpetrator (psychiatry aspect)
The situation of the victim (pediatrics aspect)

Sir Roy warns that a purely victim-centered approach runs the risk of confusing MBP with other forms of serial murder by caregivers, such as poisoning and serial suffocations. He propounds the following diagnostic criteria for MBP:

1. The illness was fabricated, i.e. faked or induced, by the caregiver
2. The caregiver denies this.
3. The illness goes away when the child is separated from the caregiver.
4. The perpetrator is diagnosed as acting out of a need to assume the sick role by proxy or as attention-seeking behavior of some other subtype.

Dr Alexander refers us to a special issue of Child Maltreatment, in which a national task force has distinguished two disorders:

Pediatric condition falsification (Meadow's criteria 1,2,3)
Factitious disorder by proxy (Meadow's criterion 4)

Comment by JKR: Dr Meadow is a lump; Dr Alexander is a splitter. The advantage of the splitting approach medicolegally is that it allows intervention and testimony on behalf of the child in cases where the motives or even the identity of the perpetrator remain unclear.

Position paper: definitional issues in Munchhausen by proxy. Ayoub CC, Alexander R, Beck D, Bursch B, Feldman KW, Libow J, Sanders MJ, Schreier HA, Yorker B. (Harvard). APSAC Taskforce on MBP, Definitions Working Group. Child Maltreatment 2002 May; 7(2): 105-111.

Prevalence of retinal hemorrhages and child abuse in children who present with an apparent life-threatening event. Pitetti RD, Maffei F, Chang K, Hickey R, Berger R, Pierce MC. Pediatrics 2002 Sep; 110(3): 557-562. Prospective ER study of 128 children under 24 months admitted to ER with ALTE. Known child abuse cases were specifically excluded. 73 had dilated funduscopy. Found three cases of child abuse. One (1) patient had RH. This was a 6-week-old presenting with apnea had bilateral RH + bilateral SDH + healing rib fractures, femur fracture, tibia fracture, skull fracture, arm fractures, confession of "shaking." The other two child abuse cases were a two-week-old with acute & chronic SDH (??), and a six-month-old with multiple ALTEs and observed mother attempting to smother him. The point is that doing a full child-abuse workup in this setting had a diagnostic yield of 2% (3/128) and doing dilated ophthalmoscopy on ALTEs had a diagnostic yield of 1/73 funduscopies or 1%. The authors consider this significant enough to recommend doing these routinely.

MSBP: medical diagnostic criteria. Rosenberg DA. Child Abuse & Neglect 2003; 27: 421-430. The "definitive" diagnostic criteria are: (A) covert video surveillance, or (B) the exclusion of all other causes for the child's condition.

MSBP: an extreme form of child abuse with a special forensic challenge. Bartsch C, Risse M, Schutz H, Weigand N. (Germany) FSI 2003; 137: 147-151. Case report of two cases. One where a child presented ten times with weakness and paralysis ultimately found to be due to clozapine intoxication. A younger sister had earlier died after multiple admissions with the same symptoms. That child was exhumed and found to have clozapine in the hair samples. Mother confessed. Other case a 30 month male with two admissions one year apart for clonidine intoxication.

Suffocation and poisoning -- the hard-hitting side of Munchausen syndrome by proxy. Vennemann B, Bajanowski B, Karger B et al. Int J Leg Med 2005; 119: 98-102 (Ger) Reports five families. Four used medication overdosing. One used suffocation. It was always the mother.

MSBP caused by ipecac poisoning (case report). Carter KE, Izsak E, Marlow J. Pediatric Emergency Care 2006; 22: 655-666. A 9 year old boy. Repetitive use causes tissue buildup leading to cardiomyopathy, rhabdomyolysis.

MEDEA COMPLEX

(see also "Domestic Violence," below)

The "Medea complex" among men: the instrumental abuse of children to injure wives. McCloskey LA. Violence Vict 2001; 16(1): 19-37. "Children of battered women stood a 42% chance of receiving escalated abuse from their fathers. It is proposed that men's abuse of children is in many instances instrumental in order to coerce or retaliate against women, echoing the Greek myth of Medea who killed her own children to spite their father." Heavy drinking (odds ratio 4.86) and life stress events (odds ratio 1.6) predicted men's abuse of their partners, but these risk factors were unrelated to child abuse. Wife battering, however, placed children at heightened risk (2.77).

The impossibility of forgiveness: shame fantasies as instigators of vengefulness in Euripides' Medea. Lansky MR. J Am Psychoanal Assoc 2005; 53(2): 437-464. (UCLA) Three types of shame fantasy are pertinent to the transformation of Medea's mental state from one of anguished and disjointed shame to diabolical vengefulness: (a) anticipatory paranoid shame, (b) the projective identification of shame, and (c) withdrawal as a defense against shame.

MCAD

(see also SUDC, below)

See also Glutaric Aciduria Type II, below.

List of local genetic centers in Appendix "J" to the State SIDS Protocol Manual, State of California, Department of Health Services, Revised Standardized Autopsy Protocol and Death Scene Investigation/Deputy Coroner Protocol, 1992. (large blue ring binder).

Fatty liver in sudden childhood death. Bonnell H and Beckwith B. Am J Dis Chil 1986 Jan; 140(1): 30-33. Studied that autopsy livers of 21 chil who died suddenly either by trauma or by natural causes, from the point of view of asking whether fatty liver was diagnostic of Reye's syndrome. Found that many of the chil had fatty livers. None of them had Reye's syndrome. Argues that fatty liver is ubiquitous in children.

The postmortem recognition of fatty acid oxidation disorders. Bennett MJ, Hale DE, Coates PM, Stanley CA, (Children's Hospital of Philadelphia) Behavioral Pathology 1991; 11: 565-570. Reprinted as Appendix "T" to the State SIDS manual. Look for **pale or white organs**, indicating fat accumulation in heart, kidney, muscle, and liver. Each disorder can have three forms: hepatic form, myopathic form, and

sudden death form. The forms can overlap. The hepatic form causes fasting hypoglycemia, Reye's syndrome-like picture, hepatomegaly, and steatosis. The myopathic form causes cardiac and systemic myopathy and muscle lipid storage. The sudden death form can occur at any age. *Urine* is the best sample (it's the one used first in living patients); it will contain dicarboxylic acids, and in MCAD it will contain acylglycine and acylcarnitine. *Swabbing the bladder wall* will provide adequate material for this test. Freeze the swab at -20. *Blood* is OK; they would prefer to have it anticoagulated and centrifuged and the plasma and cells separately frozen. Failing that, they can accept frozen whole blood. *Tissue* such as liver can be assayed for tissue enzyme activity if stored at -70, but have to take account of the postmortem loss of tissue enzyme activity which is progressive with time after death; hence the postmortem interval needs to be recorded. *Skin* frozen at -70 can be used to culture fibroblasts for confirmatory test. Here are the disease entities that they list (as of 1990):

- LCAD
- MCAD
- SCAD
- LCHAD (hydroxyacyl)
- ETF (electron-transfer flavoprotein)
- ETFD (electron-transfer flavoprotein dehydrogenase)
- CT (carnitine transporter)
- CPT1 (carnitine palmitoyl transferase 1)
- CPT 2 (carnitine palmitoyl transferase 2)

The changing face of disorders of fatty acid oxidation. Vockley J. Mayo Clin Proc 1994; 69: 249-257
Cited in Sturmer, *infra*.

Sudden, unexpected death in infants versus the sudden infant death syndrome (editorial). Zumwalt RE. Human Pathology 1994 Aug; 25(8): 733-734. There should be universal screening for MCAD now that we know it is a significant cause of SIDS. But there also needs to be a sharp upgrading of the infant death investigation protocol.

Metabolic causes of sudden and unexpected death in early life. Piero Rinaldo, MD. 1997. Extremely useful article by Yale professor. See below under "SIDS Biology."

Retrospective biochemical screening of fatty acid oxidation disorders in postmortem livers of 418 cases of sudden death in the first year of life. Boles R, Buck E, Blitzer M. J Pediatr 1998 Jun; 132(6): 924-933. Cited by MMWR 2003 for the proposition that "Approximately 5% of sudden infant deaths might be associated with metabolic diseases."

SIDS and disorders of fatty acid oxidation: where do we go from here? (Editorial)
Journal of Pediatrics 1998 Jun; 132(6): 913-914. Referring to Boles, Buck, Blitzer in the same issue. See also Neo-Gen memorandum July 1998 and a later written comment on this paper in J Peds by Neo-Gen. This editorial advocates a "splitter's" approach to true SIDS, "...it has become clear that SIDS is a syndrome rather than a single disorder, and probably has different causes..."

Fatty liver in sudden infant death autopsies. Sawaguchi T and Nishida H. (letter). Am J Forens Med Path 1998 Jun; 19(3): 294-295. These scientists point out that just screening blood for dodecanoic acid is inadequate because it only picks up MCAD. They point out that SCAD and LCAD also have significant incidence. Especially if the liver is fatty. In SCAD look for butyrylglycine and butyrylcarnitine. In LCAD look for C14-C18 monounsaturated (alkenoic) acids in liver. In a Japanese SIDS series, 13% of the babies had fatty livers. Some were + for dodecanoic, some not.

Dissecting the spectrum of fatty acid oxidation disorders. Charles A. Stanley. Journal of Pediatrics 1998 Mar; 132(3 pt 1): 384-386. The clinical man of these dsos is widely variable. The most acute is the "hepatic" presentation char by acute, life-threatening coma precipitated by fasting. Can look like Reye's or

SIDS. The second or "cardiac" presentation is with dilated or hypertrophic cardiomyopathy. The third is the acute or chronic "muscle" presentation seen in adults. Renal cysts may be seen in some forms.

Retrospective biochemical screening of fatty acid oxidation disorders in postmortem livers of 418 cases of sudden death in the first year of life. Boles G, Buck EA, Blitzer MG et al. Journal of Pediatrics 1998 Jun; 132(6): 924-933. From a previous study of 27 children who died of known inborn errors, they developed morphologic screening criteria on liver for possible inborn error: microvesicular steatosis, glucose depletion, elevated liver levels of unsaturated long chain fatty acids, and elevated liver carnitine. They screened the livers of 418 sudden deaths in infancy, including accidents, for these criteria. Of these deaths, 313 were SIDS. Of these, 14 livers (4%) had two of the criteria positive. Some of the non-SIDS deaths also were positive.

Sudden and unexpected neonatal death: a protocol for the postmortem diagnosis of fatty acid oxidation disorders. Rinaldo P, Yoon HR, Yu C, Raymond K, Tiozzo C, Giordano G. Semin Perin 1999 Apr; 23(2): 204-210. Identified 44 cases in five years: 13 neonates. The postmortem identification of these cases "remains difficult." Found that sudden neonatal death "was consistently associated with exclusive breast feeding and presumably poor caloric intake. The dx was based on the analysis of postmortem liver and bile. Liver showed microvesicular steatosis, elevated fatty acid conc, glucose depletion, and low carnitine conc. Bile carnitine analysis and acylcarnitine profiling work. Cord blood spots are also usable.

Fatty liver in sudden infant death autopsies. (letter). Sawaguchi T, Nishida H. Am J Forens Med Path 1998 Oct; 19(3): 294-295. They observed fatty liver in 13% of a 10-year series of SIDS autopsies. They screened for dodecanoic acid. But this missed the cases of SCAD and LCAD. For SCAD look for n-butyrylglycine in urine and blood. For LCAD look for elevated C14-C18 monounsaturated fatty acids in liver.

The metabolic autopsy comes of age. Bennett MJ, Rinaldo P. Clinical Chemistry 2001; 47: 1145-1146

Molecular autopsy of sudden unexplained death in the young. Ackerman MJ, Tester DJ, Driscoll DJ. Am J Forens Med P 2001; 22(2): 105-111

Epinephrine-induced QT interval prolongation: a gene-specific paradoxical response in congenital long QT syndrome. Ackerman MJ, Khosiseh A, Tester DJ, Hejlik J, Shen WK, Porter CJ. Mayo Clin Proc 2002; 77: 413-421.

Fatty acid oxidation disorders. Rinaldo P, Matern D, Bennett MJ. Ann Rev Physiol 2002; 64: 16-26

Postmortem screening for fatty acid oxidation disorders by analysis of Guthrie cards with tandem mass spectrometry in sudden unexpected death in infancy. Wilcox RL, Nelson CC, Stenzel P, Steiner RD. J Pediatr 2002 Dec; 141(6): 833-836. Acylcarnitine analysis of cord blood spots. In the first five years of the study, three cases (1.2% of Oregon's 247 cases) of SIDS were found to have fatty acid oxidation disorders by this method -- two with MCAD and one with VLCAD.

Contribution of selected metabolic diseases to early childhood deaths. MMWR 52(29): 677-679, 2003. Population-based study by the Virginia OCME of 793 children under age 3 who had sudden unexpected natural deaths. Of these eight (8) had positive postmortem *Neo Gen* screening for metabolic diseases by tandem MS. That would be 1%.

- 4 fatty acid oxidation disorders
 - 2 had + molecular testing for the G985A mutation seen in MCAD
- 4 possible organic acidemias

The authors suggest that routine newborn screening for these diseases would be worthwhile. The editorial note says that "Approximately 5% of sudden infant deaths might be associated with metabolic diseases." (citing Boles, Buck, Blitzer, 1998).

The Organic Acidemia Association. <http://www.oaanews.org>

National Newborn Screening & Genetics Resource Center. <http://genes-r-us.uthscsa.edu/>

SUDC (Sudden **Unexplained** Death in Childhood. Note that it has to remain unexplained after full autopsy, etc. The definition parallels that of SIDS.) 1:100,000 LB

“The sudden death of a child greater than 1 year of age that remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene, and review of the clinical history.” SUDC Program of the CJ Foundation. Sudden Unexpected Death in Childhood. Children beyond the age of 12 months. 1:100,000. <http://www.sudc.org> e-mail info@sudc.org Laura Crandall, Program director, 800-620-SUDC. Laura@sudc.org Hackensack, NJ. Research project under Dr Krous at Childrens Hospital of San Diego

Sudden Death in Infancy, Childhood, and Adolescence, Second edition. Roger W Byard. West Nyack, NY: Cambridge University Press, 2004. <http://www.cambridge.org> ISBN 0 521 82582 2 Attn: Order Dept, Cambridge University Press, 100 Brook Hill Drive, West Nyack, NY 10994.

Long QT syndrome and other channelopathies -- testing for. FAMILION test. <http://www.familion.com> Genaissance Pharmaceuticals, Five Science Park, New Haven, CT 06511. 203-773-1450. <http://www.genaissance.com>

See also the Death Genomics Laboratory at the Mayo Clinic, Michael J. Ackerman, MD, 507-284-0101, ackerman.michael@mayo.edu See below under NAME meeting 2005.

Sudden Arrhythmia Death Syndrome: <http://www.sads.org> See also under “Drown,” below. (Swimming induces tachyarrhythmias such as VT and *les torsades de pointes* in people with type 1 long QT syndrome.)

Cardiac Arrhythmia Research and Education Foundation (CARE), 2082 Michelson Drive, Irvine CA 92612 800-404-9500 <http://www.longqt.org>

Dr Krous suggests the following tests for metabolic disease in the family of a decedent:

Prenatal: watch out for acute fatty liver of pregnancy (AFLP), hemolysis, elevated liver enzymes, low platelets syndrome (HELLP), preeclampsia.

Asymptomatic newborn: request blood spots to be made for private lab testing by tandem MS. also urine for organic acids

Symptomatic newborn: ABG's electrolytes, serum lactate, pyruvate, ammonia, glucose

Older siblings: PLASMA acylcarnitines, urinary organic acids (he capitalizes “plasma.”

And the following for cardiac testing of siblings of a decedent:

Newborn ECG must be repeated at > 2 weeks of life because it is often false-positive in the early newborn period. Try the epinephrine stress test, because the work of Ackerman et al has revealed that epinephrine can paradoxically bring out the Long QT syndrome.

The metabolic autopsy comes of age. Bennett MJ, Rinaldo P. Clinical Chemistry 2001; 47: 1145-1146

Molecular autopsy of sudden unexplained death in the young. Ackerman MJ, Tester DJ, Driscoll DJ. Am J Forens Med P 2001; 22(2): 105-111

Epinephrine-induced QT interval prolongation: a gene-specific paradoxical response in congenital long QT syndrome. Ackerman MJ, Khosiseh A, Tester DJ, Hejlik J, Shen WK, Porter CJ. Mayo clin Proc 2002; 77: 413-421.

Fatty acid oxidation disorders. Rinaldo P, Matern D, Bennett MJ. Ann Rev Physiol 2002; 64: 16-26

Sudden unexpected death in childhood: a report of 50 cases. Krous HF, Chadwick AE, Crandall L, Nadeau-Manning JM. (San Diego). Pediatric and Developmental Pathology 2005 Jul; 8: 307-319. Treating upon SUDC in this *Festschrift* for J Bruce Beckwith MD. Cases collected via their web site at <http://www.SUDC.org> has a US incidence of ~1.5/100,000 live births, compared with 56 for SIDS, as of 2001. Most are 1 to 3 years old, male, found prone, often having a personal or family history of febrile seizures. Occasionally have a family history of SIDS or SUDS. Extensive histologic sampling of the brain is critical. Retain tissue from the areas vital to life, such as heart and brain stem. Ancillary testing is to be considered.

Review the literature on SUDC. A Swedish study (Molander, 1982) had only four pediatric cases and found rarity, 7/100,000 LB. A UK study (Southall et al., 1987) found a higher incidence of 12.5/100,000 LB. Another UK study (Keeling et al., 1989) found 11 cases in the 2-20 age range. A Pennsylvania study (Neuspiel et al., 1985) found 15 unexplained pediatric deaths out of 62 children who died unexpectedly. A US SIDS epidemiology study (Norman et al., 1990) found 16 unexplained sudden deaths of 1 year-olds. A Canadian study (Helweg-Larsen et al., 1993) found two toddlers with SIDSlike deaths, classified as "late SIDS." Overall, the present authors find that the very small numbers and lack of depth of detail in these scattered reports are unsuitable for epidemiologic or definitional purposes. the authors tabulate the reported incidence of unknown COD in the US and California for the roughly 20-year period 1979-2001. Have:

<u>Area</u>	<u>Age</u>	<u>Deaths</u>
US	1-4	5,319
	5-9	1,195
	10-14	1,402
Calif	1-4	638
	5-9	163
	10-14	213

Their case material had 14 cases of sudden unexpected death that were explained, and 36 cases of sudden unexplained death (SUDC). The 14 "explained" sudden deaths were due to accidental asphyxia, sepsis, myocarditis, MCAD, infection, ARVD, encephalitis, overdose, and Undet. For the 36 true SUDC cases, have age range 1 to 12 years with a median of 1.6 years. 64% male. 78% white. 29 term and 2 preterm. 33% of cases and 33% of their family members had a history of a seizure, often associated with a fever. 11% had a family history of SIDS. 22% were retarded children. 26% had a history of minor head trauma within two weeks of death. 33 cases were asleep when they died. 26 cases were found prone. Only 4 were cosleeping. Most were within normal growth parameters, with an average weight percentile of 32d percentile, but there were 3 <5th and 4 >95th. Basically the same for length. 11 were frankly obese with a weight-by-length over 95th percentile. 15/36 had intrathoracic petechiae.

The authors discuss genetic disorders that routine ancillary testing will not currently identify, including Long QT syndrome; they consider these to be possible causes of death in their material, but testing was not available. They note the positive family history of SIDS in some cases as suggestive of a possible genetic disease.

Where do we go from here? A multicenter study.

Hippocampal anomalies in sudden unexplained death in childhood (SUDC): report of a series. 2005 Kinney HC, Armstrong DL, Chadwick AE, Crandall L, Hilbert C, Krous H. (unpublished abstract

distributed at the 2005 NAME convention). SUDC about 175 cases a year. We found hippocampal anomalies in 8 out of 12 SUDC toddler cases in which hippocampal microscopic sections were available. The 8 cases all had sleep-related sudden death and were found in the prone position. 43% (3/8) had a history of febrile seizures. 2/8 had a family history of febrile seizures. Hippocampal anomalies included: asymmetry between the right and left sides, hyperconvoluted dentate, subicular defects, granule cell heterotopia, hamartia, and numerous ectopic neurons. Nine control cases showed microdysgenetic features in 33% (3/9) controls compared to 67% (8/12) SUDC cases. Gross hippocampal asymmetry was found in 0 controls versus 50% (4/8) of cases. Hypothesis: SUDC in toddlers is an unwitness seizure triggered by fever, head trauma, or unknown factors. Hannah.kinney@childrens.harvard.edu See also Hannah Kinney's 2005 case report of a SIDS case with serotergic brainstem abnormalities, below under "SIDS biology."

Postmortem cardiac channel genetic testing in sudden infant death syndrome, sudden unexplained death syndrome, and unexplained drowning. Michael Ackerman MD Mayo Clinic. NAME Meeting 2005. Comprehensive mutational analysis of all 60 protein-encoding exons of IKCNQ1 (LQT1), KCNH2 (LQT2), SCN5A (LQT3), KCNE1 (LQT5), and KCNE2 (LQT6) ... has been completed on 93 SIDS and 49 SUDS and 11 unexplained drownings. Of the 49 SUDS, 13 had a family history of syncope, cardiac arrest, or sudden cardiac death. Their average age at death was 14 years. Results: Pathogenic mutations have been identified for 4/93 SIDS (4.3%), **15/49 cases of SUDS (31%)**, and 3/11 cases of drowning (27%). Conclusion: Postmortem cardiac channel genetic testing should be considered as a standard part of the evaluation of autopsy negative sudden unexplained death.

Molecular autopsy of sudden cardiac death (SCD): The challenge of forensic pathologist to the complexity of genomics. (letter) Oliva A, Pascali VL, Hong K, Brugada R. Am J Forens Med P 2005 Dec; 26(4): 369. Says that the genomics of SCD syndromes is extremely complex and expensive to assay. Both coding and noncoding areas of the genome are significant, and there are many variants. Not only that, but the possible interaction of other bodily conditions and states with these genes and gene products has to be considered. ("The rich and diverse nature of genotype-phenotype associations in these conditions has recently been revealed.") The interpretation of results is difficult. Also, "Molecular testing is an extremely high-cost proposition, and forensic laboratories are typically funded by public sources." Ends by saying, in substance, that forensic pathologists of the future will have to be politically active in order to obtain funding for this.

Cites Jervell, Am Heart J, 1957 as the original source for the heritability of the long-QT syndrome. Cites the original article on Brugada's syndrome, Brugada et al., 1992. Cites DiPaolo et al., Am J Forens Med P 2004 for the proposition that long-QT syndrome should be considered in all cases of SCD where autopsy is negative. Cites Arking et al, 2004 for at least seven cardiac sarcolemmal, sodium, potassium, and calcium ion channel subunit genes, namely

KV-LQT-1 [KCNQ1]
HERG [KCNH2]
SCN5A
minK [KCNE1]
RYR2,
MiRP1 [KCNE2]
Kir2.1 [KCNJ2]

Cites Pietila et al., 2002, Splawski et al., *Science* 2002, and Ackerman et al., 2003 for the proposition that not only frank mutations but also common sequence variants (polymorphisms) have been implicated as risk factors that have to be considered. Cites Zhang et al., 2005 and Hong et al., 2005 for the proposition that noncoding regions and other things such as accessory proteins of the ion channels are responsible for arrhythmia syndromes.

Pathology of the Heart and Sudden Death in Forensic Medicine. Fineschi V, Baroldi G, Silver M. CRC Press, 2006. 536 pp. <http://www.crcpress.com>

SIDS VS. SUFFOCATION

Crib deaths: their possible relationship to postpartum depression and infanticide. Asch SS. Mount Sinai J Med 1968; 35: 214-220. Cited by Kukull & Peterson, 1977, as proposing that “the majority” of SIDS cases are infanticide. This article by a psychiatrist consists of informed speculation without data. But Reece & Krous relate its conclusions to other literature on maternal history of abusive childhood, postpartum depression, and infanticide. See Reece & Ludwig, 2d ed. (2001), p. 530. Cited by Emery, 1985, on postpartum depression probable role in filicidal cot death.

Accidental asphyxial deaths involving infants and young children. Sturmer WQ, Spruill FG, Smith Ra, Lene WJ. JFS 1976 Jul; 21(3): 483-487.

Asphyxial crib death. Bass M. NE J Med 1977; 296: 555-556. Cited by Reece & Krous (textbook, 2001) as 15 infant deaths from accidental asphyxia in Wayne County Michigan. Entrapments and hangings. Reece & Krous relate that Bass was criticized for failing to consider homicide in the hangings.

Sudden infant death and infanticide. Kukull WA, Peterson DR. Am J Epidemiol. 1977; 106: 485-486. From the abstract: “The hypothesis, advanced by Asch (1968), that a majority of SIDS cases are actually infanticides, is addressed by examination of age-comparable infant homicide rates and consideration of current theory regarding SIDS pathogenesis.” [Apparently making the point that if “the majority” of SIDS deaths were infanticides, since SIDS is so common this would be far out of line with the number of infanticides known to occur by other means. Cited by the AAP, 2001 (infra) for the proposition that 1% to 5% of SIDS are infanticide.

Two-year study of the causes of post-perinatal infant deaths classified in terms of preventability. Taylor EM, Emery JL. Arch Dis Chil 1982; 57: 668-673. Cited by Emery in his editorial on filicide, 1985, as the source for his view that filicide is about 10% of unexplained unexpected postneonatal infant deaths. (Unexplained meaning without obvious trauma, poisoning, or accident).

Two siblings with recurrent cardiorespiratory arrest: Munchausen syndrome by proxy or child abuse? Rosen CL, Frost JD, Bricker T, Tarnow JD, Gillette PC, Dunlavy S. Pediatrics. 1983; 71: 715-720

1982 Aviemore meeting and the gently battered child. Emery JL. Arch Dis Chil 1983; 58: 75-80. Discussed in Emery, 1993, as a point where “It was suggested that ‘gentle battering,’ i.e., a physical act that leaves no mark such as a hand or pillow over the face or putting the child face-down on a pillow or soft mattress, should be considered.”

Dead children from problem families in NE Wiltshire. Oliver JE. BMJ 1983; 286: 115-117. Cited by Emery, 1993, for the proposition that, like Mary Newlands (1991ff), Oliver found that siblings of child abuse victims were at dramatically increased risk for SIDS. (4%)

Infanticide, filicide, and cot deaths. Emery JL (Sheffield). Arch Dis Child 1985; 60: 505-507. This is an editorial. Cited by Meadow, *infra*, for the proposition that less than 10% of SIDS are suffocations if fully investigated. Cited by Stanton, 2003, for the proposition that “In 1985 Emery estimated the incidence of SIDS being filicide...as 2–10%.” Quoted by Reece & Krous, 2001 (textbook) as saying, “Filicide is the probable mechanism of death in approximately one in ten of the unexplained unexplained deaths.” Emery begins by reviewing the history of filicide (killing of child by parents) and infanticide (killing of infant by mother). Cases of filicide fall into two groups -- newborn and later. It is in the later cases that the differential of SIDS becomes important. There are four main causes of death: accidents, poisonings, trauma, and “gentle battering,” -- this last implying no traumatic lesions seen. Mild illness may be seen, but it doesn’t change the diagnosis: “If parents are at the end of their tether psychologically, a mild illness in the child makes him more irritable and therefore more likely to produce a final, emotional, parental

crisis.” (p. 505-506) The diagnosis requires much more than an autopsy; it requires a polyfactorial approach including the biochemical state of the child and a psychosocial evaluation of the family. Confessions have limited value; “people make confessions because they feel guilty, not because of what they did.” (p. 506) History of SIDS as a diagnosis: “a group in Seattle” came up with this concept to categorise the fact that many of the deaths were realized to be natural. Which is true, but the concept became an excuse for not doing any forensic investigation. “The development of the concept of SIDS as a natural disease was not based upon any firm evidence, and thus the possibility of most of these deaths being due to filicide remained.” How common is filicide in SIDS? Dr Emery makes the claim that in Sheffield the cot death investigation is as thorough as anywhere in the world. This has “led us to believe that in Sheffield, filicide is the probable mechanism in death in approximately one in 10 of the unexplained, unexpected deaths.” [Note, not just the repeat deaths, but the first-instance SIDS deaths. –JKR] (citing Taylor & Emery, Two-year study, 1982.) [The term “unexplained” here means without accident, poisoning, or trauma. –JKR] But he acknowledges that the incidence could vary geographically and over time; thus, “[A]s a working hypothesis, I would suggest that the figures for filicide as a major factor in unexplained cot deaths are between one in 10, and one in 50.” (p. 507) “But we can now say with much greater certainty than ever before that more than nine of 10 cot deaths are *not* due to filicide...” (p. 507)(emphasis in original --JKR) What can we do about cases of suspected filicide? These people need support; support can prevent a recurrence. But the legal system prevents us from getting close to them. Towards prevention: Consider the possible involvement of postpartum depression. The period of cot death largely coincides with that of postpartum depression. We should study this.

Should child abuse and neglect be considered when a child dies unexpectedly? Christoffel KK, Zieserl EJ, Chiaramonte J. Am J Dis Chil 1985; 139: 876-880. Summarized by Reece & Krous in textbook (2001) to the effect that these authors reviewed 43 unexpected child deaths admitted to Children’s Memorial Hospital in Chicago (not just infants). Nine of the 43 cases were found by autopsy to be child abuse. But Reece & Krous point out that of the six infants who were in the SIDS age group, all six turned out at autopsy to be SIDS.

The mistaken diagnosis of child abuse: a form of medical abuse. Kirschner RH and Stein RJ. Am J Dis Chil 1985; 139: 873-875. Presents 10 cases of erroneous diagnosis of child abuse, mostly made in the ER. 5 turned out to be SIDS. In some there was serosanguinous drainage from the nose and mouth, a common finding in SIDS. Others were other common postmortem findings such as Mongolian spot, livor mortis, dilated anal sphincter.

Interstitial haemosiderin in the lungs of SIDS: a histological hallmark of ‘near-miss’ episodes? Stewart S, Fawcett J, Jacobson W. J Pathol 1985; 145: 53-58. Compare Jackson & Gilliland’s 2000 reverse autopsy study of 206 children finding hemosiderin in 11% without a clear cause, no particular association with SIDS.

Investigation of SIDS. Emery JL and Taylor EM. NE J Med 1986; 315: 1676. Cited by Reece & Krous (textbook, 2001, p. 530): “Accidental suffocation was thought to be the cause of death in 10% of these cases, and the possibility of active intervention by one or both parents was raised in another 10%, a rate consistently double that of overt child abuse in this age group.” See Stanton, 2003. See Emery, 1985, 1993, Waite & Emery, 1996, Reece, 2001, Carpenter et al., 2005, Gornall, 2006.

Death scene investigation in sudden infant death. Bass M, Krovath RE, Glass L. NE J Med 1986; 315: 100-105. The authors surreptitiously reviewed 26 SIDS cases diagnosed by the New York ME’s office, and independently performed after-the-fact death scene investigations on them. Found one definite overlaying, five possible overlayings, three hyperthermia/asphyxias, and one shaken baby. Reece & Krous (textbook, 2001) say that this article lacked scientific method, lacked any controls, and overinterpreted the data. Nonetheless, the point was made that the New York ME wasn’t doing scene investigations.

Homicide as a cause of the sudden infant death syndrome. Cashell AW. Am J Forens Med P 1987; 8: 256-258. Cited along with Emery & Taylor, *supra*, by Krous, 2002 for the proposition that “Certain investigators have suggested that some SIDS deaths, with estimates as high as 20% of all SIDS cases, are undetected homicides.”

Nonnatural death masquerading as SIDS. Perrot LJ, Nawojczyk S. Am J Forens Med Path 1988; 9: 105-111. Of 170 deaths, 60% were SIDS, 35% specific natural causes, and 5% abuse, neglect, or questionable circumstances.

Intrathoracic petechial hemorrhages: a clue to the mechanism of death in sudden infant death syndrome? Beckwith JB. Ann N Y Acad Sci 1988; 533: 37-47. I think this is the article where it is discussed whether the thymic petechiae are on the intrathoracic or the extrathoracic portion of the gland. Cf the photographs in Gilbert-Barness E., ed., *Potter's Pathology of the Fetus and Infant*. St. Louis: Mosby-Year Book, Inc., 1997, p. 439.

Differential diagnosis SIDS/non-SIDS on the basis of histological findings of petechial thymus hemorrhages. Risse M, Weiler G. FSI 1989 Sep; 43(1): 1-7. 87% of SIDS, 55% of stillbirths, 40% of perinatal deaths had thymic petechiae. But in these groups it was all cortical petechiae. In non-SIDS, it was cortex AND medulla, and the petechiae were a lot less numerous.

Is sudden infant death syndrome a diagnosis? Or is it just a diagnostic dustbin? (letter) Emery JL. BMJ 1989; 199: 1240. As above (1985), Dr Emery argues that the SIDS (natural) diagnosis becomes an excuse for not performing a forensic investigation of the real cause of death.

Categories of preventable unexpected infant deaths. Taylor EM, Emery JL. Arch Dis Chil 1990; 65: 535-539. Further work by Emery (Emery, 1985, 1993) finding non-natural deaths.

Suffocation, recurrent apnea, and sudden infant death. Meadow R. J Pediatr 1990 Sep; 117(3): 351-357. 27 babies who were suffocated by their mothers, 9 fatal and 18 nonfatal. Median age 9 months, range 2-48 months. 15 (55%) were over 6 months. 5 had petechiae about the face or mouth. 24 had had previous reported episodes of apnea, cyanosis or seizure, sometimes invented. 18/33 older siblings had died unexpectedly; 48% of the index children had such a sibling. 8 mothers had somatization disorder. Meadow tabulates the differences between these patients and SIDS cases as follows:

	<u>Suffocation</u>	<u>SIDS</u>
Previous apnea	90%	rare
Previous unexplained disorder	44%	rare
Age over 6 months	55%	15%
Dead sibling	48%	2%

Emotional absence by the father was a consistent finding.

The author comments on the difficulty of distinguishing SIDS. He cites British work by Emery (above) for the proposition that "careful clinical and pathologic investigation, combined with laborious psychosocial studies of families, has disclosed that a significant proportion of children previously labeled as having died of SIDS are likely to have been killed by their parents." Emery estimated this proportion as 2% to 10%. Meadow states that "The acceptance that death by suffocation sometimes masquerades as SIDS should lead to reevaluation of the use of the term..." He notes that MSBP-type cases are probably overrepresented in this series because his casefinding arose out of an MSBP clinic setting. Therefore the repetitive aspect may not be representative of infant suffocations at large. As to the subsequent judicial sequelae of Dr Meadow's opinions, see below under "Cannings" and "Sally Clark."

Differences in postperinatal infant deaths between North and Southern Derbyshire. Newlands M, Adamson E, Ghulam S, Saleh M, Emery JL. Arch Dis Chil 1991; 66: 1421-1425. In which Mary Newlands found that some of the names on the child abuse register were the same as those in which cot deaths had occurred. See Emery, 1993.

Fourteen cases of imposed upper airway obstruction. Samuels MP, McClaughlin W, Jacobson RR et al. Arch Dis Chil 1992; 67: 162-170. Cited by Reece & Ludwig, 2001 p. 113 to the effect that no child had any lasting facial marks. Covert video surveillance.

Child abuse, sudden infant death syndrome, and unexpected infant death. Emery JL. Am J Dis Chil 1993; 147: 1097-1100.

From the abstract: "There is now evidence, from a variety of approaches, that indicate that between one tenth and one fifth of children currently diagnosed as cases of sudden infant death syndrome are not natural deaths." Briefly reviews the history of unexpected infant death (as opposed to epidemic/ infectious infant death, which was very common until the middle of the twentieth century). Previously unexpected deaths were usually the subject of an inquest, which was very traumatic to their families. "Around 1970, the whole situation changed, largely because of a group in Seattle, Wash. that held conferences on infant deaths in 1963 and 1970... The concept was put forward that the deaths were caused by an unknown natural disease entity... Rarely has a syndrome been taken up so enthusiastically or based on such negative criteria... It created a dramatic condition for which research money could be collected. The condition was very easy to diagnose, as in general the less found the more certain the diagnosis... Because of the close links between most grant-dependent research workers and bereaved parent organizations, research has tended to be directed to emotionally safe fields of study such as physiological reflexes, metabolic errors, and infections, and investigation of parental activity has largely been ruled out *a priori*. In 1982, following many years of confidential enquiries into all categories of postperinatal child deaths, a study [Taylor & Emery, Two-year study] submitted to a closed session of the British Pediatric Association suggested that about one in 10 deaths of children registered as unexpected may not have been natural. It was suggested that 'gentle battering,' i.e. a physical act that leaves no mark such as a hand or pillow over the face or putting the child face-down on a pillow or soft mattress, should be considered. (citing Emery, Aviemore meeting, 1982). This created a disturbance very like that produced by Helper (sic) and Kempe when they first mentioned child abuse. The organizations related to SIDS were particularly upset..." (p. 1097) Goes on to discuss overlaying as "dangerous neglect:" "instances in which the mother has gone to bed drunk and found the infant dead at her side in bed in the morning, Also, many series of cot deaths found infants to have been left unseen for many hours before being discovered dead, sometimes 8 to 10 hours later. Is this neglect or a cultural phenomenon or both?" Dr Emery goes on to describe the empirical considerations that led to his conclusions:

1. Child death review. A long period of psychosocial child death review of infant deaths that were carried out, at first hospital-based and later home-based, in which it was found that families and wet-nurses refused to put anything in writing, even confidentially, and thus "the fears and suspicions of those who knew the family best were rarely recorded in the reports." Dr Emery wonders (this is in 1993) whether US child death review committees are finding the same thing.
2. Child abuse registries. Mary Newlands noted that "some names on the child abuse register were the same as those in which cot deaths had occurred. She found that about a tenth of the children in cot deaths were siblings of those on the child abuse register."
3. Overlap of risk factors for cot death and abuse.
4. Families with repeated cot deaths. Many of the deaths were not true cot deaths. In particular, a subset of these repeat families had psychological abnormality in one or both parents.
5. ALTE admissions. Brompton Hospital used videotape to find that 18/61 admissions were deliberate suffocations.
6. Autopsy. "Beckwith [Intrathoracic petechiae, 1988] is one of the few investigators who believe that they can differentiate between a natural and an unnatural asphyxial death, that is, distinguish an external from an internal upper respiratory tract obstruction." Other features, referred to as "gross inconsistencies:"
 - (1) the distribution of hypostatic congestion that sometimes indicates that a child has been prone at death instead of supine as stated;
 - (2) the presence of pressure ischemia over the nose and mouth;
 - (3) the nature and amount of the stomach contents related to the history of the last feed;
 - (4) the presence of features such as severe napkin rash that had been hidden...;
 - (5) resuscitation trauma;
 - (6) fractured ribs. "[T]hese fractures are usually ascribed to resuscitation. We and others have gone through the movements of resuscitation on cadavers and have found that it is extremely difficult to fracture ribs in an infant by pressing on the chest or by any of the usual methods of artificial respiration. (citing Feldman & Brewer on CPR rib fractures, 1984). Fractures of the ribs, however, can be relatively easily produced by abnormal grasping of the child's thorax..." (p. 1099)
7. MSBP. Repeat cot death families often have this.
8. Parental statements. "Parents occasionally state that they have killed their children." Dr Emery finds these statements to be more often attributable to guilt than to fact. But parents are sometimes so exhausted

and frustrated with crying that they stifle the crying by manual occlusion, out of desire to silence, not out of desire to kill. Usually the child does not die.

Author's comment: "To suggest that perhaps between one in five or 10 deaths in children currently attributed to SIDS may be an unnatural death does not imply that all unexpected deaths need to come under active suspicion... We must be careful not to overreact and put the clock back a hundred years. A primary forensic approach to these deaths would, I believe, not be helpful at present. The majority of these deaths are, in my experience, multifactorial... Perhaps the first step would be to 'denaturalize' the diagnosis of SIDS and examine each death based on its own evidence so as to tell the parent the most likely cause of death and, when we have no adequate cause of death, to say so." (p. 1099)

Cited by Stanton, 2003, for the proposition that "In 1985 Emery estimated the incidence of SIDS being filicide...as 2–10%. By 1993 his estimate had risen to 10-20%."

Comment by JKR: This article was evidently written because it was pointed out to Dr Emery that, given that his suffocation hypothesis, just like SIDS, has no pathognomonic physical findings, his hypothesis could be considered to be just as speculative as the natural-death hypothesis. Here he struggles to adduce objective evidence that it is more than speculation. But he implicitly admits (and also in his 1985 article) that very rarely does the evidence for active suffocation in a particular death rise to the level of medical, let alone legal, certainty. In both individual cases and in the large, the evidence he adduces is circumstantial. However, admitting this, we could well ask the next question: if his evidence for a suffocation syndrome is not probative, is it at least as strong as, or stronger than, the evidence for a natural syndrome?

Is sudden infant death syndrome a cause of death? Gilbert-Barness E. Am J Dis Chil 1993; 147: 25-26.

Cited by Emery, 1993, above, for the proposition that "Recent years have seen a progressive disillusionment with the unitary cause hypothesis and even the continued use of a syndrome concept." But Prof Gilbert-Barness does not repeat this criticism in the sudden death chapter of the 1997 edition of *Potter's*.

Sudden infant death syndrome: links with infant care practices. Gantley M, Davies DP, Murcott A. BMJ 1993; 306: 16-20. Cited by Emery, 1993, above, for the proposition that in foreign communities where infants are never left to sleep alone, cot death is almost unknown. Cf Potter's, where they note that in Asian countries where supine sleeping is the rule, SIDS is almost unknown. Dr Choi also pointed this out in years past.

Estimates of fatal child abuse and neglect, United States, 1979 through 1988. McClain PW, Sacks JJ, Froehle RG, Ewigman BG. Pediatrics 1993; 91: 338-343. Cited by AAP 1994 and 2001, *infra*, for the proposition that less than 5% of apparent SIDS deaths are due to abuse.

Fatal child abuse and sudden infant death syndrome: a critical diagnostic decision. Reece RM. Pediatrics 1993 Feb; 91(2): 423-429. A full literature review. Points out the tendency for recurrent apnea and chaotic living conditions to be in the history for non-SIDS.

Investigation and review of unexpected infant and child deaths. American Academy of Pediatrics, Committee on Child Abuse and Neglect. Pediatrics 1993 Nov; 92(5): 734-735

1. Adequate investigation requires interagency collaboration.
2. Review of all relevant records.
3. A centralized database to be used in retrospective review (child death review team).

Neonatal and infantile pulmonary hemorrhage: an autopsy study with clinical correlation. Coffin CM, Schechtman K, Cole FS, Dehner LP. Ped Pathol 1993; 13: 583-589. Found pulmonary hemorrhage in 24% of stillborns and 74% of liveborns. Reviewed past literature to find hemosiderin in 7-18% of infant lungs at autopsy.

Recurrence of unexpected infant death. Wolkind S, Taylor EM, Waite AJ et al., Acta Paediatrica 1993; 82: 873-876. Cited by Stanton, 2003, for the proposition that "In families with a recurrence of unexpected deaths, [they] estimated the incidence [of filicide] at 55% (31/57 cases).

Distinguishing SIDS from child abuse fatalities. American Academy of Pediatrics, Committee on Child Abuse and Neglect. Pediatrics 1994 Jul; 94(1): 124-126. 1. Estimates the incidence of suffocation as less

than 5%. 2. Intrathoracic petechiae can be caused by induced airway obstruction or by reduced FIO₂. 3. "Without a complete autopsy, a careful scene investigation, and a review of the medical history, a diagnosis of SIDS cannot be made." 4. SIDS **should be diagnosed** when (a) autopsy findings are compatible and (b) there is specifically no head trauma, intracranial injury, cerebral edema, cervical cord injury, retinal hemorrhage, or mechanical asphyxia, (c) skeletal survey is negative, (d) other causes of death such as carbon monoxide and drowning are ruled out, (d) current drug exposure is ruled out. 5. Features raising the possibility of suffocation include: (a) previous apnea in the presence of the same person, (b) previous unexplained illnesses, (c) age over 6 months, (d) previous deaths in the care of the same person. Comment by JKR: This document seems to have been partly a response to Meadow's 1990 article which argued that the occurrence of suffocation masquerading as SIDS should lead to reevaluation of the use of the term "SIDS." (The features in 5, *supra*, are clearly taken from Meadow's article.) The purpose of the Academy was to preserve the use of the term "SIDS" by arguing that suffocations can be distinguished if the investigation is thorough enough and if the background is taken into account. The problem with that is that by the Academy's own admission suffocations can be distinguished only by prompt, thorough, and meticulous forensic teamwork, which may not be available. Cf. Roy Meadow, *infra* and Sturmer, *infra*. See the three subsequent AAP statements on this subject: AAP Committee on Child Abuse and Neglect and Committee on Community Health Services. Investigation and review of unexpected infant and child deaths. *Pediatrics*. 1999; 104: 1158-1160; AAP Committee on Child Abuse & Neglect. Distinguishing SIDS from child abuse fatalities. *Pediatrics* 2001 Feb; 107(2): 437-441. Clinical Report: Distinguishing Sudden Infant Death Syndrome from Child Abuse Fatalities. AAP, NAME. *Pediatrics* 2006 Jul; 118(1): 421-427, below.

Potentially dangerous sleeping environments and accidental asphyxia in infancy and early childhood.

Byard RW, Beal S, Bourne AJ. *Arch Dis Child* 1994; 71: 497-500. See under "Strangulation." Has to do with accidental hangings and strangulations. This whole area became more notable after the US Consumer Product Safety Commission got involved in 1999.

The postmortem examination. Valdes-Dapena MA. *Pediatr Ann* 1995; 24: 365-372. Agonal alveolar hemorrhage may occur in two-thirds of infant autopsies.

Leeds inquiry into infant deaths: the importance of abuse and neglect in sudden infant death. Hobbs CJ, Wynne JM, Gellertlie R. *Child Abuse Review (UK)* 1995; 4: 329-339. Cited by Stanton, 2003 for the proposition that 27% of sudden unexpected infant deaths (10/37 cases) were associated with maltreatment.

Interstitial hemosiderin in the lungs of sudden infant death syndrome: a histological hallmark of "near-miss" episodes? Stewart S, Fawcett J, Jacobson W. *J Pathol* 1995; 145: 53-58. But see Byard, 1997, below. See also "Idiopathic Pulmonary Hemosiderosis," and "Pulmonary Hemosiderin," below. See also JKR's review of hemosiderin in the *Child Abuse Quarterly* for April 2003.

Repeat cot deaths in families enrolled into a support programme. Waite A, Emery J. Proceedings of the Fourth Sudden Infant Death Syndrome Conference, Bethesda, Maryland, 1996. According to Gornall, *BMJ* 2006 Dec 2, (below), at this conference Dr Emery gave estimates of the proportion of second SIDS that are natural and unnatural or undet. According to Gornall, Dr Emery's most conservative estimate of the number of unnatural second SIDS was **34%**. And note that this was a new number based on new data coming in from the ongoing Next Infant project of the Foundation for the Study of Infant Death, a longitudinal study and support project for SIDS families and SIDS siblings.

Intra-alveolar pulmonary siderophages in sudden infant death: a marker for previous imposed suffocation. *Pathology* 1997; 29: 60-63 Becroft DM and Lockett BK. Iron stains showed previously overlooked alveolar siderophages widely distributed in the lungs of two pairs of siblings who had hospital admissions for multiple ALTE before dying suddenly at home. [In a later report (see *letter*, 1998, below), the authors report a third pair of dead siblings dying under suspicious circumstances and having intraalveolar siderophages. They also reviewed retrospectively 158 SIDS infants and found seven who had diffuse alveolar siderophages. They caution that this is different from *interstitial* siderophages, which are nonspecific, citing Byard RW et al., Assessment of pulmonary and intrathymic hemosiderin deposition in SIDS. *Ped Pathol Lab Med* 1997; 17: 351-357, above; cf. Stewart S et al, 1995, above.] Bleeding from

the mouth or nose was observed during six of ten previous ALTEs suffered by these children and three unrelated infants in the same care. "Such external hemorrhage is well described in imposed infant suffocation..." See Letter, below. Note: intra-alveolar, not interstitial; cf. Byard et al., 1997, below.

Wrong turns in SIDS research. (commentary). Abraham B. Bergman. *Pediatrics* 1997 Jan; 99(1): 119-121. Concerning the Waneta Hoyt murders, two of which originally had been published by Steinschneider in *Pediatrics* as apne-bradys, (Steinschneider A, Prolonged apnea and the sudden infant death syndrome: clinical and laboratory observations. *Pediatrics* 1972; 50: 646-654, the most often-cited academic paper in the field of SIDS research.) Cites two papers in particular: that one and Naeye RL, Pulmonary arterial abnormalities in the sudden infant death syndrome. *NE J Med* 1973; 289: 1167-1170 as having been "found to be wrong." States that "It is my subjective impression that over 80% of published papers about SIDS contain conclusions that have not been substantiated. The reasons are lodged in the twin scourges of SIDS research: imprecision in the diagnosis, and lack of appropriate controls." See also the 1972 epidemiology article to which Bergman refers as being also "wrong." See also "SIDS paper triggers a murder charge." Pinholster G. *Science* 1994; 264: 197-198.

Formal retrospective case review and sudden infant death. Byard RW, McKenzie J, Beal SM. *Acta Paediatr* 1997; 86: 1011-1012. The authors reviewed all SIDS cases in the year 1995. 24 SIDS. Three clearcut accidents leaves 21 considered to be probable SIDS. After this review, they concluded that they had 16 true SIDS. 4 accidental asphyxia, 1 thyroglossal duct cyst. Therefore 24% of the probable SIDS turned out to be non-SIDS. The changes of diagnosis were due to death scene investigation in 5%, autopsy in 5%, retrospective case review in 14%. [In other words, in 14% of the cases, the authors changed the original autopsy diagnosis not because of new evidence but simply because it was wrong. -JKR].

Assessment of pulmonary and intrathymic hemosiderin deposition in SIDS. Byard RW, Stewart WA, Telfer S, Beal SM. (Adelaide). *Ped Pathol Lab Med* 1997 Mar-Apr; 17(2); 275-282. Looked for pulmonary *interstitial* hemosiderin and thymic hemosiderin in 12 SIDS infants with histories of ALTE and 22 SIDS infants without histories of ALTE. Found that the following percentages had hemosiderin:

Previous history of ALTE	4/12	=	33%
No previous history of ALTE	4/22	=	18%

All thymuses were negative. Noted that 66% of the SIDS with hx of ALTE stained negative, so this finding was non-diagnostic. Compare Becroft & Lockett (above), who found that three pairs of suffocated siblings had *alveolar* hemosiderin, which they feel has a different significance from interstitial hemosiderin. See also under "Pulmonary Hemosiderin," below.

Update: pulmonary hemorrhage/hemosiderosis among infants -- Cleveland, Ohio, 1993-1996. MMWR January 17, 1997; 46(02); 33-35 (have). Rainbow Babies' Hospital studied 10 autopsy cases and 30 autopsy controls for acute idiopathic diffuse pulmonary hemorrhage/hemosiderosis. Pulmonary hemorrhages recurred in five of the infants after they returned to their homes. One infant died. The cases were all clustered in the same neighborhood. Nine of the affected infants and seven of the controls lived in water-damaged homes. The air concentration of *Stachybotrys atra* was significantly higher in the homes of case infants. Then they found an additional 11 cases, of which two died. So then with three deaths the Coroner reviewed all 172 infant deaths seen during the period 1993-95. He found that nine infants (5%) had + lung hemosiderosis. Two of these were homicides and one had a past history of child abuse. No etiology found for the other six. Two had had symptoms (epistaxis, hemoptysis, and four had cough or melena). *Editorial*: "The review by the Coroner indicated that some infant deaths initially attributed to SIDS actually resulted from pulmonary hemorrhage. Agonal alveolar hemorrhage may occur in approximately two thirds of infant autopsies. [citing Valdes-Dapena, *infra*]. However, the presence of extensive hemosiderin-laden macrophages within the alveoli indicates major predeath pathologic processes... macrophages require approximately 48 hours to convert blood into hemosiderin..." By JKR: The differential diagnosis would be (a) cardiac, (b) trauma, (c) pneumonia, (d) perhaps suffocation. See Letter, next below, which comments on this report. For further on *S. atra*, see *California Morbidity*, April 1998, below. Further note by JKR: In my opinion, this article is another wrong turn in SIDS research. Its findings have never been reproduced.

Covert video recordings of life-threatening child abuse: lessons for child protection. Southall DP, Plunkett MD, Banks MW, Falkov AF, Samuels MP. *Pediatrics* 1997; 100(5): 735-760. Revealed attempted suffocation in 33 of 39 suspected cases. 0 in 46 controls. See editorial, Unimaginable images: seeing is believing, by Richard D. Krugman, *Pediatrics* 1997 Nov; 100(5): 890-891, saying that it teaches that "[T]here are several straightforward clues that appear in these cases that should alert us..." namely, (1) nasal or oral bleeding, (2) other siblings have died. Says the child welfare and judicial systems "have demonstrated their inability to deal with the problem prospectively."

Health effects of toxin-producing indoor molds in California. *California Morbidity*, April 1998.
Stachybotrys atra is also known as *S. chartarum*. A toxigenic mold found in water-damaged buildings, grows on nitrogen-poor, cellulose-rich substances such as wallboard, wallpaper, can produce trichothecene and spirolactone mycotoxins. The trichothecene mycotoxins are protein synthesis inhibitors and act as immunosuppressants. Affect rapidly proliferating tissues. Can cause granulocytopenia. Spirolactones have anticomplement effects. Use a skin test to diagnose it. *S. atra* has caused hemorrhagic disorders in livestock. Thanks to Dr. Ajay Panchal for this reference. See also AAP statement, *Pediatrics* 1998; 101: 712.

SIDS Redux: is it or isn't it? (editorial). William Q. Sturmer. *Am J Forens Med Path.* 1998 Jun; 19(2): 107-108. Emphasizes the need for a detailed workup/investigation and the use of "don't know" if these are not available. Advocates limiting SIDS to the range of 2 weeks to 9 months, because in the under two weeks age group overlaying, wedging, and metabolic disease are usual causes, while in over 9 months infection and occult trauma [and heart disease --JKR] are usual causes. Also points out the the "routine" autopsy is so limited by cost constraints as to be of limited value. "With the advent of cost containment, less (e.g. procedures, tests, slides) becomes more (e.g. savings, investor profits)..." Also, "Unless there is a timely and thorough scene examination, including the 'bed of death,' with reconstruction of infant positioning and adjacent objects, the diagnosis of asphyxia (suffocation) cannot be excluded simply by denying its role in a given case." cf "Common errors in forensic pediatric pathology," below under "Autopsy technique."

SIDS with the external airways covered. Scheers NJ, Dayton M, Kemp JS. *Arch Ped Adol Med* 1998; 152: 540-547. They diagnosed these as SIDS by either autopsy or scene investigation, plus they did direct interviews with the caregivers in all 206 infants. Of these, 59 died with their airways covered, 147 without. The airway-covered situation usually involved prone sleeping with soft bedding, resulting in formation of a pocket around the nose & mouth. No difference in risk factors between the with and without airway covered groups. See also Scheers et al., Where should infants sleep, 2003, below.

A contribution to possible differentiation between SIDS and asphyxiation. Betz P, Hausmann R and Eisenmenger W. *FSI* 1998; 91: 147-152. Uses conjunctival petechiae and lung hyperinflation (emphysema) as indicia of suffocation, which were found exclusively in the suffocation group: 115 SIDS, 17 other natural, 10 head injury, 7 asphyxiation or strangulation. Conjunctival petechiae occurred in all of the asphyxiated victims, and emphysema in six out of seven. Dr. Kirschner comments that "It has been my experience that petechiae are less common in the first six months of life, and are less common in suffocation than in strangulation. The diagnosis of acute pulmonary emphysema, or hyperinflation of the lungs, is also rather tenuous in younger infants and is regarded as unreliable by most pathologists."

A practical approach to suspicious death in infancy -- a personal view. Green MA. *J Clin Pathol* 1998 Aug; 51(8): 561-563.

Child abuse and sudden infant death syndrome. Botash AS, Blatt S, Meguid V. *Current Opinions in Pediatrics* 1998; 10: 217-223.

SIDS or murder? (letter). Becroft DM and Lockett BK. *Pediatrics* 1998 May; 101(5): 953-955
 These two British researchers argue that the authors of the Cleveland pulmonary hemorrhage report failed to rule out imposed suffocation, because out of their 10 infants, 5 had recurrent episodes right after leaving the hospital. Also, the acute presentation differs from the classic slow onset of idiopathic pulmonary

hemosiderosis. Further saying that we have recently reported abundant pulmonary siderophages in two pairs of suffocated infants with recurrent ALTE. Also, “**Bleeding from the mouth or nose** was observed during 6 of 10 previous ALTEs suffered by these children and three surviving infants who had been in the same care.” Also that a working party of the British Pediatric Association noted that “commonly there is some blood-stained discharge from the nose or mouth” in imposed-suffocation cases. There was no such discharge noted in the Cleveland cases, but other clinical features of them were typical of the British suffocation cases. Authors’ reply: 1. We did consider homicide in our PICU differential of these patients and we have a high index of suspicion and experience with child abuse in our PICU. We made appropriate inquiries, even though we recognize that suffocation is difficult to rule out absolutely. 2. The temporal and geographic clustering of our cases is not explained by the suffocation hypothesis. 3. Only one of our 24 infants had had a previous ALTE; in abuse you expect more recurrence. 4. 5 of 8 infants who returned to their homes had recurrence, while only 2 of 16 who were sent to other homes had recurrence. 5. These infants had much more severe hemorrhage than would be expected in suffocation; some of them required transfusions.

Unnatural sudden infant death. Meadow R. Arch Dis Child 1999 Jan; 80(1): 7-14. (have) Retrospective review of the records and findings of 81 children judged by the courts to have been killed by their parents. Factors found:

1. Most homes were disadvantaged
2. Half the perpetrators had a history of somatizing or factitious disorder.
3. 43% of the children had facial bruises, facial petechiae, or blood on the face.

Conclusion: the risk factors and findings had some overlap with SIDS. Inadequacies in evaluation exist. Until a thorough postmortem examination is combined with evaluation of the history and circumstances of death by an experienced paediatrician, most cases of covert fatal abuse will go undetected. (*voire* Sturmer, supra.) The term “SIDS” still requires revision or abandonment. Sir Roy is not cowed by the AAP’s insistence that these cases can be readily distinguished from SIDS. He comes back at us with 81 more murder cases, 42 of which masqueraded as SIDS. (Another 29 were given a natural disease as the cause of death at autopsy; what does this say about the accuracy of autopsy?) If you take out the somatizing and factitious mothers in Meadow’s own material, the frequency of suffocation drops by half, to about the 5% figure quoted by the AAP for American material. As to the subsequent judicial sequelae and complications of Sir Roy’s testimony and opinions on occult suffocation, see below under “Cannings” and Sally Clark.” –JKR. Reece & Ludwig cite this article for the proposition that “blood in the mouth, nose, or on the face was reported in 39% and only 10 children had either bruises or petechiae on the face or neck. **More than half of the victims had neither bruises, petechiae, nor a history or finding of bleeding.**” Reece & Ludwig, 2d ed (2001) p. 112-113.

Some ‘cot deaths’ are child abuse. White C. (London) BMJ 1999 Jan; 318(7177): 147

The challenges of recognizing child abuse: seeing is believing. (editorial) JAMA 1999 Feb; 281(7): 657-659

Time to put “cot death” to bed? Green MA. BJ 1999; 319: 697-698. Cited by Stanton, 2003 in suggesting that previous estimates of suffocation in SIDS “may have been too high.”

Non-lethal and subtle inflicted injury and unexpected infant death. Byard RW, Donald T, Chivell W. J Law Med 1999; 7: 47-52. Emphasizing the unreliability of the supposed “gold standard” of the autopsy for picking up these forms of assault.

Risk factors in cosleeping. Jem Berry, MD. Jem.berry@bristol.ac.uk unpublished observations, 1999. Most cosleeping may be safe, but there are circumstances when it may be less safe. These include:

- Very large adult
- Very small baby
- Age under 12 weeks
- Newborn
- Alcohol use

Drug use
Tobacco use
Sofas = 40x increased risk

Alveolar hemorrhage in 200 babies. Jem Berry, MD, (personal communication, 1999). Observations from 200 cases. *Extensive* lung hemorrhage is more often seen in very young babies. Is seen more often in suffocations (both accidental and intentional), and this association becomes stronger as the hemorrhage becomes more extensive. But is nonspecific. [Henry Krous reports that lung hemorrhages are very common in SIDS. That is also my experience. But hemosiderin is not. –JKR]

Review of hazards associated with children placed in adult beds. Nakamura S, Wind M, Danello MA. Arch Pediatr Adol Med 1999; 153: 1019-1023. Finds that the risk of overlaying is substantial. In SIDS and co-sleeping. (letter). Rosenberg KD. (Oregon HS) Arch Pediatr Adol Med 2000 May; 154(5): Argues against the conclusion of Suad Nakamura et al. that infants should not cosleep with adults, because the risk of SIDS has only been shown in cosleeping with *smoking* mothers, and does not accrue in cosleeping with nonsmoking mothers. Nakamura replies that we're not talking about SIDS risk; we're talking about overlaying and wedging risk. We specifically excluded SIDS.

Prognosis in pediatric idiopathic pulmonary hemosiderosis. Saeed MM, Woo MS, MacdLaughlin EF, Margetis MF, Keens TG. (USC) Chest 1999 Sep; 116(3): 721-725. These patients are living longer. (Previous mean survival was 2 ½ years.) These authors report ___ patients. Mean age at diagnosis was 4 years (range of 1 year to 8 years). All presented with anemia and pulmonary infiltrates. 70% had fever, 65% had hemoptysis. The diagnosis is by lung biopsy or BAL. Five-year survival on immunosuppressants was 86%. Some died of massive hemoptysis. There is a female predominance.

Fall and suffocation injuries associated with in-home use of car seats and baby carriers. Pollack-Nelson C. Pediatric Emergency Care 2000 Apr; 16(2): 77-79. Car seats fall off of elevated surfaces. Also, suffocation results from seat overturn on soft surfaces. This review of NEISS data for 1997, infants 6 months and under, plus death certificates in the CPSC's death certificate files. Found that in 1997, 8700 infants suffered fall injuries while in car seats or baby carriers, about 1/3 of which resulted from the seat being placed on an elevated surface. There were 15 suffocation deaths resulting from seat overturn on soft surfaces. Car seats also cause desaturation and apne-brady's in small or premature infants, and are warned against by the AAP. See CAR SEATS.

Frequency of pulmonary hemosiderosis in eastern North Carolina. Jackson CM and Gilliland MGF. Am J Forens Med Path 2000 Jan; 21(1): 36-37. Retrospective study by doing iron stains on the lung tissue of all of the 206 young children (premature newborns out to age 49 months) autopsied over an 18-year period in an area subject to frequent flooding. Of these, 23 had pulmonary siderophages. (About 11%). By original diagnosis these 23 broke out as:

SIDS	7
Infection	7
Congenital anomaly	3
Prematurity	2
Undetermined	4

There were no fungi. The authors do not explain what the iron means. They refer to Dehner's study (1993, *supra*,) reporting a 7-18% rate of pulmonary hemosiderin finding in stillborns and liveborns in the past. Refer also to Stewart et al. consideration (1985) that hemosiderin could be a marker for previous "near-miss" SIDS episodes. Note that in this present study there is no particular association with SIDS. The main point of this article is to deconfirm the earlier Cleveland study, and to show that there is no evidence of an emerging fungal disease as a cause of this. Reference to the pediatric pathology literature by the authors shows that lung bleeding at autopsy is frequent in stillborns and liveborns; but the significance of pulmonary hemosiderin is left up in the air. They don't comment on the interstitial versus alveolar distinction raised by Becroft & Lockett (1997). The article ends with the somewhat cryptic statement that, "Our study confirms that an iron stain may identify sufficient pulmonary hemosiderosis to reconsider

attributing death to SIDS.” Does this mean that the *amount* of hemosiderosis is a significant diagnostic variable?

Babies in adult beds, by Suad Nakamura, PhD, Directorate for Health Sciences, CPSC. Consumer Product Safety Review, Winter 2000, p.5. “Placing babies to sleep in adult beds puts infants at risk of suffocation or strangulation, according to a CPSC study in the Arch Ped Adol Med. The study revealed an average 64 deaths per year to babies under the age of 2 years placed to sleep in adult beds, including waterbeds and daybeds. Medical authorities, such as the AAP and the SIDS alliance, also have concerns about placing babies to sleep in adult beds. AAP believes that bedsharing or co-sleeping may be hazardous under certain conditions.... The SIDS Alliance recently issued a statement that stated, ‘...bedsharing...can, under a number of conditions, actually be hazardous.’ A CPSC review of incident data from Jan 1990 to Dec 1997 linked adult beds to at least 515 baby deaths. Analysis of the deaths revealed four major hazard patterns, These included:

- n suffocation associated with [cosleeping]
- n suffocation [due to wedging] between the mattress and another object
- n suffocation due to [face down on waterbed]
- n strangulation in rails or openings on beds that [allow a baby’s body but not head]”

CPSC’s study is the first to quantify the number of fatalities resulting from the practice of co-sleeping with babies. Of the 515 deaths, 121 were [due to overlaying.] More than ¾ of these occurred to infants younger than 3 months. The other 394 deaths resulted from suffocation or [strangulation/entrapment.] ... CPSC is working with the bedrail industry... [Reference should be had to the CPSC’s web site, <http://www.cpsc.gov/> --JKR]

Petechiae of the baby’s skin as differentiation symptom of infanticide versus SIDS. (Germany).

Oehmichen M, Gerling I, Meissner C. JFS 2000 May; 45(3): 602-607. Three siblings. The oldest one and last to die was 3 years old and was found to have obvious oral-nasal-throat injuries and facial petechiae, and the mother confessed to having suffocated her smaller two infants earlier. Upon review, these infants, aged 1 year and 3 months, were found to have facial petechiae, upper thorax, and intraoral, consistent with the SVC distribution. Mother confessed on all three.

Asphyxial deaths and petechiae: a review. Ely SF and Hirsch CS. JFS 2000; 45: 1274-1277. Petechiae are caused by impaired venous drainage, a purely mechanical event. Forms of asphyxia that don’t involve compression rarely involve petechiae.

Pulmonary hemosiderin in deceased infants: baseline data for further study of infant mortality. Randy Hanzlick and Kevin Delaney. Am J Forens Med Path 2000 Dec; 21(4): 319-322. Did Prussian blue stains on the autopsy lungs of 59 infants without regard to cause of death. Examined four slides on each case (anterior and posterior upper lobes). Each of three pathologists scored the staining on each section 0 through 4, for a maximum possible score of 48 (16 per pathologist.) The overall average score per infant was 6. There were six cases with an iron score of 12 or higher; one of these was SIDS, the others were congenital heart disease, abruptio placentae, bronchopneumonia, overlaying, and drowning. “The findings of this study suggest that the presence of hemosiderin in infant lungs, especially if focally abundant and present in many or most microscopic fields, should prompt special consideration that the cause of death may not be SIDS.” Cf Jackson & Gilliland, 2000, above.

Pulmonary hemorrhage in deceased infants: baseline data for further study of infant mortality. Hanzlick R. Am J Forens Med P 2001 Jun; 22(2): 188-192. Found that lung hemorrhage is common but usually “patchy, focal, and sporadically distributed.” Resuscitation may exacerbate it. Tends to be more prominent in long postmortem interval. Position when found may affect its distribution. “The constellation of significant pulmonary hemorrhage, elevated macrophage counts, and above-average pulmonary hemosiderin load is rare.” Idiopathic pulmonary hemosiderosis (IPH) is also rare and lacks diagnostic criteria.

AAP Policy Statement February 2001: Distinguishing SIDS from Child Abuse Fatalities. (RE 0036).

AAP Committee on Child Abuse & Neglect. Pediatrics 2001 Feb; 107(2): 437-441.

<http://www.aap.org/policy/re0036.html> This attempt by Dr. Reece and the AAP. This article qualifiedly

limits the diagnosis of SIDS to the first six months of life: “Approximately 90% of SIDS deaths occur before the age of 6 months. (citing Peterson, 1988). SIDS is suspected when a previously healthy infant, usually younger than 6 months, is found dead in bed...” Appears to make the diagnostic criteria of SIDS more conservative: “Only on completion of a thorough and negative case investigation (including performance of a complete autopsy, examination of the death scene, and review of the clinical history) should a definitive diagnosis of SIDS be assigned as the cause of death.” “Cases that are autopsied and carefully investigated but reveal substantial and reasonable uncertainty regarding the cause or manner of death may be designated as undetermined.” A young infant’s death should be ruled as attributable to SIDS [only] when all of the following are true:

1. [A]utopsy findings are compatible with SIDS.
2. There is no gross or microscopic evidence of trauma or significant disease process.
3. There is no evidence of trauma on skeletal survey.
4. Other causes of death are adequately ruled out. (giving a list of examples)
5. There is no evidence of current alcohol, drug, or toxic exposure, and
6. Thorough death scene investigation and review of the clinical history are negative.”

Later, says that “Pathologists establish the diagnosis of SIDS by exclusion when they are unable to identify other specific causes for a child’s death.” And the death “remains completely unexplained.” (citing DiMaio, *Forensic Pathology*, 1989.) “Cases that are autopsied and carefully investigated but reveal substantial and reasonable uncertainty regarding the cause or manner of death may be designated as undetermined. Examples of undetermined cases include *suspected* (but unproven) infant death attributable to infection, metabolic disease, accidental asphyxiation, or child abuse.” [emphasis added --JKR]

Cites Meadow as a reference in saying that, “as the occurrence of true SIDS has decreased, the proportion of unexplained infant deaths attributable to fatal child abuse may be increasing.” [Stanton, 2003 did not find this to be the case. --JKR] “Estimates of the incidence of infanticide among cases designated as SIDS range from <1% to 5%.” (citing Steinschneider, 1972; Kukull, 1977; Rosen, 1983; Bass, 1986; McClain, 1988; Reece, 1993; Southall, 1997.) Cites the recurrent apnea/ALTE work from Britain. “It is impossible to distinguish *at autopsy* between SIDS and accidental or deliberate asphyxiation with a soft object.” [emphasis added --JKR] (citing Valdes-Dapena, 1992.) [See also the discussion on p. 231 of Giardino AP, Christian CW, Giardino ER, *Practical Guide to the Evaluation of Child Physical Abuse & Neglect*. Thousand Oaks: Sage Publications (1997), to the effect that SIDS and suffocation cannot be distinguished by autopsy. --JKR] However, certain circumstances should indicate the possibility of intentional suffocation, including:

1. Previous recurrent cyanosis, apnea, or ALTE while in the care of the same person
2. Age at death older than 6 months
3. Previous unexpected or unexplained deaths of one or more siblings
4. Simultaneous or nearly simultaneous death of twins
5. Previous death of infants under the care of the same person
6. Discovery of blood on the infant’s nose or mouth in association with ALTE’s.

This article is cited in a later editorial in JAMA (2001 May 2; 285(17): 2244-2245) for the proposition that 2% to 5% of SIDS cases are infanticide.

Discusses management of SIDS, saying, inter alia, that (a) first responders should be trained to make scene observations like position, temperature, rigor, sleep surface, etc.; (b) a preliminary diagnosis of “probable SIDS” should be given to the family if nothing appears initially; (c) parents should be informed that a full postmortem examination will be done to exclude “other causes of death.” (d) “The family is entitled to an opportunity to see and hold the infant once death has been pronounced.” (e) SIDS referral; (f) child death review. Advocates postmortem skeletal survey and specimen radiography, but does not say who should do this. Refers to an international standardized autopsy protocol (Krous, 1996). Body fluids to be collected and tested for metabolic diseases.

The following procedural recommendations are made:

1. accurate history-taking by first responders
2. prompt death-scene investigation with careful interviews by knowledgeable personnel
3. examination of the dead infant at a hospital emergency department by a child maltreatment specialist (a controversial recommendation because it seems to tread on Coroners' prerogatives and would create two sets of postmortem findings)*
4. postmortem examination following established protocol within 24 hours, including radiographic, toxicology, and metabolic
5. collection of medical history through interviews of caretakers, interviews of key medical providers, and review of previous medical records
6. supportive approach to parents
7. consideration of intentional asphyxiation in cases with a history of recurrent cyanosis, apnea, or ALTE witnessed only by a single caretaker or in a family with previous unexplained infant deaths
8. use of accepted diagnostic categories on death certificates
9. prompt informational sessions with parents when the results indicate [natural death]
10. death review

* This recommendation has since been rescinded by the AAP because of objections by NAME that manipulation of the body by non-coroners after death and without parental consent could violate applicable coroners' laws. See Addendum: distinguishing sudden infant death syndrome from child abuse fatalities, Pediatrics 2001 Sep; 108(3): 812. See NAME Board communication, 2001. Has been superseded by the new AAP clinical report of 2006, below, which "addresses deficiencies" in this effort.

NAME Board communication, 2001. Via NAME-L, March 1, 2001. From the NAME Board and Executive committee to the AAP regarding the above recommendations for infant death investigations. 1. The term "child maltreatment expert" is not well defined. May open the door for some self-proclaimed "experts" to complicate an investigation. (This point is duplicated in point 4, below.) 2. Manipulation of the deceased body after death may introduce artifacts or alter significant findings in ways that also make the death investigation more difficult. Additional manipulation may also provide fodder for attorneys who wish to argue that alleged injuries were actually the result of postmortem manipulations... 3. Examination or manipulation of the body by "experts" without proper statutory authority or family permission may constitute a tort or be a violation of criminal law. 4. Adding new "experts" complicates the problem of defining what an "expert" is for court purposes. 5. Minor differences in opinion about interpretation of injuries may complicate the adjudication of cases. 6. External examination alone is unlikely to add significant information. Goes on to insist that ME's are competent in this field. But admits that "room remains for improvement in death investigation nationwide," citing three articles by Hanzlick. (See under "Autopsy technique.")

Sudden Infant Death Syndrome: Problems, Progress & Possibilities. Byard RW and Krous HF, eds. New York: Oxford University Press, 2001. Discusses, e.g. at p. 232, the possibility of missing subtle homicide at autopsy. Reminding us of the possibility of metabolic disease when events recur in the same family. This applies to ALTE as well as SIDS. Citing the 1987 NIH Consensus Development Conference on apnea to the effect that "a minority of infants who die of SIDS may have had previous significant apneic episodes." (But this NIH statement may have relied on Steinschneider's publication of 1972 --JKR)

Alveolar hemorrhage syndromes: update on pulmonary hemosiderosis. Epstein CE and Fan LL. J Respir Dis Pediatr 2001 Feb; 3(1): 49-56. The generic term is PH -- pulmonary hemorrhage. This includes a list of differential diagnoses, which they break out into two broad groups: those that are not part of a systemic vasculitis/ vasculopathy, and those that are part of one. (Or, as they put it, "without pulmonary capillaritis," and "with pulmonary capillaritis," -- "capillaritis" not being defined but apparently meaning any derangement of the vascular structure.

Without capillaritis

Cardiac

Noncardiac

Idiopathic pulmonary hemosiderosis (IPH)

Acute pulmonary hemorrhage of infancy
 Bone marrow transplantation
 Immunodeficiency disorders
 Heiner's syndrome (cow's milk allergy)
 Celiac sprue
 Infanticide

With capillaritis (systemic vascular diseases that include pulmonary "capillaritis")
 Goodpasture's, SLE, IgA nephropathy, PAN, JRA, ITP, WG, Behcet's, Henoch,
 drug reaction...

Now as to IPH, it mainly presents in infancy and early childhood. It usually has cough and anemia as its cardinal signs, and the initial differential is that of anemia. It can have an acute presentation or an insidious presentation. The acute presentation is severe hemoptysis, with or without wheezing, dyspnea, cyanosis. The insidious presentation is recurrent episodes of pulmonary bleeding sometimes associated with fevers. Symptoms include pallor, lethargy, cough (which may be the only symptom), and failure to thrive.

SIDS, bedsharing, parental weight, and age at death. Carroll-Pankhurst C, Mortimer EA Jr. Pediatrics 2001 Mar; 107(3): 530-536. Went through the Coroner's files in Cleveland for 1992-1996, found 84 SIDS cases, compared age, maternal weight, other risk factors, and bedsharing. 30 bedsharing, 54 non-bedsharing. Age for bedsharing deaths was 9 weeks, for non-bedsharing was 12 weeks. Among an urban population at high risk for SIDS, bedsharing was strongly associated with a younger age at death, regardless of any other factors. In this subpopulation, bedsharing was an independent risk factor for sudden death, particularly if the parent was large. [But with such small numbers, these figures cannot possibly be statistically significant. -JKR Also, does not consider the UNDETS in the same population.] See also Betty Spivack's commentary in *Child Abuse Quarterly* July 2001 and Jem Berry's comments, above.

Comment on Carroll-Pankhurst article by Betty Spivack, March 12, 2001. Until we have reliable data on the frequency of cosleeping at various stages of infancy in various socioeconomic groups and stratified for other SIDS risk factors, we cannot make a decision as to whether the practice is an additional risk or protective. Until data of this sort comes out, we won't get beyond the well-demonstrated fact that cosleeping occurs in 20-40% of infants dying of SIDS. The significance of that fact will remain elusive. Additional risk: Carroll-Pankhurst art and Ped 1992; 90: 905-908, BMJ 1990; 301: 85-89, BMJ 1999; 319: 1457-1460. Protective: BMJ 1995; 311: 1269-1272, J Behav Med 1993; 16: 589-610, Sleep 1993; 16: 263-282.

Nasal and intrapulmonary haemorrhage in SIDS. Becroft DMO et al. Arch Dis Child (Br.) 2001; 85: 116-120.

Oronasal blood in sudden infant death. Krous HF, Nadeau JM, Byard RW, Blackbourne BD. Am J Forens Med Path 2001 Dec; 22(4): 346-351. Says the literature on this is limited; "little published information is available." Citing Beckwith, 1973; Krous, 1984; Krous, 1988; Meadow, 1990; Southall et al (covert video), 1997. They reviewed their database on 406 cases of SIDS, in 155 of which some type of oronasal secretion was described. Found 40 cases (26%) in which the secretion was described as sanguinous. In 28 cases (18%) it was described as "blood." Analyzing these 28 cases, they found that half the time (14 cases) the blood was seen before CPR. Of these 14 cases,

10	SIDS
2	Accidental suffocation
2	Undetermined

Of these ten SIDS cases, 4 had moderate to severe lung hemorrhage, 5 had mild, 1 had none. Two of the 28 infants with blood had superficial oronasal lacerations possibly attributable to resuscitation. In 23 the frenula were recorded and were recorded as normal; 5 had no report on the frenula.

They conclude that oronasal blood, if observed before CPR, may be a sign of accidental or inflicted suffocation. As support for this conclusion, they note their findings that

1. Of the 14 non-CPR blood cases, all but 3 were cosleeping and only 2 were supine in a safe crib.
2. Blood was reported in 14% of all accidental suffocation cases, as compared with 1% of SIDS cases.
3. Among the 10 non-CPR SIDS cases, the CPS history was only known in 3, but one of these three had multiple CPS referrals for abuse. That's a rate of 33%.

The authors advise:

1. Mucoïd or frothy bloody fluid expelled from pulmonary hemorrhage should be distinguished from frank blood.
2. An otoscope should be used to examine the oronasal skin and mucosa in cases where possible blood is seen. Frank blood is usually from local oronasal injuries. The cause of these is either attempted resuscitation or other physical injury to the face, accidental or inflicted.

Specific dangers associated with infants sleeping on sofas. Byard RW, Beal S, Blackbourne B, Nadeau JM, Krous HF. J Paediatr Child H 2001 Oct; 37(5): 476-478. Australian coroners' files had 10 cases with complete scene investigations: 4 SIDS and 6 asphyxias. The 6 asphyxias were:

2 overlaying
 1 wedging btween adult and sofa back
 2 cosleeping with a drunken or durg-intoxicated adult
 1 wedging between pillows and sofa back
 1 wedging against sofa back

(apparently some overlap between cases). Conclusion: sofas are unsafe regardless of whether there is cosleeping or not.

Death by overlaying and wedging. Collins KA. Am J Forens Med P 2001 Jun; 22(2): 155-159. Reviewed all such pediatric deaths in the South Carolina office for fifteen years. Found 21 cases diagnosed as either overlaying, SIDS versus overlaying, wedging, and other accidental asphyxia. Found

"asphyxia due to overlaying"	11
"SIDS v. overlaying"	10
wedging	8
other accidental asphyxias	<u>3</u>
total	32

Of the 21 overlaying and SIDS v. overlaying cases, the ages were 6 days to 11 months. Twelve of them were cosleeping with more than one other person. Only two adults admitted drinking. Of these 21 cases, found:

ocular petechiae	3
facial petechiae	1
intrathoracic petechiae	19
abrasions	1
pressure marks	2
oral or intraoral lesions*	0

"[A] thorough history and scene investigation are crucial. Cites Knight, *Forensic Pathology*, 2d ed., 1996, pp. 347-355 and 439-441 for the proposition that homicidal suffocation of infants may have negative autopsy findings. Says that the pattern of livor may indicate the position and pressure points. Petechiae are unusual in infants, even with neck compression. "With neck compression or strangulation, one may see ocular and facial petechiae as venules rupture secondary to the increased pressure as veins are occluded and arterial patency maintained. However, this has been noted to occur less frequently in infants and young children." (citing Knight, 1996; Byard & Cohle, 1994; Rao & Wetli on conjunctival petechiae, 1988;

Jaffe on petechiae, 1994; DiMaio's book; Krous & Jordan, necropsy study of petechiae, 1984.) As to intrathoracic petechiae, they may be caused by forceful respiratory efforts against an occluded airway; almost never seen in hangings and strangulations. "Like SIDS victims, overlaying and wedging victims usually have a completely negative autopsy. The presence of intrathoracic petechiae supports the theory of mechanical asphyxia... More importantly, when a child does have positive findings of multiple contusions, abrasions, or oral-intraoral lesions,* these probably don't result from overlaying, wedging, or other accidental suffocation." (p. 158) The author calls attention to the finding of no oral or intraoral lesions in the accidental cases, saying that the force required to produce them is inconsistent with an accidental mechanism. This article cited by Kohr, 2003, below, for the proposition that in eleven overlaying victims they found only two who had ocular petechiae and one who had facial petechiae, while nine had intrathoracic petechiae.

Reece & Ludwig, Child Abuse, second ed., 2001: p. 521, Recurrence of SIDS within a family: "...Given that the cause(s) of SIDS remain unknown, the possibility of genetically transmitted inborn errors of metabolism or other genetically transmitted conditions is raised. It also provokes questions of a forensic nature..." Citing studies from Norway and Washington State showing a 4X relative risk for SIDS among subsequent siblings of SIDS victims *among births at large*. But among families matched for maternal age and victim birth-rank, there is no increased relative risk. But notes that since back-to-sleep, what with the large increase of the proportion of atypical SIDS in the SIDS population, the recurrence risk may well be higher than unity even for matched controls, because of risk factors like poverty, chaotic conditions, smoking, postnatal drug exposure, and low birth weight. [Unless, of course, the controls were also class-matched. --JKR] Data not yet available for the post-back-to-sleep period. p. 529, Differentiating between SIDS and child abuse: Soft smothering does happen. Quotes DiMaio's book, 1989, and DiMaio, *SIDS or murder*, 1988, to the effect that "Unfortunately, some cases are homicide by soft smothering." Quotes Emery, 1985, to the effect that "filicide is the probable mechanism of death in approximately one in ten of the unexplained unexpected deaths." But there is a natural urge of health professionals to protect innocent, grieving parents "from heavyhanded false accusations of child abuse." Says the dispute involves weighing compassionate support against investigative needs. Goes on to discuss the literature by looking at the following lines of inquiry:

- Articles in which suffocation was explicitly considered
- Munchausen articles
- Substance abuse-related infant fatalities
- Twins (and child abuse, not twins and SIDS)
- Radiology as part of the autopsy
- Child death review teams as part of the investigation
- Perpetrator profiling

P. 529-531 Articles in which suffocation was explicitly considered. The authors review the following articles: Emery & Taylor (Sheffield), 1986 performed complete death investigations on all infant deaths over a 24-year period and concluded that 10% of the deaths were accidental suffocations and another 10% possibly non-accidental suffocations. Asch, 1968 speculated that postpartum depression might commonly cause infanticide. Bass, 1977 reported 15 asphyxial infant deaths thought to be accidental entrapments or hangings. Christoffel et al., 1985 autopsied 43 child deaths, of which many turned out to be child abuse. But, Reece & Krous point out, all the ones in the SIDS age group turned out to be SIDS. Kirschner & Stein, 1985 on mistaken diagnosis found five SIDS misdiagnosed in the ER as child abuse. Bass et al., 1986 did independent death-scene investigations of 26 cases diagnosed as SIDS by the New York ME's office and found that at least seven of them were accidental deaths. (A controversial study.) Perrot, 1988 found that eight out of 170 SIDS cases had been misdiagnosed based on no autopsy or no scene investigation. Emery et al, 1988 found that ten out of 60 SIDS cases had natural causes of death on review. [My summary of this review would be that these eight articles from the early days discovered an unacceptably high error rate in the then-existing methods of diagnosing SIDS, mostly because of no autopsies, poor autopsies, or no scene investigations. Work by Blackbourne, O'Halloran, Byard and others has shown that today this error rate does not happen. Accordingly, SIDS-homicide rates deduced from this body of research are in my opinion of questionable value today. JKR.]

Uses these lines of information to construct a table (Table 20.3) describing three analytical categories into which an infant death might be placed depending on the above types of information:

	“Consistent with SIDS”	“Less consistent with SIDS”	“Suggestive of abuse”
History			
Age			
External			
Pregnancy			
Scene			
Family Hx			
Internal			
Review			

Recommends thirteen steps toward evaluation of SIDS v. Suffocation deaths:

1. Accurate history-taking by personnel at the time of death
2. Expert external examination of the body at the hospital
3. High-quality postmortem examination within 24 hours
4. Routine skeletal surveys
5. Prompt death-scene investigation
6. Detailed previous medical records
7. Detailed medical history from the caretakers
8. Child death review
9. Restricting death certificates to accepted diagnostic categories
10. Recognizing the difficulties involved
11. Supportive approach to the parents
12. Adequate funding
13. More research

Infanticide: is its incidence among postneonatal deaths increasing? An 18-year population-based analysis in California. Krous HF, Nadeau JM, Silva PD, Byard RW. Am J Forens Med Path 2002 Jun; 23(2): 127-131. No, it is not increasing. The absolute number of infanticides is staying the same:

Infanticide (absolute numbers)

1983	21
1994	47
1998	25

Postneonatal death from all causes versus live births

	Deaths	LB	Rate
1981	1,565	420,418	372 / 100,000 LB
1998	1,001	521,265	192 / 100,000 LB

SIDS

1981	575	137 / 100,000 LB
1998	246	47 / 100,000 LB

So the SIDS rate as a percentage of postneonatal deaths

1981	37%
1998	25%

And the infanticide rate was unchanged as a percentage of LB

1983	4.8 / 100,000 LB
1998	4.8 / 100,000 LB

But increased as a percentage of deaths and as a ratio to SIDS deaths.

Their graph (fig 1) shows the infanticide rate flat as a percentage of LB, but rising as a ratio to SIDS (fig 3). Therefore, the apparent rise is due entirely to a decrease in SIDS. SIDS decreased at approximately the same rate as overall infant mortality. The authors comment that “The percentage of SIDS cases that are undiagnosed homicides will probably never be determined with certainty, given the extreme difficulty in distinguishing SIDS from intentional suffocation with a soft surface.” Further, that “It has been stated that infanticide cases differ from SIDS by having a history often characterized by recurrent, apparently life-threatening events and a history of sibling death... (citing Meadow) Other findings such as unexplained facial bruising, cutaneous or conjunctival petechiae, or overt oronasal bleeding when the body is discovered should also raise a suspicion of infanticide (citing Krous, Blackbourne, Oronasal blood, and Southall, covert video).”

“[S]oft suffocation cannot be differentiated from SIDS, absent evidence such as unexplained acute facial bruising, torn frenula, or a confession.” (130) The differential diagnosis is SIDS, positional asphyxia, and soft suffocation. The authors state that in order to differentiate these, one must have:

- meticulous death scene investigation
- postmortem examination including metabolic
- skilled interrogation of witnesses

The authors conclude that “any significant diagnostic shift from SIDS to infanticide is unlikely.”

Cosleeping and sudden unexpected death in infancy. Person TLA, Lavezzi WA, Wolf BC. Arch Path Lab Med 2002; 126: 343-345. Retrospective review of the investigative files in 58 SUDS cases.

58 SUDS

27 cosleepings

11 cosleepings had been diagnosed as SIDS

7 cosleepings had parental intoxication

See Henry Krous’s review in *Child Abuse Quarterly* for July 2002. Dr Krous summarizes that the authors conclude that cosleeping is potentially dangerous. Further, that some cosleeping deaths are either overlayings or “lethal rebreathing in compressible bedding materials.”

Considering suffocatory abuse and Munchhausen syndrome by proxy in the evaluation of children experiencing apparent life-threatening events and sudden infant death syndrome. Truman TL, Ayoub CC. Child Maltr 2002 May; 7(2): 138-148. See under “MSBP” and “ALTE” Describes 138 children admitted over a 23 year period for recurrent ALTE, unexplained death, or SIDS. Had 35 deaths. Of these, the final diagnosis after autopsy and comprehensive chart review was:

SIDS

10

Non-SIDS	25	
Suspicious for child abuse		13
Definite child abuse		2

They identified risk factors for inflicted suffocation which were:

1. Recurrent, poorly explained ALTE's
2. The same caregiver is the witness to most of the ALTE's
3. Blood in the mouth or nose
4. Bruising inconsistent with resuscitation
5. Siblings with other "medical problems." (Especially SIDS or ALTE)

Recommend:

Attention to risk factors
Involvement of child protection teams
Mandatory autopsies
Death scene investigations

Pulmonary siderophages and unexpected infant death. Schluckebier DA, Cool CD, Henry TE, Martin A, Wahe JW. Am J Forens Med P 2002 Dec; 23(4): 360-363. (Denver CME) Retrospectively reviewed two years of all infant deaths n=43. Used iron stains and blindly counted siderophages per 20 hpf, ranging from less than 5 to over 500. Categories set up were:

Category 1	less than 5 siderophages/20hpf
Category 2	5-200 " "
Category 3A	100-500 " "
Category 3B	over 100 in any one lobe
Category 4	over 500 " "

Cases analyzed:

SIDS	16
Asphyxia	5
Undetermined	6
Other	16

All the SIDS cases fell into category 1 (less than 5 siderophages/20hpf).

Found:	Category 1	32	16 SIDS, 1 SUDS, 3 undetermined, 1 drown. 1 positional, 1 interstitial pneumonia, 1 CHD, 8 diseases and accidents
	Category 2	6	1 CHD, 3 NAT, 2 undetermined
	Category 3	4	1 NAT, 1 mechanical asphyxia, 1 UNDET probable asphyxia, 1 UNDET/UNDET
	Category 4	1	Probable asphyxia by suffocation

Concluded: "This study provides further evidence that unexplained pulmonary siderophages can be a marker for trauma or repeated hypoxia/ asphyxia. Siderophages may also be increased for other reasons, but not to the same degree. Siderophages are not increased in SIDS or in acute asphyxial deaths. Because iron-laden macrophages often are not recognized on routine hematoxylin and eosin examination, iron

stains may be helpful in the evaluation of infant deaths. If siderophages are present in increased numbers without an obvious explanation, further investigation is warranted.”

Interestingly, in this study of 43 coroners’ autopsies including eleven with pulmonary hemosiderosis, in only one case did the original pathologist pick up the fact that there were pigmented macrophages in the lungs, and none of the cases had iron stains done in the original processing. –JKR

Is postnatal depression a risk factor for sudden infant death? Sanderson CA, Cowden B, Hall DM, Taylor EM, Carpenter RG, Cox JL. Br J Gen Prac 2002 Aug; 52(481): 636-640. From the abstract: In New Zealand an association has been shown between postnatal depression and SIDS. We prospectively used multivariate risk analysis on 32,984 live births in the Sheffield area, including 42 SIDS. We administered the Edinburgh Postnatal Depression Scale to (all 32,000?) mothers at one month postpartum. Found the following risks for SIDS:

Smoking	7
Postpartum depression	3
Poverty area	2

SUDI associated with maltreatment: evidence from long term follow up of siblings. Stanton AN. Arch Dis Chil (UK) 2003 Aug; 88: 699-701. All Sudden Unexpected Death in Infancy cases over a 14-year period (1982-1996) in North Yorkshire were followed up over a multi-year followup period ending in 2000 (four to eighteen years). Had 69 families with 72 sudden unexpected infant deaths, aged six days to 27 months, nearly all occurring during the 1980’s. All the families received longitudinal followup care by the same pediatrician with multidisciplinary support throughout the study period. Nearly all the deaths were diagnosed as SIDS. Five study families had child protection issues raised in one form or another, and these were as follows:

One mother had two infant deaths, at 4 months and 8 months, with histories of recurrent apnea, and was found to have MSBP.

One mother had two infant deaths, at 7 months and 27 months, with histories of recurrent apnea, and was suspected of MSBP.

One mother from a violent family was placed under child protection care both before and after her baby died of SIDS at 2 months.

One mother from a chaotic family was under protective supervision when her baby died of SIDS at 2 months. A second baby suffered from neglect.

One family with a history of child neglect had their baby die with marasmus at age 5 months.

All the other 64 families had no child protection issues through the long followup period. One of these families had two sudden infant deaths, but the history strongly suggested the presence of a familial disease or condition. In the whole study, only two families (the two MSBP families) were suspected of suffocating their children. That would be 2/69 families (3%) or 4/72 deaths (6%). If all forms of maltreatment involvement of the families are included, there were five families (7% of all SIDS families) and seven deaths (10% of all SIDS deaths) with some form of maltreatment involvement of the family.

As the author points out, 93% of the SIDS families were innocent, as were 90% of the deaths. On the other hand, four out of the six deaths from repeated-death homes were probable homicidal suffocations (67%). From this the author draws the conclusion that the overwhelming majority of SIDS deaths, even in low-income homes, have no likely forensic or criminal aspects, and call for supportive care rather than child protection intervention.

The author concludes: “The association of SUDI and maltreatment within families was at the lower end of previous estimates, 3–10%. Child protection intervention is rarely needed, but investigation and followup

for maltreatment is mandatory where ALTE are reported with a second baby, and after a recurrence of apparent SUDI.” The author relates the observed suffocation rate of 3% and maltreatment-involvement rate of 10% to previously published estimates of 10-20% (Emery, 1993), 27% (Hobbs & Wynne, 1995), 8-18% (CESDI Report, 1996), and 20-40% (Green, 1999). The author comments that her study suggests that Emery’s and others’ estimates of filicide incidence in SIDS “may have been too high”. The author points out that the earlier estimates did not have the benefit of prospective study, team evaluation, large numbers of cases, and followup care; hers is the first study to have these attributes.

Comments by JKR:

1. The author could well have added that all four of the homicidal suffocations in this study came from two homes where even at the time of the first death the history would have alerted any expert to the likelihood that the death was non-natural. This fact offers reassurance to personnel dealing with SIDS cases.

2. It is interesting that the greatly improved level of autopsy practice and child protection implemented since the early 1990’s have produced not higher but *lower* rates of homicide detection in the SIDS setting. That argues that the suffocation estimates coming from the earlier published literature are obsolete, and, like much of the other SIDS literature from earlier years, were products of the haphazard case selection, small study populations, poor quality autopsies, limited case experience, and primitive child death investigations that prevailed prior to the widespread implementation of investigative protocols in the 1990’s.

See also news story in BBC Online at <http://newsvote.bbc.co.uk/>. The BBC article relates that two recent high-profile court cases have led to acquittals in SIDS v. Suffocation prosecutions. In one case, Trupti Patel was acquitted of murdering three of her children who died mysteriously. (The last one had rib fractures, which her counsel explained away as being due to CPR.) Interestingly, Trupti’s mother had five mysterious infant deaths. In the other case, Sally Clark, who had been jailed for life for murdering her two sons, had her conviction quashed, the court of appeal saying that evidence of the rarity of two cot deaths in one family was “unreliable.” The basis for these court rulings was that the courts were convinced that SIDS or something like SIDS does run in families. That is, that unknown forms of natural death/ heritable disease do run in families, and can cause multiple unexplained deaths of infants. Science is as yet not advanced enough to discover all these forms of disease. Therefore criminal convictions based solely on the multiplicity of deaths are unsupportable.

Comment by JKR: I agree with the British courts’ rulings. Multiplicity of deaths, without more, is just as consistent with a heritable disease as it is with homicide. For a criminal prosecution, there has to be something more than just multiplicity. In Trupti’s case, there was more than multiplicity -- there were rib fractures. But the court dismissed those, or failed to understand their significance. Therefore, on the court’s reasoning, this was a multiplicity-alone case, and on such an understanding, acquittal was appropriate. As to multiplicity, see Reece & Ludwig, next below. See also Hill R, Multiple SIDS -- coincidence or beyond coincidence? Paediatr Perin Epid 2004 (below) which argues on statistical grounds that multiple SIDS are mathematically more probable than multiple homicides.

Other comment by JKR: This is a judicial rejection of “Meadow’s Rule,” the maxim that “One SIDS is SIDS, two SIDS is suspicious, and three SIDS is homicide.” It comes in the context of the rejection of the rule’s author, Sir Roy Meadow, who was shown to have fabricated evidence. This showing undermined a great deal of British pediatric legal medicine and necessitated the review of hundreds of past infanticide convictions.

Inflicted compressional asphyxia of a child. Kohr RM. JFS 2003 Sep; 48(5): 1148-1150. (Terre Haute). Case report of a two year old in a day care center who refused to go to sleep, so she was taken out in the hall and placed face down on a floor mat, and a 130-lb. adult female attendant with a leg cast placed her legs on top of the child’s back for 30-40 minutes as a form of physical restraint. The child was found dead. Child had a pattern abrasion on her back which matched the attendant’s leg cast. **There were no petechiae** and no autopsy findings. The authors find no comparable cases in the published literature. They compare the police “restraint asphyxia” cases. Compare overlaying literature. Quote Adelson’s textbook pp. 555-557 as saying that weight on the back of a child can prevent respiration. Say that gradual force can give traumatic asphyxia without any petechiae. Comparing grain silo burials and other slow compression

deaths having a tamponade effect. (Citing Wardrope et al. on the Hillsborough stadium crowd tragedy, BMJ 1991, Byard on accidental child death, 2000, for the finding of no petechiae in gradual traumatic compression, Knight's textbook, 1991.) Citing guinea pig compression studies showing that large weights could produce death in 10 minutes, while smaller weights required 30-40 minutes. Citing Kim Collins, Death by overlaying, Am J Forens Med P 2001 for the proposition that only two out of eleven younger victims had ocular petechiae and only one had facial petechiae, while nine had intrathoracic petechiae. Citing Monteleone's book (1994) for the proposition that compressional asphyxia in children often has no findings.

Where should infants sleep? A comparison of risk for suffocation of infants sleeping in cribs, adult beds, and other sleeping locations. Scheers NJ, Rutherford G, Kemp JS. Pediatrics 2003 Oct; 112(4): 883-889. Compared all suffocation deaths of infants reported to the CPSC during two periods, 1980-1983 and 1995-1998. Had 513 infant suffocation deaths in the first period and 883 in the second period. Conclusion: **"The most conservative estimate showed that the risk of suffocation increased by 20-fold when infants were placed to sleep in adult beds rather than in cribs.** The public should be clearly informed of the attendant risks." They really did two studies: (a) Is the rate of reported infant suffocation associated with certain sleep surfaces increasing? and (b) What is the relative risk for various sleep surfaces as of the later time-period? As to (a), they concluded that yes, the deaths in adult beds increased from 152 to 391 and those in sofas and chairs from 33 to 110, while those in cribs decreased from 192 to 107. As far as (b), they compared 348 suffocated infants under 8 months with 4220 living infants under 8 months and found that the relative risk of suffocation on an adult bed was 40X. If overlayings were taken out, it was still 32X. If they doubled their estimate of the rate of bedsharing for nonsuffocated infants, it was still 20X.

SIDS and unclassified sudden infant deaths. Krous HF, Beckwith JB, Byard RW et al., Pediatrics 2004 Jul; 114(1): 234-238. The new San Diego stratified definition of SIDS, intended to capture the findings that lead to Undet diagnoses and rope them within the definition of SIDS. See also Fleming et al's similarly stratified definition in the CESDI study, HM Stationery Office 2000: 112.

Class IA	Classic SIDS features present and completely documented
Class IB	Classic SIDS features present but incompletely documented
Class II	Meet Class I criteria except for one or more of the following: <ol style="list-style-type: none"> Age less than 21 days or more than 9 months Similar deaths among siblings or in the same care, not accounted for by infanticide or genetic disease A history of perinatal conditions such as prematurity Overlaying not excluded Abnormal growth & development Marked inflammatory changes or other pathology not thought to be the cause of death

Unclassified SIDS: either not autopsied or with equivocal findings for natural or unnatural COD

- Not autopsied
- Findings equivocal for natural versus unnatural cause of death

Sudden unexpected death in infancy and covert homicide in infancy. Levene S, Bacon CJ. Arch Dis chil (UK) 2004; 89: 443-447. Use the CESDI study. Reduce Emery's 10-20% down to 10% or less. See also Hill R, Multiple SIDS -- coincidence or beyond coincidence? Paediatr Perin Epid 2004; 18: 320-326, (below) which also uses the CESDI database to show mathematically that **multiple SIDS are statistically more probable than multiple homicides.** See Cannings, next below. Cited by Dr Reece in reviewing Pollack, 2006 (below) as having loosely estimated that 10% of SUDI were covert homicides.

The report of the Cannings Review Team, 2004. The Attorney General's report to the House of Lords. The Solicitor General's report to the House of Commons. Regarding Cannings-type cases (SIDS cases and SBS cases). As described in a Director of Public Prosecutions press release accompanying the report, Cannings-type cases are SIDS cases resulting in criminal conviction for infanticide. Angela Cannings was

convicted of such because three of her infants died SIDSlike deaths. In allowing her appeal, the Court of Appeal said that new evidence had come to light which had not been available to the jury, suggesting that the children may have inherited a genetic flaw which could have contributed to their deaths.. According to the press release, “The Court said...that with unexplained infant deaths, in many important respects, we are still at the frontiers of knowledge and that, for the time being, where there is a serious disagreement about the cause of death between reputable experts, a prosecution should not be started or continued unless there is additional compelling evidence.” In light of this the DPP decided to review all “pending cases.” But then the Attorney General, apparently under prodding from Parliament, decided to go further, and established an interdepartmental group (IDG) to review not only pending cases but past cases. That group wrote a report which the Attorney General now submits to Parliament. From the report: Introduction.

“1. On 19th January this year (2004), on the day that the Court of Appeal published its judgment allowing the appeal of Angela Cannings against conviction, I [the Attorney General of the Realm] announced that I had established a review of all cases of convictions in the last 10 years of a parent for the unlawful killing of babies and infants under 2. At the same time I asked the Crown Prosecution Service to review the current cases involving an unexplained infant death. A copy of my statement to the House [of Lords] is attached as Annex A.

2. The judgment came at a time of growing concern about the safety of convictions of parents for so-called cot deaths. This judgment had serious and far-reaching implications and showed clear unease about the safety of certain convictions. I shared the unease of the Court of Appeal. Young and vulnerable children need the protection of the law. Yet if unfair accusations or, worse still, convictions for the death of a child occur, it increases the tragedy...

4. This review arose out of the growing concern that miscarriages of justice had occurred in relation to the cases of prosecutions of mothers for killing their infant children in SIDS cases. Three particular cases led to this concern: the convictions and subsequent appeals of Sally Clark and Angela Cannings and the acquittal of Trupti Patel.

5. Sally Clark had been convicted of the murder of her two infant children. Her two very young children had died suddenly and for no immediately obvious reason. The prosecution’s case that she had murdered them relied heavily on expert evidence to show that the children had died from harm caused to them by their mother and not from natural causes.

6. After a long campaign to overturn that conviction, the Court of Appeal eventually found that Sally Clark’s conviction was unsafe and should be overturned. The Court of Appeal...was particularly concerned that certain potentially relevant evidence had not been disclosed to the defence at the trial...and particularly criticised the pathologist, Dr. Alan Williams, for the non-disclosure of potentially relevant evidence. There was also criticism of one piece of evidence given by Professor Sir Roy Meadow.

7. I...acted swiftly to establish an Interdepartmental Group (IDG)...

8. The remit of the Group was initially to examine other cases involving Dr Williams as an expert, in order to determine whether similar instances of non-disclosure had...taken place...

10. The work of the IDG into cases involving Dr Williams was overtaken by the later and larger review which I announced on the 19th January this year...

11. Shortly after the Sally Clark judgment, Trupti Patel was acquitted of charges of murdering her three infant children. The prosecution case against her involved no evidence from Dr Williams though it did involve evidence from Professor Meadow. However, it had the similarity that it concerned sudden deaths of infant children, establishment of the cause of which depended on the increasingly controversial expert evidence as to the cause of unexplained cot deaths...

13. It was against this background of increasing concern by the public as well as criminal justice professionals that on the 19th January 2004 the Court of Appeal gave its detailed reasons for the decision...that it was allowing the appeal of Angela Cannings against her conviction for the murder of her two children.

14. In its reasons the Court expressed the view that, in the light of present medical knowledge, where there was a sudden and unexplained infant death; where there had been a prior unexplained infant death in the family; where there was a dispute between medical experts as to whether the infant had been unlawfully killed; and where there was no extraneous evidence of physical harm; convictions for those deaths were likely to be unsafe. The Court of Appeal reached this judgment in the light of an extensive review of the substantial medical research into SIDS, including recent new evidence. This tended to show that multiple unexplained infant deaths could be compatible with an innocent explanation.

15. The judgment therefore showed that, in relation to unexplained infant deaths, where the outcome depends exclusively, or almost exclusively, on a serious disagreement between distinguished and reputable experts and natural causes cannot be excluded as a cause of death, it will often be unsafe to proceed...

18. [The IDG] agreed [to review the last 10 years of convictions for the deaths of children up to 2 years of age.]...

20 - 35. [The final number of such convictions came to 297.] [I established the Central Review Team (CRT).]

The CRT in its report to me established five categories of past infant death convictions, based on how "safe" the CRT thought the medical evidence was, particularly with reference to any disagreement among reputable medical experts who gave evidence at the trials, as considered in the light of the CRT's expertise.

Cannings category 1: SIDS cases leading to a conviction for infanticide, falling foursquare within the facts of the Cannings case. 3 cases.

Cannings category 2: SBS cases for possible referral to the defendant. 88 cases. These convictions are not necessarily considered unsafe. However, "there is an issue which merits further consideration by a defendant's representative."

Cannings category 3: Other death convictions in which "detailed consideration of the case papers gave sufficient cause for concern in relation to the medical evidence relied upon at trial so as to warrant further consideration. 31 cases -- 22 SIDS cases and 9 SBS cases.

Cannings category 4: Convictions appear safe. 175 cases.

SBS category: 97 SBS cases, of which 9 were considered category 3 and the rest apparently category 4.

The Attorney General then wades into the SBS area (paragraphs 43 – 59). This will be discussed under "Shaken," below.

Review of child care cases finds few instances that raise "serious doubt." Dyer C. BMJ 2004 Nov 27; 329(7477): 1256. Discussing the Cannings inquiry.

Three subsequent infanticides covered up as SIDS. Bohnert M, Perdekamp MG, Pollak S. Int J Legal Med 2004; 119: 31-34. This family had three SIDS in nine years. Cases had marked pulmonary hemorrhage and pulmonary interstitial emphysema and visceral petechiae. Confessed. Note by JKR: Such cases are so rare that they are publishable when they happen.

Delayed infant death following catastrophic deterioration during breast-feeding. Krous HF, Chadwick AE, Stanley C. J Paediatr Child H 2005; 41(4): 215-217. The authors located nine cases, of which two were theirs. Conclude that these infants were suffocated by the mother's breast. Extremely rare, but it happens.

Repeat sudden unexpected and unexplained infant deaths: natural or unnatural? Carpenter RG, Waite A, Coombs RC, Daman-Willems C, McKenzie A, Huber J, Emery J. Lancet 2005; 365: 29-35. This was a prospective study carried out by the Care of the Next Infant project of the Foundation for the Study of Infant Death. They accumulated 6373 babies who were later siblings of infants who had died of SIDS. They followed these families longitudinally for years. 46 of the babies ultimately suffered unexpected sudden death, either in infancy or after infancy. Of these, the authors conclude that only six (13%) were "unnatural" deaths.

Cited by Fern Hauck and Carle E. Hunt, 2006, for the proposition that "[S]econd infant deaths in families are not rare and at least 80% to 90% are natural. Recurrent infant death from SIDS in subsequent siblings is 6 times more likely than from homicide."

But the freelance journalist Gornall, BMJ 2 DEC 2006 points out serious problems with the methodology of this paper, which was published or at least became public during the appellate litigation of the Angela Cannings case and was cited therein. See Gornall, 2006, below.

Changes in the timing of SIDS deaths in 1989 and 1999: Indirect evidence of low homicide prevalence among reported cases. Pollack HA. Paediatr Perin Epid 2006; 20: 2-13. According to Dr Reece's review in the Spring 2006 issue of the *Quarterly*, the author hypothesizes that the back-to-sleep campaign should reduce the incidence of SIDS, but should not reduce the incidence of imposed suffocation. How to test this hypothesis? Look: true SIDS occurs overwhelmingly in the first three months of life; suffocation will occur randomly throughout the first year of life. So, did the age distribution of sudden infant death change with the drop in the incidence of true SIDS due to back-to-sleep? If it did, that would argue that there are a lot of homicides mixed into the SIDS population. So: the author found that the age distribution of sudden infant deaths did not change with back-to-sleep. Therefore, there were probably not a lot of suffocations mixed in. Dr Reece characterizes this as "an interesting approach."

The author found that from 1989 to 1999 the SIDS population changed to include more blacks, more unmarried mothers, more teen mothers, and more smoking mothers.

Dr Reece notes that the CESDI study found that maltreatment was the cause of death in 6.4% of SUDI cases, with an additional 8.1% having maltreatment as a contributing factor. Levene and Bacon (SUD and cover homicide in infancy, Arch Dis Chil 2004; 89: 443) estimated that 10% of SUDI were covert homicides.

Clinical Report: Distinguishing sudden infant death syndrome from child abuse fatalities. AAP, NAME. Pediatrics 2006 Jul; 118(1): 421-427. Says that this report "addresses deficiencies and updates recommendations in the 2001 AAP policy statement of the same name." Note that this present paper is called a "clinical report," not a "policy statement." This one takes note of the recent attempted redefinition of SIDS (Krous, Beckwith, Byard et al., Pediatrics 2004; 114: 234-238), but says that some deaths should be designated as "Undetermined;" this listing is independent of the Krous Beckwith formulation, which would designate many of these as "SIDS." The listing for "should be Undet" reads as follows: "[S]uspected (but unproven) infant death attributable to infection, metabolic disease, asphyxiation, or child abuse." Goes on to say that SIDS is a diagnosis of exclusion, and specifically this means excluding

- acute or remote inflicted trauma
- significant bone disease
- significant and contributory unintentional trauma
- meningitis
- sepsis
- aspiration
- pneumonia
- myocarditis
- trauma
- dehydration
- fluid & electrolyte imbalance
- significant congenital defects
- inborn metabolic disorders
- asphyxia
- drowning
- burns
- poisoning
- toxic exposure

Goes on to discuss suffocation. It may be "difficult if not impossible" to differentiate this from SIDS or accidental suff. States that estimates of the incidence of infanticide among SIDS cases range from less than 1% up to 5% (citing Southall, Covert video, 1997; Reece, A critical diagnostic decision, 1993; Bass et al., scene investigation in SIDS, 1986; McClain et al., 1988; Kukull et al., SIDS and infanticide, 1977). In this context takes special note of a history of ALTE and multiple SIDS. Gives a complete capsule review of the literature of occult suffocation. Gives the following circumstances as indicating the possibility of suff:

- [a history of] recurrent ALTE in the same care
- age at death older than 6 months
- previous unexpected death of sibling
- a previous death of another child under the same carer
- twin SIDS
- evidence of previous pulmonary hemorrhage

As to recurrent SIDS, cites Meadow, 1990, 1999 for the suggestion that the index of suspicion for unnatural death should be higher in families in which an unexplained infant death has occurred previously, and adds that “more recent publications, however, provide some reassurance that a percentage of recurrent, unexplained infant deaths may be, in fact, natural.” (citing Carpenter et al, Lancet 2005; Hill, beyond coincidence?, 2004). [But, unbeknownst to the AAP and NAME, there is every indication that the Carpenter paper’s numbers were doctored, and are artificially low. See Gornall, 2006, below. –JKR] Says that the risk of recurrence is controversial. Once the death has been thoroughly evaluated, the parents should be told that the risk of SIDS in subsequent children is not likely increased.

Discusses the initial scene management of SIDS, suggesting that a preliminary dx of “possible SIDS” be given to the parents at the scene if it looks like SIDS. Says that the family may be given an opportunity to see and hold the body, under direct observation, provided this is consistent with local protocols, statutes, and medical examiner policies. Discusses child death review. Discusses the importance of the autopsy, particularly toxicology and looking for inborn errors of metabolism. Discusses postmortem imaging. As far as the pathologist “may elect” to retain tissues for future studies if an inborn error of metab is suspected by fatty liver or the history.

This report should be read in conjunction with the AAP’s 2005 policy statement on SIDS.

Child protective services referrals in cases of sudden infant death: a 10-year, population-based analysis in San Diego County, California. Krous HF, Haas EA, Manning JM, Deeds A, Silva PD, Chadwick AE, Stanley C. Child Maltreatment 2006 Aug; 11(3): 247-256. What if any is the diagnostic significance of a prior history of CPS referral in the family of a SIDS case? Studied data on 533 families that had a coroners-case infant death. Broke the deaths down into the following groups for statistical purposes: SIDS, other natural, accident, inflicted injuries. Found that in each group at least 27% of the families had a prior CPS referral. The rates did not differ. “[O]ur data, as well as those of others (citing AAP, 2001; O’Halloran et al, 1998; Stanton & Simpson, 2001; Truman & Ayoub, 2002) suggest that prior CPS referral alone regardless of disposition does not reliably distinguish SIDS from deaths from other causes, including inflicted injuries.... Nevertheless, a family or case history of substantiated CPS referrals may merit special consideration given the implications for the safety of surviving siblings.” Gives a chart of total referrals and substantiated referrals for the *index infant*.

referral status	SIDS n=384	NAT n=74	ACC n=61	Homi n=34
no report found	338	61	54	27
at least 1 referral	42	11	5	6
as a percentage of group	11%	15%	8%	18%
referred at birth	18	5	1	1
excluded	4	2	2	1
substantiated	16	7	2	3
% substantiated	38%	64%	40%	50%

Special report: was message of sudden infant death study misleading? Gornall J. BMJ 2006 Dec 2; 333: 1165-1168. This British freelance journalist criticizes the influential 2005 repeat SIDS paper of Carpenter et al. in *Lancet*, an early draft of which was relied on by the Court of Appeal in quashing the conviction of Angela Cannings and by the AAP in revising its discussion of recurrent SIDS in the 2005 AAP SIDS position paper and the 2006 joint AAP/NAME clinical report on distinguishing SIDS from child abuse. The *Lancet* paper by Carpenter et al. reports on a large longitudinal support and study project of the Next Infant project of the Foundation for the Study of Infant Death, a study which was organized way back by

the late Dr John Emery (*q.v.*), who died in 2000. It reports on 6373 later-born siblings of SIDS victims. It reports that of these babies, 46 suffered sudden unexpected death, and of these 46, only 6 (13%) were found to be unnatural deaths. That optimistic finding was influential and was relied upon by the above-mentioned authorities. Specifically, in its 2005 Policy Statement on SIDS, the AAP stated that “Carpenter et al. concluded an 87% probability that a second SIDS death within a family would be of natural cause... Therefore, the task force supports the position that the vast majority of either initial or second sudden unexpected infant deaths within a family seem to be natural rather than attributable to abuse, neglect, or homicide.” The problem with that, as Gornall reports, is that the last publication of these data during Dr Emery’s lifetime (Waite et al., 1998), reporting on 5000 babies in the project who were later-born siblings of SIDS victims, reported that 35 out of the 5000 had suffered unexpected death, and that of these 35 repeat SIDS deaths, 14 (40%) were considered “unnatural.” That would include overlayings, open coroner’s verdicts, and evidence of other preventable accidents, as well as gentle battering (Dr Emery’s term for imposed suffocation). But when the surviving members of the research group, led by Carpenter, ultimately published results from 6373 babies (including the earlier-reported 5000), they reported that only 13%, or 6 out of a total of 46 second-SIDS deaths were unnatural. What happened to the other 8 of the original 14 unnatural deaths? How did we go from 14 unnatural deaths in 1998 to only 6 in 2005, and from 40% to 13% with substantially the same data? The answer, Gornall reports, is that after Dr Emery’s death, the remaining members of the research group altered the data by redefining the categories of “natural” and “unnatural” deaths. Instead of placing under “unnatural” all the deaths where there was evidence of a preventable cause, the group now placed in that category only the deaths that were clearcut filicide. This change dramatically lowered the proportion of unnatural deaths, and led to the conclusion stated in the *Lancet* paper that the proportion of repeat SIDS that are unnatural is only 13%. Specifically, in the above redefinition of terms, by which these 8 unnatural deaths as well as some subsequent ones were recategorized as “natural” deaths. As noted above, they were recategorized because they were not clearcut filicide. The *Lancet* authors felt that Dr Emery would have approved of this change, because he was always sympathetic to the plight of bereaved parents. But as Gornall points out, such considerations have no place in statistical research; definitions have to be fact-based. The late Dr Emery had as of 1996 at the Fourth Sudden Infant Death Syndrome International Conference in Bethesda expressed the view that at least 34.5% of repeat SIDS are “unnatural” (i.e. preventable) deaths. So it is evident to Gornall that Dr Emery would not have endorsed Carpenter et al.’s recategorization methodology. Indeed, Gornall quotes a written report prepared by Dr Emery in 1999 for Angela Cannings’ defense team, which stated that “The occurrence of repeat unexpected deaths is a topic on which I have been doing research over the last few years and have been able to do a confidential study involving approximately 100 such deaths. These have all been families who have presented as having two cot deaths. My findings have been that approximately a third of the deaths have been due to a whole series of rare natural causes which had been missed by those doing the necropsy. A third of the deaths were associated with different forms of child abuse and having a whole number of features which indicated that they were unnatural deaths. In the final third no suspicion of unnatural death was found and no natural cause was found and these children constitute what may be termed at the moment as ‘true idiopathic unexpected child deaths.’ ... The occurrence of two unexpected deaths in a family thus raises a definite suspicion of unnatural death which in my experience is confirmed...in a third of such cases.” Gornall also quotes Dr David Hall, past president of the Royal College of Paediatrics and Child Health, as saying that the analysis in the Carpenter *Lancet* paper was “seriously flawed” and “seriously misleading.” Dr Hall had communicated his concerns to the editors of the *Lancet*.

The relation between child deaths and child maltreatment. Jenny C, Isaac R. Arch Dis Chil 2006; 91: 265-269. A review article that covers epidemiology worldwide. The Quarterly’s reviewer, Dr Herman Giddens, says that “the authors have done an admirable job of packing a lot of information into this paper and compiling an extensive bibliography.”

Pulmonary intra-alveolar siderophages in SIDS and suffocation: A San Diego SIDS/SUDC research project report. Krous HF, Wixom C, Chadwick AE et al. Pediatric Developmental Pathology 2006; 9: 103-114. Found no difference between SIDS and suffoc. But some of their “SIDS” were inappropriate. See below under “Pulmonary siderophages.”

A comparison of pulmonary intra-alveolar hemorrhage in cases of sudden infant death due to SIDS in a safe sleep environment or to suffocation. Krous HF, Haas EA, Masoumi H, Chadwick AE, Stanley C. FSI 2007. Wanted to compare SIDS (San Diego SIDS) occurring in a safe sleep environment (i.e. clean SIDS, supine alone on a firm surface with head uncovered) with overlayings and inflicted suffocations. Had the autopsy lungs of

34 clean SIDS
37 accidental suffocations
3 inflicted suffocations

Took sections from all lobes of each lung. Scored the severity of pulmonary hemorrhage by grading each slide as follows: 0 = none, 1 = mild, 2 = moderate and focal, 3 = moderate and multifocal, 4 = diffuse and severe. (Gives exemplar photomicrographs). Found

<u>Grade</u>	<u>0</u>	<u>1</u>	<u>2</u>	<u>3</u>	<u>4</u>
SIDS	4	15	12	3	0
Suffoc	6	15	5	9	5

This was statistically a non-association. Conclusion: “Our data confirm that the severity of pulmonary hemorrhage as an independent finding cannot distinguish cases of SIDS from suffocation, whether accidental or inflicted.”

SIDS BIOLOGY

(see also SIDS GENETICS, below)

DEFINITION: Government Code Sec. 27491.41(a): “*For purposes of this section, ‘sudden infant death syndrome’ means the sudden death of any infant that is unexpected by the history of the infant and where a thorough postmortem examination fails to demonstrate an adequate cause of death.*” This is the 1969 definition (Bergman, 1970). It has been superseded in the medical community by the 1989 amended definition, which reads, “the sudden death of an infant under one year of age which remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene, and review of the clinical history.” (Willinger et al., 1991). See the resources at the end of this Section.

See also Health & Safety Code Sec. 123725 et seq., directing the State Department of Health to establish a SIDS advisory council and requiring public health nurses to respond promptly.

SIDS and related ICD-9 and ICD-10 coding:

SIDS ICD-10 7980/R95

“unknown or unspecified causes” ICD-10 799.9/R99

“suffocation in bed” ICD9 E913[0]

“suffocation -- other” ICD9 E913[1] or ICD10 W76-7 and W81-4

See also the State SIDS Manual: State of California, Department of Health Services, Revised Standardized Autopsy Protocol and Death Scene Investigation/Deputy Coroner Protocol, 1992. DHS 4437 (large blue ring binder).

Created by the Department of Health Services pursuant to the statutory mandate of Government Code Sec. 27491.41(d). Provides that we can “take tissue samples” without consent for research purposes if no visible disfigurement. Sec. 27491(g). This protocol is being revised as of 2005. Carrie Florez 916-650-0323 cflorez@dhs.ca.gov

California SIDS Program

Maternal and Child Health Branch
 State Department of Health Services
 11344 Coloma Road, Suite 560
 Gold River, CA 95670-6304
 800-369-SIDS
info@californiasids.com
<http://www.californiasids.com/>

National SIDS/ Infant Death Resource Center (NSIDRC) www.sidscenter.org 866-866-7437. Part of the US Department of Health & Human Services Health Resources and Services Administration. This is an online compendium of resources on SIDS and infant death. Includes bereavement support, professional resources, and others. Also directs to the various listservs propagated by the University of Florida Institute for Child Health Policy at <http://mchenet.ichp.ufl.edu/> Also <http://www.sids-network.org> See below.

Federal SIDS program
 800-505-CRIB
<http://www.nichd.nih.gov/sids/sids.cfm>

The important need for autopsy standards. <http://sids-network.org/experts/> To help improve uniformity of information, the Division of Maternal and Child Health of the Department of Health and Human Services held a meeting in 1975 which resulted in investigative and autopsy protocols for examining the SIDS infant. These protocols were included in an article entitled, [see below]:

Crib deaths and focal fibrinoid necrosis of the infant larynx. Valdes-Dapena, Marie A. JFS 1958 Sep; Valdes-Dapena, Marie A. JFS 1958 Sep; 3(4): [This lesion, microscopic foci of acute inflammation in the anterior larynx, usually one on either side, is a rather common autopsy finding in SIDS cases. –JKR]

Sudden infant death: chemical analysis of the vitreous humor. Sturmer WQ, Dempsey JL. JFS 1973 Jan; 18(1): 12-19

The examination of the sudden infant death syndrome infant: investigative and autopsy protocols. Jones AM and Weston JT. JFS 1976; 21(4): 833-841. See 1975 DHHS conference, above.

CDC guidelines for investigation of child abuse
 MMWR 45 (RR-10: 1-22) June 21, 1996 (have).
<http://www.cdc.gov/mmwr/preview/mmwrhtml/00042657.htm> or <http://aepo-xdv-www.epo.cdc.gov/wonder/prevguid/m0042657/entire.htm>

SIDS should **not** be diagnosed if the criteria of (a) examination of the death scene, (b) review of the clinical history, and (c) complete autopsy are not met. These criteria “are often not met.” Also, practices for case investigation vary in the US. As of 1996 only four states (CA, MN, MO, NM) had detailed written protocols for scene inv. Therefore, as of 1996, the proportion of so-called SIDS diagnoses that were supported by a scene investigation was “probably very low.” So pursuant to a 1992 Congressional mandate they set forth the scene protocol that we now use. Refers to the deliberations of a 1991 expert panel next below. “Any SUID that has not been thoroughly investigated should be classified as undetermined or unexplained. For about 15% of SUIDs, a thorough investigation will determine or identify a cause of death other than SIDS.” (citing Valdes-Dapena, 1992).

Defining SIDS: deliberations of an expert panel convened by the National Institute of Child Health and Human Development. Willinger M, James LS, Catz C. Ped Path 1991; 11: 677-684.

SIDS alliance pushes for states to pass standardized autopsy and death scene protocols for cases of sudden unexpected infant death. Press Release, The SIDS Alliance, 4/7/98. (have). <http://sids-network.org/experts/> and citing the importance of standardized autopsy protocols: <http://www.cdc.gov/epo/mmwr/preview/mmwrhtml/00001294.htm> See also the AAP policy statements of 1993 (Investigation and Review of Unexpected Infant and Child Deaths (RE9336), Pediatrics 1993 Nov; 92(5): 734-735, <http://www.aap.org/policy/05100.html> Death of A Child in the Emergency

Department(RE9413), Pediatrics 1994 May; 93(5): 861-862, <http://www.aap.org/policy/00206.html>
 Distinguishing SIDS from Child Abuse Fatalities (RE9421), Pediatrics 1994 Jul; 94(1): 124-126,
<http://www.aap.org/policy/00299.html> See also a proposed model state statute called the Child Death
 Investigation Act, AAP Pediatric Policy Reference Guide, February 1993.
<http://www.aap.org/policy/m958.html> and <http://www.aap.org/policy/958.html>

Here's a good one: The site of Drs. Krous and Byard for their SIDS Global Strategy Task Force at
<http://www.sidsglobal.org/>

The National SIDS/Infant Death Resource Center
 8280 Greensboro Drive Suite 300
 McLean, VA 22102
 866-866-7437 (or 800-866-7437?)
<http://www.sidscenter.org>

US Department of Health and Human Services
 Health Resources and Services Administration
 Maternal and Child Health Bureau
 National SIDS/Infant Death Resource Center
<http://www.hrsa.gov>

First Candle/SIDS Alliance
 1314 Bedford Avenue Suite 210
 Baltimore, MD 21208
 410-653-8226
<http://www.firstcandle.org>

First Candle has recently (2006) prepared an elaborate training manual for SIDS counsellors and trainers on CD. It also includes a 200 page program manual with an extensive bibliography.

Resources for families:

County Health Department SIDS Program: 213-639-6441
 LA SIDS Hotline: 800-SIDS-LA
 State SIDS Hotline: 800-369-7437
 Compassionate Friends:
 LA: 310-474-3407
 Glendale-pasadena: 818-957-0254
 Chino Hills: 909-597-1561

See also the various web sites, including First Candle and SIDScenter.org

SIDS Web addresses:

SIDS Foundation of Southern California
 3428 ½ Motor Avenue
 West Los Angeles 90034
 1-800-9 SIDS LA
sidsfsc@AOL.com
<http://www.sidsfoundationofsoutherncalifornia.org>

LA County Health Dept SIDS site
<http://www.lapublichealth.org/mch/sids/sids.htm>

Minnesota SIDS Center
<http://www.asipl.org>
 National SIDS Alliance (since renamed)

<http://www.sidsalliance.org/>

National SIDS Alliance research area

<http://sids-network.org/>

<http://www.sidsalliance.org/professionals>

SIDS/SUDS

<http://perso.infonie.fr/tracy.cook/sudden.htm#autopsy>

SIDSNET listserv

<http://www.sidslist.org/>

SIDS families

<http://www.sidsfamilies.com>

American SIDS Institute

<http://www.sids.org>

National SIDS & Infant Death Program Support Center

Baltimore

<http://sids-id-psc.org>

Kidshealth: The Nemours Foundation

http://kidshealth.org/parent/pregnancy_newborn/index.html

http://kidshealth.org/parent/pregnancy_newborn/home/cosleeping.html

http://kidshealth.org/parent/pregnancy_newborn/medical_problems/sids.html

The CJ Foundation

<http://www.cjsids.com/>

Sudden Unexpected Death in Childhood (over 1 year) 1:100,000 children. The SUDC program of the CJ Foundation includes extensive information on metabolic disease. <http://www.sudc.org> See above under SUDC.

American Academy of Pediatrics

The journal *Pediatrics* abstracts <http://www.pediatrics.aappublications.org>

AAP position papers <http://www.aap.org>

Back to sleep home page <http://www.nichd.nih.gov/sids/>

Policy statement on bedsharing

<http://www.aap.org/policy/pe000272.html>

Latest policy statement on sleeping position and sleep environment

<http://www.aap.org/policy/re9946.html>

Consumer Product Safety Commission

Releases on bedding, cosleeping, the safe nursery

<http://www.cpsc.gov/>

Back to Sleep Campaign

<http://www.nichd.nih.gov/>

National Center for Education in Maternal and Child Health

<http://www.ncemch.org/>

National Guidelines for Death Investigation. US Department of Justice.

<http://www.ncjrs.org/pdffiles/167568.pdf>

National SIDS and Infant Death Program Support Center
1314 Bedford Avenue, Suite 205-B
Baltimore MD 21208

National SIDS Resource Center
2070 Chain Bridge Road, Suite 450
Vienna, VA 22182
703-821-8955
<http://www.circsol.com/sids>

SIDS Alliance
<http://www.sidsalliance.org/professionals>

SIDS/SUDS web site
<http://perso.infonie.fr/tracy.cook/sudden.htm#autopsy>

American SIDS Institute
<http://www.sids.org>

National SIDS Resource Center
<http://www.sidscenter.org/>

US HEW
US Department of Health and Human Services
Health Resources and Services Administration
Maternal and Child Health Bureau
National SIDS/Infant Death Resource Center
<http://www.hrsa.gov>

British one: Foundation for the Study of Infant Deaths
<http://www.sids.org.uk/fsid/>

California SIDS Program
Maternal and Child Health Branch
State Department of Health Services
11344 Coloma Road, Suite 560
Gold River, CA 95670-6304
800-369-SIDS
info@californiasids.com
<http://www.californiasids.com/>

The California State Health Department Epidemiology and Evaluation Section
714 P Street, Room 476
Sacramento 95814
Attn: Carrie Florez, Research Analyst II

Back to sleep homepage <http://www.nichd.nih.gov/sids/>

NICHD <http://www.nichd.nih.gov>

Statistics CDC <http://wonder.cdc.gov> and the National Center for Health Statistics
<http://www.cdc.gov/nchs>

The Discovery of Sudden Infant Death Syndrome: Lessons in the Practice of Political Medicine, by Abraham Bergman, MD. New York: Praeger, 1986.

Awakening from grief: finding the road back to joy, by John E. Welshons. Little Falls, NJ: Open Heart Publications, 1999. P O Box 110, Little Falls, NJ 07424. 800-555-0844. An excerpt provides the following guidelines: 1. Let your tears feed your heart. 2. Tell your story. 3. Open to the possibility that the universe is perfect. 4. Become aware that no relationship is ever lost. 5. Create a sacred space of remembrance. 6. Write a letter to the loved one who has died or left. 7. Keep a journal. 8. Find safe and appropriate outlets for your anger. 9. Find forgiveness. 10. Let go of your guilt. "Once you know God knows everything, you're free." (cf Alcoholics Anonymous, "You're only as sick as your secrets.") 11. Work with your anger at God, or the universe. 12. Incorporate meditation, contemplation, and prayer into your daily life. 13. Be more mindful of your own physical health. 14. Be creative. 15. Consider professional therapy or counselling. 16. Laugh. 17. Relax and take your time. 18. Exercise...

Sudden Death in Infants: Proceedings of the Conference on Causes of Sudden Death in Infants, 1963. Dr. John Butts advises: This 1963 conference was sponsored by the Public Health Service and held in Seattle. Includes original research by Dr. Adelson including a Cleveland study of 130 infant deaths and comments by Dr. Valdes-Dapena on a Philadelphia family with multiple unexplained infant deaths.

Sudden Infant Death Syndrome: Risk Factors and Basic Mechanisms. PMA Publishing, 1988. The proceedings of the 1984 International Symposium on SIDS held in Santa Monica. Dr. Beckwith's article, "A pathologist's perspective on SIDS diagnosis and the search for predisposing factors" states, "As we pointed out in 1969, the majority of cases thus defined had a substantial number of characteristics in common, including death during apparent sleep, a rather narrow age range peaking between two and three months, and a repetitive spectrum of minor morphological lesions at postmortem examination. This latter group of cases had enough features in common to justify the use of the term 'syndrome,' and to suggest that they were dying by a similar, unitary mechanism."

Sudden Infant Death Syndrome: Problems, Progress and Possibilities. Edited by Roger W. Byard and Henry F. Krous. Forward by J. Bruce Beckwith. New York: Oxford University Press, 2001. ISBN 0340759178. <http://www.oup-usa.org>

SIDS. Hoppenbrouwers T and Hodgman J. Women's and Children's Hospital, Newborn Division, Rm L919, 1240 North Mission Road, Los Angeles 90033, Attn: Dr. Toke Hoppenbrouwers. ISBN 09742663-0-2. Published 2003. Available by pdf at <http://www.hoppenbrouwers.net/toke/sids.asp>. Although written for a lay audience, this treatise is compendious. It presents the epidemiologic arguments in graphic form, which makes them much easier to understand. It has received enthusiastic word-of-mouth reviews.

Safe in the Arms of God: Truth from Heaven About the Death of a Child. MacArthur J. Nashville, TN: Thomas Nelson, Inc., 2003. ISBN 07852-6343-8. <http://www.thomasnelson.com>

Mommy Please Don't Cry: There Are no Tears in Heaven. DeYmaz L., 2003. Available from Multnomah Gifts, P O Box 1720, Sisters, OR 97759

To Find Hope: Simple Wisdom for Those Who Grieve. KK Ryan. Mahwah, NJ: Paulist Press, 1997. ISBN 0-8091-3735-6. <http://www.paulistpress.com>

Sudden apparently unexplained death during infancy: pathological findings in infants found dead. Werne J, Garrow I. Am J Path 1953; 29: 633-652. Cited by the AAP (2001) as when "the medical community began a search to understand and prevent sudden infant death syndrome." Along with Adelson & Kinney, 1956.

Sudden and unexpected death in infancy and childhood. Adelson L, Kinney ER. Pediatrics 1956;17:663-697

Sudden death related to spinal injury. Towbin A. Lancet 1967; 2: 940-942 He observed epidural hemorrhages in the cervical spines of five children who had died sudden unexpected death. Caffey (1974) cites this article as support for his (Caffey's) hypothesis that manual whiplash shaking of the head may be a major causal factor in sudden unexplained infant deaths ("cot" or "crib" death) associated with epidural hemorrhages of the cervical spine as found by Towbin.

Prolonged apnea and the sudden infant death syndrome: clinical and laboratory observations.

Steinschneider A. Pediatrics 1972; 50: 646-654. As summarized in 2003 by the AAP Committee on the Fetus and Newborn (*q.v.*), "Apnea documented by cardiorespiratory monitoring during prolonged hospitalizations was reported for 2 infants, both of whom were siblings of 3 infants who had died suddenly at home. Both siblings subsequently died unexpectedly after discharge from the hospital. More than 2 decades later, evidence of infanticide for all 5 infants in the original report became known." The research subjects were the children of Waneta Hoyt. See Abraham B. Bergman, Wrong turns in SIDS research, 1997.

Pulmonary arterial abnormalities in the sudden infant death syndrome. Naeye RL. N E J Med 1973; 289: 1167-1170. Claimed to find pulmonary arterial thickening as an index of chronic hypoxia. Cited by Bergman (Wrong turns in SIDS research, 1997) as having been found to be wrong.

The important need for autopsy standards. <http://sids-network.org/experts/> To help improve uniformity of information, the Division of Maternal and Child Health of the Department of Health and Human Services held a meeting in 1975 which resulted in investigative and autopsy protocols for examining the SIDS infant. These protocols were included in an article entitled, [see below]:

SIDS Research: Part I: An overview of major trends. JFS 1976; 21(4): 833-841. Includes autopsy protocol.

Sudden infant death syndrome (crib death). Guntheroth WG. Am Heart J 1977 Jun; 93(6): 784-793. Occurs during sleep, between 1 and 6 months of age. "There is growing evidence that the victims have had previous hypoxic episodes. Although suffocation is no longer considered a tenable explanation, other forms of airway obstruction are still postulated by many... Arrhythmia has been proposed, including long QT syndrome, but the evidence is against arrhythmia. near-miss apnea is likely as the primary event. Produced by several reflexes. Apnea is common; the crucial aspect appears to be the failure to resume respiration. Failure of alarm/arousal by apnea. If apnea persists for over 45 seconds, a dangerous positive feedback develops into hypoxic apnea. This will persist until one of two things occurs: circulatory failure, or gasping. Gasping is effective autoresuscitation in neonates, but after the neonatal period, gasping is not resuscitative, because the infant has lost the fetal capacity for anaerobic survival. This fetal capacity lasts for about 1 month after birth -- remarkably coincident with the one-month hiatus in SIDS occurrence. So SIDS is apnea, and infants who have had near-miss SIDS should be monitored.

SIDS in infants with BPD. (markedly increased risk). Werthammer J, Brown ER, Neff RK et al. Pediatrics 1982; 69: 301-304.

Fatty liver in sudden childhood death. Bonnell H and Beckwith B. Am J Dis Chil 1986 Jan; 140(1): 30-33. Studied that autopsy livers of 21 children who died suddenly either by trauma or by natural causes, from the point of view of asking whether fatty liver was diagnostic of Reye's syndrome. Found that many of the children had fatty livers. None of them had Reye's syndrome. Argues that fatty liver is ubiquitous in children.

Elevated levels of hypoxanthine in the vitreous humor indicate prolonged cerebral hypoxia in victims of sudden infant death syndrome. Rognum TO, Saugstad OD, Oyasaeter S, Olaisen B. Pediatrics 1988; 81: 395-398. This finding is relied on by Toro et al. (2006) to correlate a finding of increased extramedullary hematopoiesis in SIDS livers with a possible history of intrauterine asphyxia. It is also referred to by the international consortium seeking standardization in SIDS definitions and autopsy procedures (Bajanowski et al., FSI 2006).

Drug-addicted mothers, their infants, and SIDS. Rosen TS and Johnson HL. Ann NY Acad Sci 1988; 533: 13-30. Higher rate of prematurity, fetal distress, VD, perinatal asphyxia, in these polydrug abusers. Studied 111 pregnant women in a drug abuse clinic. See under "Drug Abuse," below.

Risk factors for SIDS. Results of the NICHD SIDS Cooperative Epidemiological Study. Hoffman HJ, Damus K, Hillman L, Krongrad E. Ann NY Acad Sci 1988; 533: 13-30. Contains the CHIME study, monitoring 1079 infants who either had ALTE or were SIDS siblings and who were monitored for 718,000 hours. Finding that apnea and extreme apnea occurred in all groups of infants, did not cause death. It resolves before they get into the SIDS age range. See discussion in the AAP Policy Statement on monitoring, 2003.

The role of apnea in the SIDS: a personal view. David P. Southall. Pediatrics 1988 Jan; 80(1): 73-84. He describes three mechanisms of apnea, which is defined as "transient absence of ventilation of all or part of the alveolar bed." The mechanisms are (1) absence of inspiratory efforts; (2) partial or complete airway obstruction; (3) prolonged expiratory apnea, a group of respiratory disturbances associated with cyanotic episodes showing rapid onset of severe arterial hypoxemia suggestive of V/Q mismatch.

- (1) Pauses in inspiration are normal during REM sleep and are age-dependent. Abnormally prolonged ones could be this: "A primary failure of the central inspiratory generator in the brainstem... This relatively rare cause of infantile apnea may result from a tumor, a head injury, an infection, or a congenital malformation. There are usually additional neurological signs indicating damage to the surrounding brainstem." 74 Another mechanism for such prolonged pauses could be abnormal chemoreceptor function. Meaning CO₂ receptors. Another could be abnormal bronchial C-fiber receptors in the carina. Another form could be abnormal suprapontine inhibition of the central inspiratory generator. As far back as 1899 Hughlings Jackson showed that sz activity in the limbic system could inhibit inspiratory drive.
- (2) Partial or complete airway obstruction. This happens normally with valsalva as in crying, etc. Otherwise subglottic stenosis, Pierre-Robin syndrome, viral croup, hypotonia of the upper airway. This occurs in cerebral palsy, Down's, Prader-Willi, but also in apparently healthy infants. This is a form of obstructive sleep apnea. It could have an anatomic basis in the skull-neck area. The Q of G-E reflux has been raised. But there has been a lack of correlation with apnea on sleeping polygraphs. Another form of this airway obstruction mechanism is imposed suffocation.
- (3) Prolonged expiratory apnea. Or "sudden atelectasis apnea braking syndrome," because it is thought to be triggered by atelectasis. These cyanotic episodes persist despite tracheal intubation, so the defect is thought to lie in the alveolar bed or bronchi.

Now what is the relationship of all this to SIDS? (A) Steinschneider in 1972 studied a family with prolonged pauses in insp and both of these babies subsequently died suddenly without adequate explanation. MGH investigated large numbers of ALTEs using pneumography in 1978 and found that some of these babies had prolonged pauses and other abn. Other studies. Mass confusion. Overlap with normals. Home monitoring, theophylline, etc. Southall did a study 1983-86 of cyanotic episodes in 133 symptomatic children under 5. They were able to record a cyanotic or apneic episode in 52 children. Of these, 41 had prolonged expiratory apnea. 7 had sleep-related airway obstruction. 2 had smothering. 1 had sz. 1 had prolonged pauses of unknown cause. Thus, prolonged expiratory apnea was the most common. Then, let's not forget the special cases of apnea due to Arnold-Chiari malformation, myelomeningocele, spina bifida cystica, tumors, and other anatomic disorders of the medulla and upper cervical spinal cord. Let's also not forget TEF, even status post repair. (B) Prospective investigation of subsequent SIDS victims was done. Steinschneider in 1982 published a prospective polygraphic study of 1301 infants showing increased apneic pauses in a subgroup of subsequent SIDS victims. From 1979 to 1982 Southall studied 10,000 infants in followup post hospital discharge, of whom 29 subsequently had SIDS. They did hospital and followup 24hr ECG and pneumogram recordings. In term infants the future SIDS victims had significantly higher average heart rates. The preterm future SIDS victims had more episodes of bradycardia. Another study of 9,856 infants by Southall published 1987 implicated prolonged expiratory apnea in SIDS in two out of

five children who died of SIDS. (C) Animal studies. (D) Epidemiologic data: SIDS increased in infants of smoking mothers, drug addicted, LBW, premature, multiple gestation, male, and BPD. Overlap with the risk factors for fatal respiratory infections between 6 weeks and 6 months. From Kelly et al, 1982, Shannon et al, 1982, 1982a. Also 3/4 of SIDS victims had a minor respiratory tract infection in the days preceding death. SIDS cases cluster around RSV and pertussis outbreaks. "If the abnormal apnea hypothesis is to be tenable, it must conform to this epidemiology." 80. Does the pattern of occurrence of prolonged expiratory apnea fit this epidemiology? Well, it may; on theoretical grounds, an alveolar disorder would fit with the possible effects of these known factors on alveolar development or alveolar function at the relevant ages and in the relevant family settings. The BPD correlation is a parenchymal lung disorder and would fit with prolonged expiratory apnea epidemiologically. (See Werthammer, 1982 for BPD correlation). The other mechanisms of apnea are too uncommon to correlate epidemiologically. Autopsy pathology: pulmonary edema in 90% and petechiae in 75%. Krous pointed out that the petechiae occur only on the membrane surfaces and not in the parenchyma of the heart or lungs or diaphragm or chest wall. (But they do occur in the parenchyma of the thymus gland -- JKR). Beckwith suggested this pattern is consistent with large negative intrathoracic pressures in the presence of airway obstruction. Beckwith et al, 1970. This pattern, Southall thinks, could fit with "as in prolonged expiratory apnea, they could result from large inspiratory efforts in an attempt to reinflate large areas of atelectatic alveoli." 81. (But grossly we don't see such areas. -- JKR). Isolation of RSV in "relatively high" proportion of SIDS and autopsy evidence of minor infection in 75%. Uren et al, Med J Austr 1980.

In summary, the epidemiologic and pathologic evidence "would be compatible with [prolonged expiratory apnea with V/Q mismatching] as the final mechanism of death in a significant proportion of [SIDS] cases." Other forms of apnea could only account for very small numbers or do not fit the typical SIDS history.

Severe hypoxaemia in pertussis. Southall DP, Thomas MG, Lambert HP. Arch Dis Child 1988; 63(6): 598-605. Cited by Vennemann et al. (2007) on vaccination, for the proposition that B. pertussis infection in infants frequently causes apnea.

Reactive astrocytes and macrophages in the brainstem of SIDS victims? Eleven age- and sex-matched SIDS and control cases. Oehmichen M, Linke P, Zilles K, Saternus KS. Clin Neuropathol 1989; 6: 276-283. Reactive astrocytes were more found in the SIDS than in the controls.

Desquamative interstitial pneumonia in an infant: mimicry of sudden infant death syndrome. Irwin L. Golden and Russell Sherwin. Am J Forens Med Pathol 1989 Dec; 10(4): 344-348. Case report of a 3 1/2 month old black female born at 33 weeks with three weeks NICU for neonatal sepsis, fed at 4:00 a.m., cosleeping with both parents, found unresponsive at 7:00. Has aggregates of macrophages in many alveoli and an interstitial mononuclear (and ?PMN) infiltrate.

Cigarette smoking as a risk factor for SIDS: a population-based study. Haglund B, Cnattingius S. Am J Publ H 1990; 80(1): 29-32.

Signs of illness preceding sudden unexpected death in infants. Gilbert RE, Fleming PJ, Azaz Y, Rudd PT. BMJ 1990; 300 (6734): 1237-1239. Cited by Vennemann et al., 2007 in their discussion of the protective effect of vaccination, to the effect that "The seasonal distribution in the occurrence of SIDS and the high prevalence of respiratory tract symptoms suggests infection is a factor in SIDS." As to infective agents, see Rambaud et al., 1999.

Prospective cohort study of prone sleeping position and SIDS. Dwyer T, Ponsonby A-LB, Newman NM, Gibbons LE. Lancet 1991; 337: 1244-1247.

Defining SIDS: deliberations of an expert panel convened by the National Institute of Child Health and Development. Willinger M, James LS, Catz C. Pediatr Pathol 1991; 11: 677-684. This gives the rationale for the new (1989) definition of SIDS involving scene examination and clinical history review. See "Definition," above.

Pathologic findings in SIDS. Berry PJ. J Clin Pathol (Br) 1992; 45(Suppl): 11-16

Combined effect of infection and heavy wrapping on the risk of sudden unexpected infant death. Gilbert R, Rudd P, Berry PJ, Fleming PJ et al. Arch Dis Child (Br.) 1992; 67: 171-177. Proposes an Avon Clinicopathologic classification system:

- I-A: No abnormal findings
- I-B: Noncontributory findings
- II-A: Findings which may have contributed to death
- II-B: Extensive or severe findings but not a complete explanation for the death
- III: Death fully explained by the findings.

The sudden infant death syndrome: pathologic findings. Valdes-Dapena MA. Clin Perin 1992; 19(4): 701-716.

Bacterial toxins: a possible cause of cot death. McKendrick N, Drucker DB, Morris JA, Telford DR, Barson AJ, Oppenheim BA, Crawley BA, Gibbs A. J Clin Pathol (Br) 1992; 45: 49-53 (have). These guys performed nasopharyngeal swab cultures on 22 matched pairs of SIDS and live normal controls. 68% of SIDS had toxigenic isolates, compared to 36% of controls.

Relationship of SIDS to maternal smoking during and after pregnancy. Scheondorf KC, Kiely JL. Pediatrics 1992; 90: 905-908. Cited by the CDC in "Guidelines" (1996) along with Haglund, above, for the proposition that maternal smoking is one of the "most significant" risk factors.

Sleeping prone and the risk of SIDS. Guntheroth WG, Spiers PS. JAMA 1992; 267(17): 2359-2362.

Development of thermoregulation in infancy: possible implications for SIDS. Fleming PJ, Azaz Y, Wigfield R. J Clin Path 1992 Nov; 45(11 suppl): 17-19. Metabolic rate rises during the first three months and fatpad thickens, and peripheral vascular sympathetic response to cold improves. 40% of the body's heat production at this age occurs in the head. In animal studies, small changes in the temperature of the hypothalamus have strong effects on control of respiration. Consider the possible effects of an overheated head. This is the Thermoregulation Hypothesis. See Guntheroth & Spiers, 2001.

AAP Position Statement: Task Force on Infant Positioning and SIDS. Kattwinkel J, Brooks J, Myerberg D. Pediatrics 1992 Jun; 89(6 part 1): 1120-1126. Healthy infants should be put on the back or side. 4 tables, 70 references. Questioned by Hunt & Shannon in next ref. But note that SIDS incidence has fallen since this position statement was issued, and the fall began at or a little before that year. I wonder if this would cause Hunt & Shannon to revisit their views, *infra*. Note that this Position Statement has been superseded by a new Policy Statement as of March 2000. See below.

SIDS and sleeping position. Hunt CE and Shannon DC. Pediatrics 1992 Jul; 90 (1 part 1): 115-118. Pointing out that the prone-SIDS association is an association, not a causality. Pointing out that if we imply that it is a causality, (a) parents will blame themselves or other caregivers for prone positioning as if it "caused" the death. (b) parents whose infants died anyway even though they were supine will fall under needless suspicion and will be confused. (c) The supine position also has risks. JKR note: even though SIDS incidence has fallen by 40% since the 1992 advisory and the 1994 government "back-to-sleep" campaign, and even though this fall is widely attributed to those policy interventions, Hunt & Shannon's arguments are still of interest. Their predictions (a) and (b) have been borne out by events.

Is sudden infant death syndrome a cause of death? Gilbert-Barness E. Am J Dis Child 1993; 147: 25-26. Cited by Sawaguchi and Nishida (2001) for the proposition that the diagnostic entity of SIDS serves no constructive purpose. See also Huber, below.

Sudden infant death syndrome: the new clothes of the emperor. Eur J Pediatr 1993; 152: 93. The diagnostic entity of SIDS serves no constructive purpose and is confusing.

Prone sleeping position and sudden infant death (editorial). Poets CF and Southall DP. N E J Med 1993 Aug; 329(6): 425-426

Sudden death in infants under one year of age. Czegledy-Nagy EN, Cutz E, Becker LE. Pediatric Pathology 1993 Sep; 13(5): 671-684. This retrospective Toronto study of 101 consecutive SIDS cases. They stratified the SIDS cases into the following three strata or types:

A-1 term infants 2wks to 6 mos old with completely negative autopsies

A-2 preterm same

B misc minor clinical or pathologic abnormalities present (mostly minor URI)

A-1 was 39%, A-2 was 2%, B was 59% of SIDS. Authors argue that a more reliable SIDS database could be obtained by separating out the A and B cases. Viz. Bruce Beckwith's similar view. JKR: Stratification of infant deaths by social and domestic circumstances is also being discussed, and is a much more lively possibility than this purely anatomic stratification. See "SIDS Epidemiology."

Factors potentiating the risk of SIDS associated with the prone position. Ponsonby AL, Dwyer T, Gibbons LE et al. N E J Med 1993 Aug; 329(6): 425-426. The potentiating factors were (1) natural-fiber mattress, (2) swaddling, (3) recent illness, (4) heated bedroom.

Infant positioning and SIDS: a selected annotated bibliography. National SIDS Resource Center, 1995? (have). Thirty six pages of annotated references, of which the most recent are to 1995.

Minor inflammation lesions and sudden infant death: cause, coincidence, or epiphenomena? Byard RW and Krous HF. Pediatr Pathol Lab Med 1995; 15: 649-654.

The effect of passive smoking and tobacco exposure through breast milk on SIDS. Klonoff-Cohen HS, Edelstein SL, Lefkowitz ES, et al. JAMA 1995; 273(10): 795-798.

The pathologist's contribution to the prevention of cot death, and why it is important to abolish the concept of SIDS as a nosological entity. Huber, J. In: Rognum TO, ed. Sudden infant death syndrome: new trends in the nineties. Boston: Scandinavian University pPress, 1995: 46-49. Cited by Sawaguchi and Nishida in Am J Forens Med P 2001 Jun (see below) as saying that the concept was originally adopted (1978) "without a strong justification."

The pathological approach to sudden infant death -- consensus or confusion? Recommendations from the Second SIDS Global Strategy Meeting, Stavanger, Norway, August 1994... Am J Forens Med Path 1996 mar; 17(2): 103-105. Argues for more detailed invest and tissue retention.

Guidelines for Death Scene Investigation of Sudden Unexplained Infant Deaths: Recommendations of the Interagency Panel on Sudden Infant Death Syndrome. MMWR Recommendations and Reports, June 21, 1996. Vol 45, no. RR-10. (have).

Positioning and SIDS: Update. AAP policy statement RE9647. Pediatrics 1996 Dec; 98(6): 1216-1218. Back is best, but side is ok.

Does bed-sharing affect the risk of SIDS? AAP Policy statement RE 9725. Pediatrics 1997 Aug; 100(2): 272. By mother is ok if no soft bedding. By others may incr risk of SIDS. Unclear.

Cerebellar cortex delayed maturation in SIDS. Cruz-Sanchez FF, Lucina J, Ascaso C et al. J Neuropathol Exp Neurol 1997 Apr; 56(4): 340-346 Delayed decline in the number of external granular layer neurons.

SIDS and parental smoking: a literature review. Golding J. Paediatr Perin Epid 1997 Jan; 11(1): 67-77

Detection of RSV nucleic acid in archival postmortem tissue from infants. Cubie HA et al (Edinburgh). Pediatr Pathol Lab Med 1997 Nov-Dec; 17(6): 927-938. 99 cases if SIDS and 58 known cause of death. In-situ hybridization. Found in 24 % of SIDS and 19% of comparison group. Higher detection rate in winter in both groups.

Bacterial toxins and sudden unexpected death in a young child. Bentley AJ et al FSI 1997 Aug; 88(2): 141-146 Found STSS toxin in brain tissue of a six year old who died fol resp infec

Sudden infant death and prevalence of whooping cough in the Swedish and Norwegian communities. Lindgren C, Milerad J, Lagercrantz H. Eur J Pediatr 1997; 156(5): 405-409. Cited by Vennemann et al. on vaccination's protective effect (2007) for the proposition that Bordetella pertussis may be especially important as an association between epidemic pertussis and SIDS. See Southall et al., 1988, above.

Unexpected death in two young infants mimics SIDS: autopsies demonstrate tumors of the medulla and heart. Rajs J, Rasten-Almqvist P, Nennesmo I. Am J Forens Med Path 1997 Dec; 18(4): 384-397. One baby had a small diffuse infiltrating oligo of the medullary reticular formation. The other one had a large cardiac fibroma.

Sudden infant death syndrome: postmortem findings of nicotine and cotinine in pericardial fluid of infants in relation to morphological changes and position at death. Rajs J, Rasten-Almqvist P, Falck G, Eksborg S, Andersson Bs. (Stockholm) Pediatr Pathol Lab Med 1997 Jan-Feb; 17(1): 83-97. Pericardial fluid of 67 SIDS and 18 non-SIDS infants had no statistically significant difference in pericardial nicotine/cotinine levels. However, only SIDS victims had cotinine levels above 30 ng/ml, and these also had myocardial necrosis and liver necrosis and were cosleeping.

Combined effects of sleeping position and prenatal risk factors in sudden infant death syndrome: the Nordic Epidemiological SIDS Study. Oyen N, Markestad T, Skaerven R et al. Pediatrics 1997; 100: 613-621. Cited by William T. Basco, Jr., MD, FAAP in Medscape Viewpoint Preterm Infants and Sleeping Position, Medscape Pediatrics 2006; 8(2), posted 10/3/2006 for the demonstration that in preterm infants, prone sleeping position is associated with a marked increase in rates of SIDS. In reviewing an art by Bhat et al. in Pediatrics 2006 (see below) which finds that prone preemies had much more apneas and less arousal than the same preemies supine.

Neo-Gen Screening memorandum, July 1998

Re congenital metabolic diseases on filter paper postm blood. List of dxs possible. In SUDS file.

Metabolic causes of sudden and unexpected death in early life. (have) Piero Rinaldo, MD., Associate Professor of Genetics and Pediatrics, Director, Biochemical Disease Detection Laboratory, Yale University School of Medicine, Department of Genetics, 333 Cedar Street, New Haven, Conn. 06520. (203)-785-7659. August 1997 issue of the OAA Newsletter. <http://www.oaanews.org/oaais2/article5.htm> Also see OAAnews@AOL.com. "OAA" stands for "Organic Acidemia Association." Fatty acid oxidation plays a major role in energy production during periods of fasting. The vast majority of these disorders present with life-threatening episodes of metabolic decompensation (acidosis with or without ketosis, hypoglycemia, hyperammonemia, etc.), often within the first year of life.

- (1) Autopsy procedures: (a) skin collection good for culturing fibroblasts up to 48h post-death.
- (b) fluids either frozen or dried. (c) frozen liver good up to 72h post-death.
- (2) Diagnostic protocol: (a) liver histol showing fatty. (b) Abnormally elevated concentrations of C8-C18 fatty acids and glucose in liver. (c) elevated total and free carnitine in liver and bile.
- (d) Abnormal acylcarnitine profile in bile. (e) acylglycines and organic acids in urine.
- (3) Inborn errors in SIDS:
 - a. Biotinidase deficiency
 - b. Methylmalonic acidurias
 - c. Hydroxymethylglutaryl CoA lyase deficiency
 - d. Glutaric aciduria type 1 (GA-1) (See "Shaken," below)
 - e. Disorders of energy metabolism
 - f. Fumarase deficiency
 - g. Mitochondrial respiratory chain defects
 - h. Fatty acid oxidation disorders
- (4) Fatty acid oxidation disorders

Gives a lengthy table

"[I]t has been postulated that FAO disorders might be responsible for 3 to 5% of SIDS cases, and possibly a much greater proportion of children who die suddenly and unexpectedly from birth to five years of age."

Prone position may increase temperature around the head of the infant. Oriot D, Berthier M, Saulnier JP et al. *Acta Paediatr* 1998 Sep; 87(9): 1005-1007

Prolongation of the QT interval and the SIDS. Schwartz PJ, Stramba-Badiale M, Segantini A, et al. *N E J Med* 1998; 338: 1709-1714.

A molecular link between the SIDS and the long-QT syndrome. Schwartz PJ, Priori SG, Dumaine R, et al. *N E J Med* 2000; 343: 262-267.

Prolongation of the QT interval and the SIDS. (Editorial). Jeffrey A. Towbin and Richard A. Friedman. *NE J M* 1998 Jun 11; 338(24): 1760-1761. Says incidence 1.6/1000 LB in US, 0.7 in Italy, 2.5 in UK. "In this issue Schwartz and colleagues present compelling evidence that the long QT syndrome should be considered an important factor in the pathogenesis of SIDS." They took ECGs on day 3 of life on 34,000 Italian newborns. 24 died of SIDS. The SIDS infants had a longer QTc than the nonsids survivings or the nonsids deaths (10 nonsids deaths). SIDS QTc=435 +/-45msec, nonsids QTc=400+/-20. P,.05. The definition of a "prolonged" QTc is 440 msec. Editors recommend genetic screening (for defective ion-channel genes) of fams w prev SIDS, ALTE, or prol QT. See next below. See the very negative comments on this article in *Pediatrics* for April 1999, below. In Letters, *N E J Med* 2000 Dec 21; 343(25): 1896-7, three German guys present their work showing that long-QT infants have fetal bradycardia. (Beinder et al.) Jon Skinner of Auckland points out that even if the gene and the condition are present when SIDS occurs, this doesn't mean that an earlier screening ECG would have shown it, or indeed that the presence of the gene necessarily causes a long QT. Also quoting Schwartz's own work showing that beta blockers do not prevent cardiac events in this group. Phoon points out the high rate of false positives in screening ECG's at that age. The authors reply. Note that this LQTS has been linked to a specific gene, SCN5A, and that gene can be independently tested for.

Comments on a SIDS article in another journal. Jerold F. Lucey, MD, Editor in Chief, *Pediatrics*. *Pediatrics* 1999 Apr; 103(4): 812. Compares the LQTS work to the earlier specious work on apnea which led to the useless fad of apnea monitors. Says the statement that "some patients should receive beta blockers" is alarming. Says many flaws in the data. He recruited several other specialists to comment. One, the head of the pediatric pulmonary unit at MGH, adduces the following flaws in the study: (1) The at-risk infants had no heart block or Vtach. (2) the measurements were only visual on five beats, not by computer. (3) not blinded. (4) need to know SENS and SPEC of his test in order to evaluate the risks and likely benefits of chemotherapy for the 72 infants who have a + test, of whom 1 is statistically expected to get SIDS. Southall comments that no details are provided on the autopsies. Also comments on technical aspects of the ECG recordings. But note, that later there has been publication by Schwartz of a molecular link between LQTS and SIDS.

[Near-miss SIDS caused by long-QT syndrome] Peter J. Schwartz et al., *N E J Med* 2000 Jul 27; 343: 262-267. Case report of a 44 day old infant found not breathing by his parents. Resuscitated and found to have a marked prolongation of the QT. Negative family history and the parents' ECG's were normal. The child had a gene mutation in exon 16 of the coding sequence of SCN5A, the gene for the long QT syndrome. The parents were genetically normal.

Reducing the risk of SIDS: a review of the scientific literature. Henderson-Smart DJ, Ponsonby AL, Murphy E. *J Paediatr Child Health* 1998 Jun; 34(3): 213-219. Australia. Covering baby's head is strongly related to SIDS. Bedsharing among smokers.

The value of a thorough protocol in the investigation of sudden infant deaths. Sadler DW. (UK) *J Clin Pathol* 1998 Sep; 51(9): 689-694. These guys grew out *Pneumococcus* or *Hemophilus* from the CSF in 16% of cot deaths. They advocate early CSF culture in all SIDS.

Brainstem 3H-Nicotine receptor binding in the SIDS. Nachmanoff DB, Panigrahy A, Filiano JJ et al. J Neuropath Exp Neurol 1998 Nov; 57(11): 1018-1025. In-utero cigarette-exposed SIDS infants failed to upregulate their nicotine binding at three cardiorespiratory centers in the brain stem, suggesting maldevelopment.

Objective measurements of nicotine exposure in victims of SIDS and in other unexpected child deaths. Milerad J, Vege A, Opdal SH, Rognum TO. (Stockholm). J Pediatr 1998 Aug; 133(2): 232-236. The smoking association factor has been based on self-report histories by mothers. These authors tried to quantitate it objectively by measuring cotinine levels in postmortem pericardial fluid from all the sudden child deaths in southeastern Norway aged 0 to 7 yrs: 24 SIDS, 12 infections, and 9 accidentals. $P < .05$.

	Exposed	Heavily exposed
SIDS	92%	25%
Infec	67%	0%

Median cotinine levels

SIDS	15.8 ng/ml
Infec	7.1 ng/ml
Acc	12.9 ng/ml

Sudden natural death in infants and young children. Handy TC and Buchino JJ. Clin Lab Med 1998 Jun; 18(2): 323-338

SIDS and early family interpersonal relationships. Lindsay JS. Medical Hypotheses 1999 Apr; 52(4): 315-317. Has the human neonate evolved to have some needs (meaning some vital needs) that must be worked out between a mother and her baby?

Human herpesvirus-6 and sudden death in infancy: report of a case and review of the literature. Hoang MP, Ross KF, Dawson B, Scheuermann RH and Rogers BB. JFS 1999; 44(2): 432-437. This relatively newly described virus causes febrile illness in early childhood. This 7 month old boy had at autopsy atypical lymphoid infiltrates in the liver, kidney, heart, spleen, lymph nodes, and bone marrow, associated with erythrophagocytosis. HHV-6 was detected by PCR and by in-situ hybridization on formalin-fixed paraffin-embedded tissue.

Microbiology in SIDS and other childhood deaths. Rambaud C, Guibert M, Briand E, Grangeot-Keros L, Coiulomb-L'Hermine A, Dehan M. FEMS Immunol Med Microb 1999; 25(1/2): 59-66. Cited by Vennemann et al. (2007) to the effect that a number of different viruses and bacteria have been implicated in SIDS.

Maternal placental abnormality and the risk of SIDS. Li DK and Wi S. Am J Epidemiol; 1999 Apr; 149(7): 608-611. California 2,107 SIDS cases via DC and 21,000 control infants. SIDS infants had a 1.4% incidence of placental abnormality (abruption or previa), while 0.7% of controls, odds ratio of 2.1

Review of hazards associated with children placed in adult beds. Arch Ped Adol Med 1999; 153: 1019-1023. Retrospective review of CPSC data from 1990-1997. "Placing children younger than 2 years to sleep in adult beds exposes them to potentially fatal hazards that are generally not recognized by the parent or caregiver."

Progress in reducing risky infant sleeping positions -- 13 states, 1996-1997. MMWR 48(39): 878-882, 1999. (CDC). They assessed the response to the AAP's "Back to Sleep" campaign initiated in 1994, using the Pregnancy Risk Assessment Monitoring System previously set up to study maternal mortality (q.v.) Finds in brief that there remains a high degree of geographic and demographic variation in how this campaign is working. Generally, "substantial progress," As far as race, found that Black mothers were significantly more likely to put their infants on their stomachs (about 2 X more likely). Stomach sleeping

was also more prevalent among the poor. Also it was more common in the middle Atlantic and Southern states. Identifies the need to develop group-targeted prevention programs.

The amount of brainstem gliosis in SIDS victims correlates with maternal cigarette smoking during pregnancy. Storm H et al. Acta Paediatrica 1999; 88: 13-18. In reviewing this in Child Abuse Quarterly, Dr. Krous notes that, "Smoking has emerged as the major remaining risk factor for SIDS."

Changing infants' sleep position increases risk of SIDS. Mitchell E, Thach B et al. Arch Ped Adol Med 1999 Nov; At six months, infants shift over from reflex protection of the airway to learned protection. They also learn to roll over at this time. During this transition they are vulnerable to sudden death if their sleep position is changed from back to prone during this transition. This study found that SIDS was 18 times more likely if the infant's sleep position changed from back to prone during this time interval, particularly if there was soft bedding. Study in New Zealand.

Sudden infant death syndrome and early family interpersonal relationships. JSB Lindsay. Medical Hypotheses 1999; 52(4): 315-317. (have) Confusing and vague article that basically argues that infant isolation, particularly from the smell of the parents, could be a factor. Mentions that female infants are weaned earlier and held less than males in our culture, but does not explain why females have a lower SIDS rate. Confusing.

The amount of brainstem gliosis in SIDS victims correlates with maternal cigarette smoking during pregnancy. Storm H, Nylander G, Saugstad OD. Acta Paediatrica 1999; 88: 13-18.

Changing concepts of SIDS: implications for infant sleeping environment and sleep position. AAP Policy Statement (RE9946). Pediatrics 2000 Mar; 105(3): 650-656. (have) Reviews the evidence about prone sleeping and considers reducing other remaining risk factors. <http://www.aap.org/policy/re9946.html> Supersedes and replaces the position statement of 1992. Goes against side sleeping because they can roll. It in no way concedes any ground at all to Hunt & Shannon's criticisms of 1992 re making people feel guilty. Also goes into the following other modifiable risk factors for SIDS:

- Soft bedding
- Overheating
- Smoking
- Bedsharing
- Prematurity/LBW

Also mentions possible protective factors, such as pacifiers and breastfeeding. Also discusses a differential diagnosis including:

- Infanticide
- Arrhythmia

Also discusses proposed mechanisms, as hypothesis of delayed development of arousal mechanism because of delayed development of serotonin receptors in the arcuate nucleus and now other brainstem sites, a site thought to be involved in the hypercapnic response according to Hannah Kinney and co-workers. [The main ref for this research is given next below.] Gives ten recommendations.

[brain stem development in SIDS]. Kinney H et al. Journal of Neuropathology and Experimental Biology 2000; 59(5): 377-384.

[Link between smoking and SIDS confirmed] Wisborg K et al. at Aarhus, prospective questionnaire study of the mothers of 25,000 live births. 20 cases of SIDS for a SIDS rate of 0.8 per 1000. The infants of women who smoked had 3X the risk, even when adjusted for parity, alcohol, caffeine, maternal BMI, social class, marital status, and PNC. The risk of SIDS increased with the number of cigarettes. Suggest that cessation of smoking in the society could reduce SIDS mortality by 40% overall. Arch Dis Child 2000; 83: 203-206, reported in Medscape by Reuters Health.

[SIDS risk highest for infants of single mothers and mothers under 20] (Reuters Medical News on Medscape Sep 2000). A death certificate study from the UK government found that SIDS was 5X more likely if the birth certificate was signed only by the mother. This study was criticized by the Foundation for Infant Deaths, a private group, on the ground that , “Increasing numbers of baby deaths are being registered as ‘unascertained’ rather than cot death...which means they are not being included in the government cot death statistics. ...The number of baby deaths registered as ‘unascertained’ has increased more than five-fold since 1994,” the Foundation says. Foundation therefore suspects that the real incidence of SIDS is higher than the DC’s indicate.

Environmental tobacco smoke and sudden infant death syndrome. Kirsten Waller, MD, MPH. Calif Dept of Health Services. ARHP Clinical Proceedings (American Reproductive Health P), 2000. (abstract) (have) <http://www.arhp.org/clinical/clinical1/sids.htm> A pooled odds ratio of 3.0 for maternal smoking. But what about environmental tobacco smoke in general? This is a literature review. Two studies found increased risk with paternal smoking, but they weren’t able to control for maternal smoking. More studies are needed that control for other SIDS risk factors. 41% of all US children aged 2 months to 3 years live with at least one smoker. Only one study tested for cotinine (Milerad et al., 1994); it found cotinine in the pericardial fluid of 56% of sixteen consecutive SIDS autopsies. Concludes that there is growing evidence that points to ETS (environmental tobacco smoke) as an independent risk factor for SIDS. A constant positive association despite varying variables.

Decreased serotonergic receptor binding in rhombic lip-derived regions of the medulla oblongata in the SIDS. Panigrahy A, Filiano J, Sleeper LA et al. J Neurop Exp Neurol 2000; 59: 377-384. Showing that serotonin system is involved in SIDS deaths. See also Kinney et al., 2001 (below); Okado et al., Medical Hypotheses, 2002;

H. pylori may be implicated in SIDS. Reuters Medical News on Medscape Oct. 2000 citing work by Dr. Jonathan R. Kerr and coworkers of the University of Manchester in Archives of Diseases in Childhood 2000; 83: 429-434 to the effect that they got DNA for two H. pylori genes out of stomach, trachea and lung from 28/32 SIDS victims and 1/8 controls. He proposes that if this organism is aspirated into the lungs, its urease can liberate ammonia resulting in an ammonia intoxication. Normal H. p prevalence is under 2% and here it was in 88% of SIDS.

Unsafe sleep practices and an analysis of bedsharing among infants dying suddenly and unexpectedly: results of a four-year, population-based, death scene investigation study of sudden infant death syndrome and related deaths. Kemp JS, Unger B, Wilkins D, Psara RM, Ledbetter TL, Graham MA, Case M, Thach BT. Pediatrics electronic pages 2000 Sep; 106(3): e41. See below under “Cosleeping.”

Medullary serotonergic network deficiency in the SIDS: review of a 15-year study of a single dataset. Hannah C. Kinney, James J. Filiano, and W F White. J Neurop and Experim Neurol 2001 Mar; 60(3): 228-247. Propose that a subset of SIDS is due to an abn of the ventral medulla and medullary reticular formation , caudal raphe and arcuate nucleus (putative homologue of the rhombic lip-derived cat respiratory chemosensitive fields) lead to a deficiency of serotonergic response to hypercapnic or hypoxic stress during a critical period of development. This group of nuclei and their connections to the phrenic n. and hypoglossal n. proposed to represent a unified respiratory-related complex, which they term the “medullary serotonergic network.” Impaired gasping response to hypoxia (autoresuscitation), etc. Impaired hypoglossal response to prone? But this is all biochemical; there are no anatomic lesions for most of this. Well, that’s not exactly true; there is a higher 5-HT neuron count and a higher 5-HT neuron density in the medulla. See the 2006 San Diego study by Paterson et al., below.

[Bedsharing contributes to SIDS-like deaths.] Carroll-Pankhurst C and Mortimer EJ Jr., Pediatrics 2001 Mar; 107: 530-536. Reuters Medical News on MedScape, March 29, 2001. Retrospective review of 84 so-called SIDS deaths from Cleveland, 1992-96. 30 reported bedsharing. 54 either had no information or reported sleeping alone. Bedsharing deaths were significantly younger: 9.1 weeks versus 12.7 weeks. The mean pregravid weight of the bedsharing mothers was higher, 84 kg versus 67 kg. “This study provides ev of a rel btw some SIDS-like deaths and parent-infant bedsharing,

particularly if the parent is large.” “Because they are significantly younger at the time of death, the group of bedsharing infants is very likely to include cases of infant deaths that are not SIDS, but rather accidental suffocation resulting from full or partial overlaying, or another form of airway obstruction.” “It is important to distinguish that we do not feel that bedsharing causes SIDS, but rather SIDS-like deaths.” Conversely, the new findings do not suggest that all SIDS deaths that involve bedsharing are the result of suffocation. Said that “when it becomes possible” to distinguish between SIDS deaths and SIDS-like deaths caused by suffocation during bedsharing, then we will have a “pure pool” of true SIDS deaths on which to conduct research on causes. Says, physicians should explain to their patients that bedsharing poses some risk, even if it is very slight. See next.

SIDS in UK not linked to immunization. Fleming PJ. BMJ 2001 Apr; 322: 822-825. (have) The DPT/OPV period is exactly the same as the SIDS period.. But this study of 303 SIDS infants, 65 NAT infants, and 1515 living controls found that immunization was strongly associated with *lowering* of the statistical risk of SIDS. But after controlling for sleep environment, there was no statistically significant association one way or the other. What does this mean? It means that sleep environment is such a strong risk factor that it reverses the otherwise strong statistical favorable effect of immunization. –JKR

Thermal stress in SIDS: is there an ambiguity with the rebreathing hypothesis? Guntheroth WG and Spiers PS. Pediatrics 2001 Apr; 107(4): 693-698. (Seattle) (literature review) Is it rebreathing or overheating? It's overheating. Caused by infections, room heat, overdressing, and prone position. Heat loss from the anterior surface of an infant is much faster than from the posterior surface. They claim they were able to solve for the co-risk-factors for rebreathing. “Although some of the risk factors for rebreathing could be explained by the effects of thermal stress, several [risk] factors for thermal stress could not be explained by the rebreathing hypothesis.” “A strong association between thermal regulation and ventilatory control was found for prolonged apnea.” They want overheating to be added to the Back to Sleep campaign. My initial comment: The risk would be that if you send a double message, you start to lose your audience. On the other hand, if these guys are right, it could save lives. In fact, it might move the incidence of SIDS down a whole nother notch, which would be a major blow against the syndrome. Our scene invest protocol includes heat-related items, and also under meds notes Tylenol intake.

Editorial. Alan H. Jobe, MD, PhD. JAMA 2001 May 2; 285(17): 2244-2245. Apparently says that 2% to 5% of SIDS cases are “caused by infanticide.” (citing the AAP 2001 statement in Pediatrics) Also that the “standard” coroner's autopsy will not reveal the real COD in many cases.

Medullary serotonergic network deficiency in the SIDS: review of a 15-year study of a single dataset. J Neurop Exp Neurol 2001 Mar; 60(3): 228-247. Kinney HC, Filiano JJ, White WF. This 24-page monograph the result of 15 years of grant-supported anatomical work on the medullae of 52 SIDS victims and 15 “acute controls” and 18 chronic cases with a history of an oxygenation disorders, plus 5 fetal brainstems and 4 adult brainstems for baseline normal-development study. Testing an initial hypothesis that a subset of SIDS results from a failure of something in the ventral medulla arcuate nucleus in mediating homeostatic responses to life-threatening challenges such as asphyxia, hypoxia, or hypercapnea during sleep. That initial hypothesis was later refined to posit that a subset of SIDS is caused by a dev abn in a medullary network composed of rhombic-lip-derived serotergic neurons in the caudal raphe and the arcuate nucleus. The caudal raphe means the whole anterior and lateral rim of the medulla. Other workers (Matturi, 2000; Mallard, 1999) have demonstrated hypoplasia of the arcuate nucleus in a few SIDS cases (5% in our laboratory, 30% in Matturi's material.) Such hypoplasia can be diagnosed in conventional sections, but you need at least **ten serial sections**. They do not yet suggest the clinical use of serotonin receptor autoradiography in the diagnosis of this subset of SIDS cases until their work is confirmed by other laboratories. [But it has been confirmed by other laboratories. See the 2006 San Diego Study by Paterson et al., below. –JKR] Autoradiography using tritiated LSD, concentrating on the ventral medulla and the medullary reticular formation, that are thought to influence chemoreception, respiratory drive, upper airway reflexes, and blood pressure responses to stimuli.

Serotonin transporter gene variation is a risk factor for SIDS in the Japanese population. Narita N, Narita M, Takashima S, et al., Pediatrics 2001; 107: 690-692. Reviewed by Henry Krous in Child Abuse Quarterly for Jly 2001. 5-HT transporter is the reuptake component in the brain serotonin excitatory

receptor complexes. Compared frozen brain tissue from 27 SIDS with blood from 115 living controls, used DNA PCR. The L and LX alleles of the 5-HT transporter gene were more frequent in SIDS than in controls. These alleles could give greater reuptake, resulting in lower serot conc in the synaptic space. See Panigrahy, 2000 and Kinney, 2001, above.

Alpha-2 receptor binding in the medulla oblongata in SIDS. Mansouri J, Panigrahy A, Filiano JJ, Sleeper LA, St John WM, Kinney HC. N Neuropathol Exp Neurol 2001 Feb; 60(2): 141-146. Did not find any signif diff in alpha-2 receptor binding between SIDS and control css, sugg that if there is a gasping defect during stress it is not related to this.

The black infant's susceptibility to SIDS and respiratory infection in late infancy. Spiers PS, Guntheroth WG. Epidemiology 2001 Jan; 12(1): 33-37. As infants get older, their SIDS rate tends to parallel their rate of respiratory infections. Sugg that in black infants, particularly older ones, SIDS may be an infectious disease of the respiratory tract. But does not explain why the SIDS age group is younger in Blacks., or why most black SIDS occurs very early.

Paternal marijuana use linked to SIDS. Reuters Medical News on Medscape, July 28, 2001. Citing Klonoff-Cohen H and Lam-Kruglick P (UCSD), Arch Ped Adol Med 2001 Jul; 155: 765-770. Interviewed the parents of 239 SIDS and 239 control live infants and found that after controlling for cigarette smoking, maternal use of "recreational" drugs was not associated with SIDS risk, but fathers' MJ use was at P=.01 to .05.

Postmortem molecular analysis of SCN5A defects in sudden infant death syndrome. Ackerman MJ, Siu BL, Sturner WQ, Tester DJ, Valdivia CR, Makielski JC, Towbin JA. JAMA 2001 Nov; 286(18): 2264-2269. (have) Reporting SCN5A sodium channel gene missense mutation in exon 17 found in frozen myocardial tissue from two of 93 infants diagnosed with SIDS or possible SIDS. "Approximately 2% of SIDS cases appear to be caused by mutations in SCN5A. However, there are other ion channel mutations associated with long QT syndrome that we didn't test for, so it is likely that many more SIDS cases are accounted for by these types of mutations."

Environmental hyperthermic infant and early childhood death: circumstances, pathologic changes, and manner of death. Krous HF, Nadeau JM, Fukumoto RI, Blackburne BD, Byard RW. Am J Forens Med path 2001 Dec; 22(4): 374-382. Ten documented cases -- eight in vehicles and 2 in beds. Age range 53 days to 9 years. Living victims have liver necrosis and DIC. Bodies have intrathoracic petechiae. Since SIDS also have that, and SIDS have been shown by Guntheroth & Spiers to be associated with thermal stress, these may be the extreme end of a heat continuum that includes SIDS. In that case, meticulous scene investigation is required to differentiate.

SIDS doesn't exist. (letter) Sawaguchi T, Nishida H. Am J Forens Med P 2001 Jun; 22(2): 211-212. Says that the term SIDS was first used in 1969. These writers admit that the entity was initially adopted "without a strong justification," and that the arguments of its opponents against it as a conceptual entity are persuasive. But the term is useful as a way of organizing research. They suggest that over time, as various actual etiologies for sudden death are discovered, these discoveries will peel away slices of what is now considered SIDS. Eventually all the deaths may be accounted for by specific etiologies. But in the meantime the term is useful for aggregating all the deaths that are not yet accounted for. The writers tell us that the following authorities oppose using the term "SIDS.:" Professor J. Huber of the Netherlands, 1993, 1995 (see above), Professor Enid Gilbert-Barness, 1993.

Changing concepts of sudden infant death syndrome: implications for infant sleeping environment and sleep position. AAP Task Force on Infant Sleep Position and SIDS. Pediatrics 2001; 107: 650-656. Cited by William T. Basco, Jr., MD, FAAP as the original recommendation that all infants sleep supine. In Medscape Viewpoint, 2006, below. See also the AAP 2005 recommendations, below.

Risk factors for SIDS: changes associated with sleep position recommendations. Paris CA, Remler R, Daling JR. (UW) J Pediatr 2001; 139: 771-777. Found that between 1985 and 1995 the incidence of SIDS as obtained from death certificates in Washington State decreased from 2.6 per 1000 LB to 1.2 per

1000 LB. This retrospective death certificate study asked the question whether the timing of that decrease showed an inflection point around the 1992 issuance of the AAP recommendation for nonprone sleep position. The statistics supported the hypothesis that it did. The authors compared death certificates from the period 1985-1990 with death certificates from the period 1992-1998.

The risk factors for SIDS did not change, but all of the ones other than sleep position increased in magnitude. The risk factors remained

prone sleep position
low birth weight (<2500 grams)
prenatal exposure to cigarette smoke
unmarried mother
Black race
late start of prenatal care

The relative risk for infants born to black rather than white mothers increased over this ten-year period from 1.4 to 2.5. The authors suggest that the nonpositional risk factors act independently of sleep position. (A commentator cites Guntheroth, JAMA 1992 to the effect that the magnitude of the sleep position risk factor is in the range of 3X to 9X relative risk.) Comment by JKR: This type of data (repeated in other studies with or without the prone-sleeping aspect going back to 1968) can be read as saying that once prone infants and unsafe sleep-surface infants are taken out, SIDS is a disease of single cigarette-smoking black households with low birth weight and late prenatal care.

Sudden Infant Death Syndrome: Problems, Progress & Possibilities. Byard RW and Krous HF, eds. New York: Oxford University Press, 2001. The definitive summary as of 2001. It is summarized and updated by a 2003 article, Sudden infant death syndrome: overview and update, below.

Medial smooth muscle thickness in small pulmonary arteries in sudden infant death syndrome revisited. Krous HF, Floyd CW, Nadeau JM, Silva PD, Blackburn BD, Langston C. From the abstract: "Increased relative medial thickness of smooth muscle in small pulmonary arteries, peripheral extension of smooth muscle into the alveolar wall arteries, and right ventricular hypertrophy, in response to purported prolonged hypoxia, have been reported in sudden infant death syndrome. Compared relative medial thickness of smooth muscle in 88 SIDS infants and 17 controls, using morphometry. Found that the thicknesses did not differ. "[O]ur data suggest that SIDS is an acute event not preceded by recurrent or prolonged apnea and hypoxia."

Cosleeping and sudden unexpected death in infancy. Person TLA, Lavezzi WA, Wolf BC. Arch Path Lab Med 2002 Mar; 126: 343-345. Retrospective review of 58 SUDS autopsies done for upstate New York coroner's offices between 1991 and 2000.

27	cosleeping
14	in adult bed
9	on a couch
7	parental intoxication documented
31	not cosleeping

No anatomic differences were detected at autopsy between the cosleeping infants and the noncosleeping infants. They did not differ demographically.

Concluded: "cosleeping is a dangerous practice." Compression by either the adult or by soft bedding. Information about parental smoking was not available. Does not give demographic risk factors such as LBW etc. Note that their figures could also be used to argue that sleeping alone is a dangerous practice: more babies died sleeping alone. [I'm not clear on how these data show that cosleeping is a dangerous practice; they could be read to show that sleeping is a dangerous practice. --JKR]

"Back to sleep" doesn't cause aspiration. Malloy MH et al., Pediatrics 2002 Apr; 109: 661-665. SIDS mortality fell by almost 40% between 1991 and 1996, from 118 per 100,000 to 72 per 100,000, due to

back-to-sleep, but there was no increase in deaths from aspiration. In the same time, the overall infant mortality fell from 319 to 249, a decrease of 22%. Yet there were some aspiration deaths(!)

Lung tissue concentrations of nicotine in SIDS. McMartin KI, Platt MS, Hackman R, Klein J, Smialek J, Vigorito R, Koren G. J Peds 2002 Feb; 140: 205-209. 44 SIDS and 29 non-SIDS. SIDS had 19 ng/g, nonSIDS had 7 ng/g, $P=.001$.

Respiratory syncytial virus infection and the primary care physician. Park JW, Barnett DW. Southern Medical Journal 2002 Mar; 95(3): 353-359. Almost all children get it. Usually mild. But it is the single most important cause of bronchiolitis and pneumonitis in infancy and contributes to mortality in a subset of high-risk infants. Paramyxovirus RNA virus. Lower income are 5X to 10X more likely to require hospitalization. Pathology: necrosis of the respiratory epithelium of the small airways, peribronchiolar monon infiltr, plugging of lumens, hyperinflation, and atelectasis. Secretions contain RSV-specific IgE, histamine, leukotriene C4. These mediators cause bronchoconstriction, as they do in asthma. In RSV pn, epith necr ext to bronchi and alveoli. Incr work of breathing. Venous admixture. Retention. Early signs are rhinorrhea, pharyngitis. Cough appears 1-3 d later. Fol by audible wheezing, low-grade fever. Progression has air hunger, retractions, cyanosis, hyperexpanded chest. Signs of severe illness are central cyanosis, tachypnea over 70, listlessness, apneic spells, hyperexpanded chest almost silent to ausc. High risk for apnea in prematures, also age under 1 mo, and those with a previous hx of apnea. Has been implicated in SIDS. (citing Williams et al., BMJ 1984, Church et al., Am J Dis Child 1984). Infect in early infancy is a predisposing factor for later chronic lung disease e.g. recurrent wheezing. We know that.

[Vertebral artery constriction in prone sleeping.] Stefan Puig MD, University of Vienna. Presented to the American Roentgen Ray Society, April 29, 2002.

Subtle developmental abnormalities in the inferior olive: an indicator of prenatal brainstem injury in the SIDS. Kinney HC, McHugh T, Miller K, Belliveau RA, Assmann SF (Harvard). J Neurop Exp Neurol 2002 May; 61(5): 427-441. Analyzed by (computer-assisted cell counting on sections stained with H&E/Luxol fast blue) the number and density of neurons and reactive astrocytes in the inferior olive in 29 SIDS cases and 29 controls. There was a statistically significant difference ($p=.002$) in the average number of neurons per cubic mm (they say cubic, I wonder if they mean square?) (7,687 +/- 255 neurons in SIDS versus 8,889 +/- 255 in controls), adjusted for conceptional age. Reactive astrocytes were present in both groups and did not differ. There was no variation depending on sleep surface or found position. "We propose that at least some SIDS victims experience intrauterine brainstem injury including the olivocercular-cerebellar circuitry derived from the rhombic lip. [The rhombic lip gives rise to the external granular layer of the cerebellum, the arcuate nucleus, the inferior olivary nucleus, and ?other ventral medullary surface areas. See the serotonergic work. -JKR] Also need more work on the interactions if any of the ventral medulla and cerebellum in cardioventilatory control. [Note by JKR: they adjusted for conceptional age, but did they adjust for birth age, prenatal and birth history, race, body size, body mass index, diet, and social class? Who were the controls? Were the controls matched for prenatal and birth history, race, body size, body mass index, diet, and social class? Also, I would point out that in infants under 2 months, what they're probably seeing is myelination gliosis, a normal process.]

Why is smoking a risk factor for SIDS? Gordon AE, El Ahmer OR, Chan R, Al Madani OM, Braun JM, Weir DM, Busuttill A, Blackwell CC. (Edinburgh) Child Care Health Dev 2002 Sep; 28 Suppl 1: 23-25. Smoking is a risk factor for both SIDS and respiratory infections. This study investigated blood samples and respiratory epithelial cell aspirates from smokers and nonsmokers for the effect of cigarette smoke on mucosal surface colonization and induction of inflammatory mediators, principally circulating cytokines such as interleukins in smokers. Found that both were increased. The same could be true in infants. Note that these are the same guys who produced the paper on gene polymorphism for the IL-1 gene in SIDS, see below under "SIDS genetics."

Toxicologic analysis in cases of possible SIDS -- a worthwhile exercise? Langlois NEI, Ellis PS, Little D, Hulewicz B. Am J Forens Med Path 2002 Jun; 23(2): 162-166. Yes, it is worthwhile bc it allowed them to exclude SIDS in three out of 117 possible SIDS cases. In these three cases they unexpectedly found methadone -- one fatal intoxic and two exposure. Here were their cases:

SIDS	99
UNDET	4
NAT	10
AHT	3
TOX	

Cardiac pathology in sudden unexpected infant death. Dancea A, Cote A, Rohlicek C, Bernard C, Oligny LL. (Quebec) J Peds 2002 Sep; 141(3): 336-342. Retrospective study of all autopsies of sudden infant deaths between 7 days and 2 years of age occurring in the province over a 13 year period. Had about 820 cases. 32% died awake, 68% died while asleep. Found that 82 cases had cardiac pathology, of which 54% were a malformation and 46% nonstructural pathology. "Cardiac pathologic features are frequent when the child is witnessed to be awake at the time of sudden death." See under "Cardiac."

Unexpected infant death: occult cardiac disease and sudden infant death syndrome: how much of an overlap is there? J Peds 2002 Sep; 141(3): 303-305. Commenting on Dancea et al., above.

Sudden unexpected death in hospitalized children. Buchino JJ, Corey TS, Montgomery V. J Peds 2002 Apr; 140(4): 461-465. See also Wilcox R, Steiner RD, letter, J Peds 2003 Mar; 142(3): 357 on metabolic disease.

The triple-risk hypothesis in SIDS. Guntheroth WG and Spiers PS. Pediatrics 2002 Nov; 110(5): e64. The purpose of these authors was to test Kinney's hypothesis that SIDS is due to in utero hypoxic injury to the brain stem, noting Naeye's 1976 finding of brainstem gliosis in SIDS. Noting also Raring's 1975 finding that SIDS incidence follows a bell curve over age from birth to one year, showing that the etiology must be multifactorial. Kinney proposed that fetal hypoxia brainstem injury was one leg of the triple risk triad. These authors retrospectively studied an unspecified number of infants born to mothers suffering from placenta previa, abruptio placentae, and excessive bleeding during pregnancy, and who therefore "presumably are" at increased risk for hypoxia and brainstem injury during fetal life, and therefore should be at increased risk for death or SIDS according to Kinney's fetal brainstem hypoxic injury hypothesis. They found that these infants had a high nonSIDS mortality but (?) no (?) [not stated] increase in SIDS mortality. Also found that the period of maximum death for these maternal-anemia infants was the neonatal period, not the SIDS period. And that is exactly what would be expected, because other types of fetal-injured and developmentally abnormal infants have a much higher mortality in the neonatal period than they do in the postneonatal period. Opine that "none of the triple risk hypotheses presented so far have significantly improved our understanding of the cause of SIDS. Point out that infants with known or probable hypoxic injury tend to die in the neonatal period and tend to have malformations. Give a historical review of SIDS. In 1952 Kinney et al. argued that "not all SIDS victims are normal," and postulated congenital brain abnormalities. Bergman (1970) proposed the multifactorial hypothesis. Wedgwood (1972) grouped the factors into three groups as the triple-risk hypothesis, consisting of

General vulnerability
Age-specific risks
Precipitating factors

Raring (1975) looked at the bell-shaped curve of incidence and noted that it resembles that of polygenic inheritance and other multifactorial outcomes. ... Naeye (1976) purported to find gliosis in the brainstems of 50% of SIDS victims. Kinney et al. (1983) reported brainstem gliosis in 22%. Thought to be coming from previous (?intrauterine) hypoxic events. Proposed neurotransmitter changes in the muscarinic and serotergic systems. "The major issue is when did the brainstem abnormalities, astrogliosis, or neurotransmitter changes occur and whether either is specific to SIDS." Kinney found hypoplasia of the arcuate nucleus in 5% of SIDS, but the present authors note that such cases "should not be included under the rubric of SIDS, by definition (because they have a primary developmental defect and therefore are not "healthy infants."

Evaluation of diagnostic tools applied in the examination of sudden unexpected deaths in infancy and early childhood. Arnestad M, Vege A, Rognum TO. FSI 2002; 125: 262-268. See Dr Krous's summary in *Child Abuse Quarterly* for July 2002. Retrospective review of 309 SUDS in Norway 0 to 3 years of age.

309 SUDS

- 73 explained deaths
 - 42 NAT (12 pn, 9 "sepsis," 21 other nat)
 - 14 ACC
 - 7 neglect or abuse
 - 10 other (?)
- 236 unexplained deaths
 - 193 classic SIDS
 - 43 borderline SIDS

Compared the usefulness of three information sources:

Sources compared:

- The circumstances
- The history
- The autopsy

Over the whole study population, the overall most useful tool was the autopsy, in particular histol, cults, and neuropathology. But in the cases of accident and abuse, the scene investigation and the gross postmortem findings were the most useful. The history was overall the least useful. Tox and radiology were not very useful. Dr Krous notes that the authors cautioned that with the decreasing SIDS incidence as a proportion of SUDS deaths, the proportion of cases in which the scene investigation will be useful is increasing.

State Supreme Court overturns ruling on day care death. By Noreen Gillespie, Associated Press, Nov 25, 2002, originally appearing in Newsday at <http://www.newsday.com/news/local/wire/ny-bc-ct—scoconn-daycarede1125nov25,0,6651533.story> also http://sids-etwork.org/experts/daycare_liability_overturned.htm *LePage v. Horne*, Conn S Ct. SIDS in day care of a 10 week old girl who was sleeping on her stomach contrary to the instructions of her parents. Held, DV for D bc P failed to call an expert in the day care industry to T to the industry standard of care. The court did not reach the Q of causation.

SNP S1103Y in the cardiac sodium channel gene SCN5A is associated with cardiac arrhythmias and sudden death in a white family. Chen S, Chung MK, Martin D, Rozich R, Tchou PJ, Wang Q. (Cleveland Clinic) J Med Genet 2002 Dec; 39(12): 913-915.

Postmortem screening for fatty acid oxidation disorders by analysis of Guthrie cards with tandem mass spectrometry in sudden unexpected death in infancy. Wilcox RL, Nelson CC, Stenzel P, Steiner RD. J Pediatr 2002 Dec; 141(6): 833-836. Acylcarnitine analysis of cord blood spots. In the first five years of the study, three cases (1.2% of Oregon's 247 cases) of SUDS were found to have fatty acid oxidation disorders by this method -- two with MCAD and one with VLCAD.

AAP Policy Statement: Apnea, SIDS, and Home Monitoring. Committee on the Fetus and Newborn. Pediatrics 2003 Apr; 111(4): 914-917. Multiple studies have been unable to establish the alleged efficacy of home monitoring in decreasing the risk of SIDS. Should limit its use to specific clinical indications for a specified period. Implies that the use of monitors could be affirmatively harmful if it leads parents to think that these devices are protecting their infant against SIDS. Further says that neonatal apnea does not correlate with or increase the risk of SIDS.

The committee gives a discussion of this last item. "The hypothesis that apnea is the pathophysiologic precursor of SIDS was first proposed in 1972 (citing Steinschneider and discussing the sequel to his work.) The apnea theory never has been proven despite extensive independent research in the several decades after that report..." Further, "Peer-reviewed evidence indicates that apnea is not predictive of or a precursor to

SIDS. To the contrary, the evidence indicates that there is no clear, unequivocal relationship between apnea and SIDS.” Discusses the CHIME study (Hoffman et al., Ann NY Acad Sci, 1988), which found that apnea and extreme apnea occur in all infant groups and are recurrent only in preemies. And it resolves before the SIDS age range.

Infant Botulism. MMWR 2003. 52(2): 21-24. Infant botulism results from the germination in the large intestine of swallowed spores of *C. botulinum*. Dx is by detection of botulin toxin in raw stool samples or stool-enrichment cultures. Can present as FTT. DDX includes sepsis, dehydration, Werdnig-Hoffman, Guillain-Barre, MG, drug intoxication, metabolic disease, meningitis. But I notice that they do not mention sudden death. They report four nonfatal cases from Staten Island: all recovered, some after needing ventilatory support:

Case 1 a breast-fed 7 week old presented with fever 105 constipation, listlessness, and weak head control.

Case 2 a formula-fed 10 week old w constipation, difficulty sucking and swallowing, requiring mechanical ventilation.

Case 3 a breast-fed 18 week old with somnolence and difficulty swallowing, altered cry, loss of facial expression, respiratory muscle weakness, needing mechanical ventilation.

Case 4 a 3 week old had constipation, lethargy, decreased appetite, sluggish pupillary reflexes, difficulty swallowing, altered cry, weak sucking, peripheral weakness.

Effect of position on sleep, heart rate variability, and QT interval in preterm infants at 1 and 3 months corrected age. Ariagno RL et al. (Stanford) Pediatrics 2003 Mar; 111(3): 622-625. Prone sleeping caused alterations in heart rate variability and increased the QT interval in preemies, but only at one month corrected age. The authors interpret this to mean that prone sleeping increased the vulnerability for SIDS at this age in this subgroup.

Vagal overactivity: a risk factor of SIDS? Shojaei-Brousseau T et al. Arch Dis Child 2003 Jan; 88(1): 88. A hypothesis.

QT dispersion in infants with ALTE syndrome. Goldhammer EI et al. (Haifa). Studied 89 ALTE infants and 18 controls. Found that QTc was significantly greater in the ALTE group. These were infants avg 2 ½ months old who were found limp.

A comparison of respiratory symptoms and inflammation in sudden infant death syndrome and in accidental or inflicted infant death. Krous HF, Nadeau JM, Silva PD, Blackbourne BD. Am J Forens Med P 2003 Jan; 24(1): 1-8. Chronic inflammatory infiltrates are commonly found in the autopsy histology of the upper and lower respiratory tract of SIDS victims. But no one knows for sure what pathologic significance to attach to these, because the literature contains very few control cases from infants dying suddenly without any natural disease. Dr. Krous and his colleagues have attempted to remedy this deficiency by doing histology on the upper and lower respiratory tracts of 33 infants who died by accident or infanticide (mostly mechanical asphyxia, suffocation, or drowning), and comparing the sections to those of 155 SIDS cases. They blindly compared sections from the trachea, bronchi, bronchioles, and peripheral lung, and scored them 1 to 4 for lymphocytic infiltration, eosinophilic infiltration, neutrophilic infiltration, chronic bronchitis, bronchiolitis, interstitial pneumonitis, and bronchopneumonia. They also searched the histories for any symptoms of URI. The findings for moderate (grade 2 - 3) infiltrates were:

	<u>SIDS</u>	<u>Controls</u>
Proximal trachea	35%	71%
Distal trachea	27%	70%
Bronchi	27%	70%
Bronchioles	0%	3%

Interstitial pneumonitis	17%	15%
Bronchopneumonia	6%	6%

The authors found that 85% of both the SIDS and the control groups had mild interstitial pneumonitis, and the frequency of moderate interstitial pneumonitis in the two groups was **almost the same** (17% and 15%). Bronchopneumonia was found in a very few cases from both groups.

The authors did autopsy cultures on 138 of their SIDS cases and found that 78% of these grew out contaminants and the others were nondiagnostic -- a finding that will surprise no one.

The authors consider that their study, the first ever done with adequate controls, rules out the possibility that SIDS is caused by respiratory infection “in the sense of organ parenchymal destruction and dysfunction.” (The hypothesis of interleukins or a possible “hyperimmune response” to minor infection being left open, though the implications against it are plain.)

Reviewer's comment [by JKR]: This study for the first time provides a statistical basis for what forensic pathologists have known anecdotally for years -- that moderate chronic inflammation of the respiratory tract is not pathologically significant with respect to the cause of death in SIDS cases. I should like, however, to note that the authors' controls, while adequate for the design of this study, were not normal controls. They were children who died unnatural deaths in which an element of neglect, and in some cases violence, was likely. In view of this, the fact that their respiratory tracts were pathologically identical to those of a percentage of SIDS cases raises more questions than it answers. Chronic respiratory inflammation is one of the findings typical of what is known as “SIDS with findings,” or “Class II SIDS.” These are infants in whom the lack of an anatomic cause of death is complemented by the presence of poor growth percentiles, thin body habitus, reduced fat stores, thymic and adrenal atrophy, atypical circumstances of death, unsafe sleep surfaces, and crowded living arrangements in lower- or working-class apartments. One wonders whether what this study is really saying (apart from the fact that minor respiratory inflammation is not fatal) is that a subpopulation of high-risk homes exists in which small children are less likely to survive.

[Symposium on SIDS] Arch Ped Adol Med 2003 Mar; 157(3): 286-290 et seq. Critical reference with comments by others.

SIDS: Overview and update. Byard RW, Krous HF. Pediatric and Developmental Pathology 2003 Mar-Apr; 6(2): 112-127. Review article, actually an update of their book. “Despite calls to abandon the designation [probably referring to Spiers & Guentheroth, 2002, above. –JKR], SIDS **remains a viable term** for infants who die in their sleep with no evidence of accident, inflicted injury, or organic disease after a full investigation has been conducted according to standard guidelines.” [But having said that it remains a viable term, they are compelled to refine it so that it will not be an obfuscating term. See Beckwith et al., 2003, 2004, below.]

Defining the sudden infant death syndrome. J Bruce Beckwith MD. Arch Peds Adol Med 2003 Apr; 157(3): 286-290. With invited critiques. Term was first proposed in 1969. “This term played an important role by focusing attention on a major category of postneonatal infant death, providing support to grieving families, and diminishing the guilt and blame characteristic of these deaths.” It remains a diagnosis of exclusion, rather than inclusion of characteristic features such as death during sleep or characteristic age distribution. This has led to overinclusion and underinclusion, with resulting controversy. He suggests the 2-tiered approach, “with a more general definition intended primarily for case management and death administration, and a more restrictive one intended primarily for research purposes, which distinguishes those deaths closely fitting the classic SIDS profile from those with one or more less typical features.” He begins by rehearsing the famous 1972 Steinschneider fiasco of 5 infant deaths up to 28 months with a history of apnea, leading to a fad for home apnea monitoring. This was a result of overinclusion. Overinclusion also has resulted in missing lethal genetic disorders and infanticide. But when there is underinclusion, “the family is often denied the benefits of the SIDS grief support system. Unwillingness to diagnose SIDS can also lead to inappropriate suspicion or blame directed toward parents or caregivers, including unfounded self-accusation.” Back-to-sleep resulted in SIDS reduction “primarily

among those infants who composed the most typical SIDS cases in the past. (citing Hauck, "Changing epidemiology," in Byard & Krous, 2001.) As a result, the age distribution of the remaining cases has broadened out. He reviews the history of the definition. 1969 conf gave it a name "to focus attention and research activity," and to create 'a certifiable cause of death.' There was agreement that the definition should be kept as broad as possible, so as to promote research and avoid mistakenly excluding cases that would later turn out to be the same thing. Thus the 1969 definition was:

"The sudden death of any infant or young child, which is unexpected by history, and in which a thorough post-mortem examination fails to demonstrate an adequate cause for death."

He comments that, "If a prize were offered for the poorest definition of a disease or disorder in the scientific literature, this one would be a strong contender! It contains no limiting criteria, lists none of the features common to most cases, and suggests that this syndrome is only one of exclusion."... It doesn't even include "during sleep." Yet, despite the vagueness of the def, it yielded consistent epid profiles, both as to circumstances of death and the autopsy findings. But, the vagueness of the def led to the inclusion of many inappropriate cases, and led some to dismiss the term as worse than useless. In the 1989 revision stim by NICHD, only 4 of the 12 invited panelists were pathologists. My purposes ... to incorporate the most consistent features of the syndrome and to distinguish between typical and nontypical cases to enhance the quality of research. But this was not adopted. The resulting 1989 definition modified the old one only by the inclusion of an age criterion. Says that this failure was largely the result of nonpathologists and people who had little experience with the syndrome.

"The sudden death of an infant under one year of age, which remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene, and review of the clinical history."

Subsequent Sydney conf 1992 again panel proposed stratifying. Rejected by session. B proposed the terms "Category I" and "Category II," because session had objected that "atypical" cd be offensive to families. Also Category III for no-autopsy cases. Gives this proposed definition in a separate box:

Generic characteristics

The sudden and unexpected death of an infant younger than 1 year and usually beyond the immediate perinatal period, which remains unexplained after a thorough case investigation, including performance of a complete autopsy and review of the circumstances of death and of the clinical history. Onset of the lethal episode was presumably during sleep. Minor inflammatory infiltrates or other abnormalities insufficient to explain the death are acceptable.

Category I

An infant death that meets the generic criteria and also meets all of the following standards:

- age between 3 weeks and 8 months
- no similar deaths in siblings, close genetic relatives, or other infants in the custody of the same caregiver
- no ev of significant trauma, abuse, neglect, or accident
- no ev of unexplained moderate or severe stress in the thymus, adrenals, or other organs
- intrathoracic petechiae are a supportive but not required finding

Category II

An infant death that meets the criteria for Category I SIDS except for one or more of the fol:

- age outside the Category I range
- similar deaths in close genetic relatives where infanticide is ruled out (genetic consultation indicated)

inflammatory changes or other abn somewhat greater than Category I

Cases in which accidental asphyxia is considered possible. (Can be either Category I or Category II at the discretion of the pathologist.) “A dx of suffocation or asphyxia ... should be made only with strong supporting evidence. Sometimes infants may, during a death struggle, get into situations that falsely suggest mechanical asphyxia.”

Category III

If no autopsy

Suggests that formulating the next revision be restricted to pathologists. [Alas, that did not happen. –JKR]

Comments by the invited critics:

Joel Haas: I agree with Beckwith.

Henry Krous: Agrees. Suggests that the inclusion criteria could be made even more specific by requiring negative screens for metabolic disease and tox. Suggests that there could be a constellation of findings that constitutes Category I SIDS. If there is, why not say so? Comments that “[W]idespread implementation of infant care practices that have reduced the number of SIDS deaths has led to cases coming disproportionately from lower socioeconomic groups with chaotic backgrounds; such cases are often more difficult to diagnose.” He adverts to the SIDS vs. UNDET debate among forensic pathologists. “There are advantages and disadvantages to each side of the debate. Local customs and resources, as well as personal philosophy, often drive the opinions of individual pathologists. The identification of specific markers of SIDS would go along way to resolving this issue.”

David Becroft: Due to definitional confusion internationally, the term *unascertained* is booming, accounting for very low rates of SIDS in some countries... Says that the naïve or spotty performance and interpretation of death scene investigation has led to a large amount of anecdote and suspicion. Agrees that pediatric pathologists should “take the lead,” but does not say that other specialties should be excluded.

Cutz: Implicitly rejects Beckwith’s proposal. Apparently wants to keep the unitary definition with the understanding that there is heterogeneity of cases within it. The phenomenon of unexpected death in infancy is multifactorial. The triple-risk hypothesis captures this heterogeneity better than a two-tiered definition would. The scarcity of suitable controls frustrates research. Suggests that molecular biology holds the way forward to understanding many of the deaths.

Rognum: praises B’s idea but seems to stand pat on a unitary definition, in this case the 1995 Stavanger definition. [But he later did get on board with the San Diego revision. –JKR]

Jem Berry: Identifies the two purposes of a definition: to support families and to support research. Thus under B’s proposal, all families would be supported, but research could be appropriately stratified. But the problem of definitional imprecision remains, because the proposal does not [directly] address the problem of accidents, inappropriate care, or even abuse. “In about 5% to 10% of cases otherwise classifiable as SIDS, confidential multidisciplinary review reclassifies the deaths as due to the actions of a carer.” He then gets down to specific case characteristics that case diagnostic problem not addressed by the proposal:

- high postmortem body temperature
- a single pathogen in the blood with no historical or anatomical correlate
- moderate dehydration
- increased acute phase reactants
- old fractures
- drugs
- alveolar siderophages
- history of ALTE

Infant sleeping position and the risk of sudden infant death syndrome in California, 1997-2000. Li DK, Pettiti DB, Willinger M, McMahon R, Odouli R, Vu H, Hoffman HJ. Am J Epidemiol 2003; 157: 446-455. (Kaiser Oakland) An interview study in 11 counties with interviews of the mothers of 185 SIDS cases and 312 age- and race-matched controls. The controls consisted of living infants. Found:

thing	odds ratio
put prone	2.6
put on side	2.0
put on side and found prone	8.7
put prone after accustomed to sleeping on back	8.2
put side	6.9

Cited by AAP 2005 Policy Statement for the proposition that side sleep and prone sleep confer the same risk. Therefore, the AAP concludes that side sleep is no good.

Infant sleep position: associated maternal and infant factors. Chung EK, Hung Y-Y, Marchi K, Chavez GF, Braveman P. Ambulatory Pediatrics 2003 Sep-Oct; 3(5): 234-239. Which kinds of mothers DO NOT back-to-sleep? Answer: education less than college, income below poverty level, multiparous, black and Asian-Pacific Islander, non-English-speaking. Here were the adjusted odds ratios for back-to-sleep:

uneducated	.59
poor	.65
multiparous	.80
black	.49
A/PI	.65
non-English	.69
infant over 7 months	.70

[Bedsharing with siblings, soft bedding, increase SIDS risk.] CDC News Release May 5, 2003 citing an analysis of African-American SIDS deaths in Chicago, the Chicago Infant Mortality Study, http://www.nichd.nih.gov/new/releases/infant_sids.cfm directed by Fern R. Hauck, MD, pub in *Pediatrics* for May 2003. 260 SIDS cases from Chicago from Nov 1993 to Apr 1996. "Our study found a dramatic increase in SIDS risk for prone sleeping on soft surfaces... Additionally, infants should never be placed to sleep on a couch with anyone or in a bed with other children," says Dr Hauck. They used same-race controls with the same birth weight. Sofa sleeping appears to be "highly dangerous;" had 15 SIDS on sofas.

thing	relative risk
cosleeping with siblings	5.4
soft bedding	5.0
stomach	2.4
stomach AND soft bedding	21.0

Distinct cytokine profile in SIDS brain: a common denominator in a multifactorial syndrome? Kadhim H, Kahn A, Sebire G. Neurology 2003 Nov; 61: 1256-1259. Detected high interleukin 1-beta and TNF-alpha immunostaining of neurons (graded subjectively on a four-step scale from - to +++) in the arcuate nucleus and dorsal vagal nucleus of 17 so-called "SIDS" and 8 nonSIDS autopsies. The SIDS cases ranged in age from 3 to 8 months, and almost all had coryza, fever, or reflux histories. The nonSIDS were a congenital heart disease, a diaphragmatic hernia, a congenital toxoplasmosis, a cirrhosis, two congenital AIDS, a meningitis, and a mononucleosis. Found that both groups had staining in various parts of the brain. But there was a pattern of overexpression of IL-1b in the arcuate nucleus in all 17 SIDS brains and only 1 nonSIDS brain. And TNF in all 17 SIDS but only 2 nonSIDS. The degree of increased IL-1b

staining in the arcuate nucleus was +++ in SIDS versus+ or – in nonSIDS. The degree of increased TNF staining was ++ versus +/- in nonSIDS. (Much more detail about the levels is given in the article). They also stained other parts of the brain, including the neocortex, and found that the increase was region-specific to these particular nuclei. The authors admit that they do not know how these cytokines got there or what these cytokines would do to the brain functioning, if anything. They say that IL-1b is involved in turnover of neurotransmitters and therefore could affect neuron excitability. Involvement of the ascending reticular activating system could affect rousability. They note Hannah Kinney's work (1995) finding decreased acetylcholine receptor binding in the arcuate nucleus, and abnormal polysomnograms in future SIDS infants found by Franco et al., 1999. Also, Froen et al, 2000 found that intrathecal instillation of IL-1 produced respiratory depression in piglets. [My criticism: The nonSIDS controls are not controls. They are severely ill infants with fatal infections involving the brain. These types of brains are severely damaged even before death and deteriorate rapidly after death. Comparing SIDS cases to brain damage only tells us that brain damaged brains are brain damaged. Need normal controls from instant-death situations. As academics, these doctors did not have access to such material. –JKR] See also the same authors' 2005 paper on larger brains in SIDS cases than in hospital cases.

The brainstem and vulnerability to sudden infant death syndrome. (editorial) Thach BT. *Neurology* 2003 Nov; 61: 1170-1171. Commenting on the work of Kadhimi et al. (above), finding increased staining for interleukin 1-beta in the arcuate nucleus and dorsal vagal nucleus of SIDS compared to controls. Says that that article (like all SIDS articles) has not age-matched controls and may be subject to autolysis; also that the scoring of staining was subjective and not quantitative. (See also my criticisms, above. –JKR) But if replicated by others, the presence of increased inflammatory mediators in the brainstem would correlate with the history of minor infection often present in SIDS versus control infants. Invites speculation on two issues: (1) Where are these cytokines coming from? (2) What does their presence mean about the mechanism of death? As to question (2), the laryngeal reflex is invoked, citing Vege et al. on CSF IL-6 levels, 1999, and Stoltenberg et al., 1994 on intrathecal IL-1 injection causing central apnea in piglets. The laryngeal reflex is a normal reflex of glottic closure that protects the airway against liquids during swallowing. As to question (1), the cytokines could be either produced locally by neurons (neurons can produce them), or brought in from outside either by the bloodstream or by retrograde axonal transport, maybe from the larynx, by way of the cranial nerves. If the cytokines are coming in by retrograde axonal transport, that would argue that a throat inflammation (such as RSV) causes death by indirectly affecting the brain stem. If the cytokines are produced locally, that would imply abnormal functioning of brainstem neurons for an unknown reason, and we're back to the triple-risk model. (In other words, have hit another dead end. –JKR).

Factors relating to the infant's last sleep environment in sudden infant death syndrome in the Republic of Ireland. McGarvey C, McDonnell M, Chong A, O'Regan M, Matthews T. *Arch Dis Child* 2003; 88: 1058-1064. Showed that cosleeping was a risk factor for SIDS, along with prone. See below under "Cosleeping."

Out of the blue, a lightning bolt to the heart. By Sandeep Jauhar. *The New York Times*, February 10, 2004. Describes Brugada's Syndrome, and SCN5a defect that causes sudden cardiac death mostly in adults, and is diagnosed by a "shark fin-like" deformity on the ECG. Said to be responsible for as many as 20% of the sudden cardiac deaths in persons with structurally normal hearts.

Study of the brainstem, particularly the arcuate nucleus, in SIDS and SIUD. Maturri L, Ottaviani G, Alfonsi G, Crippa M, Rossi L, Lavezzi AM. *Am J Forens Med P* 2004 Mar; 25(1): 44-48. Examined brainstems from 106 SIDS, 30 control infants, and 51 stillborns. Found "a remarkable variability" of the arcuate nucleus in both size and neuronal density. Does not describe any gliosis or anything remotely approaching gliosis (*q.v.*). The description of this nucleus will vary depending on what level it is sectioned at. See below under "Autopsy technique" for details. They cut over 200 slides on each brainstem and didn't find anything.

Knight's Forensic Pathology, 3d ed., 2004. Saukko P and Knight B. New York: Oxford University Press, 2004. Chapter 21, Sudden Death in Infancy.

Sudden infant death syndrome and unclassified sudden infant deaths: a definitional and diagnostic approach. Krous HF, Beckwith JB, Byard RW, Rognum TO, Bajanowski T, Corey T, Cutz E, Hanzlick R, Keens T, Mitchell EA. Pediatrics 2004 Jul; 114(1): 234-238. The long-awaited redefinition of SIDS with introduction of a new category, defined as “unclassified sudden infant deaths.” Uses inclusion criteria rather than diagnosis of exclusion.

Category I SIDS: Standard definition plus normal everything -- history, scene, autopsy. Age has to be 21 days to 8 months.

Category I-B SIDS: Standard definition plus presumptively normal everything, but not everything documented.

Category II SIDS: = Category I SIDS except that one or more of the following bad things is present:

- Age range outside Category I limits
- Similar deaths among close relatives or in the same care
- NICU history
- Overlaying or mechanical asphyxia not excluded
- Abnormal growth & development
- Marked inflammatory changes (but not enough to be COD)

Unclassified sudden infant deaths: deaths that fail to meet the criteria for Category I or II above, but where no cause has been definitely established.

Near-SIDS. (The authors do not use this term, but this is what they are referring to. Placed in a separate category because the normal diagnostic methods for SIDS cannot be used.)

Pathology research into sudden infant death syndrome: where do we go from here? Krous HF, Byard RW, Rognum TO. Pediatrics 2004 Aug; 114(2): 492-494. Argues for a new statute giving blanket authorization for retention of specimens and doing research on them. Because obtaining consent for research on a case-by-case basis is unworkable. Alder Hey and similar outcries about specimen retention have dramatically reduced the possibility of doing research on SIDS material. [Later they successfully do brainstem 5-HT research on 31 SIDS cases and 10 controls by going through the Medical Examiner's office, which under California law does not need individual consent to do research. Paterson et al., JAMA 2006, below.]

Evaluation of the Reid index in infants and cases of SIDS. Karger B, Fracasso T, Brinkmann B, Bajanowski T. (Ger) Int J Legal Med 2004 Aug; 118(4): 221-223. The Reid index was not different in SIDS from controls in a group of 124 autopsies grouped into typical SIDS, SIDS with inflammation, and non-natural deaths.

Terminology used by pathologists in reporting on sudden infant deaths. J Clin P 2004 Mar; 57(3): 309-311. (UK) Surveyed 63 pathologists. Found marked variability in their use of the terms “SIDS” and “Unascertained.” Bedsharing was categorized different ways. Pathologists commented on the inadequacy of the history available to them. Conclusions: (1) There is a serious need for consistency. (2) Pathologists should be provided with a full history. [In other words, some of the inconsistency in diagnosis may have been due to inadequate history. JKR]

SIDS -- a syndrome in search of a cause. N E J Med 2004 Sep; 351(10): 978-986.

The sudden infant death syndrome gene: does it exist? Opdal SH, Rognum TO. Pediatrics 2004; 114(4): e506-e512. See below under “SIDS genetics.” A review article concerning the various genetic syndromes. Finds that “It is likely” that there are SIDS genes operating as a predisposing factor [not as a cause]...” Harks back to the trivalent hypothesis. Says that lethal mutations would account for only about 2% of SIDS.

Sudden Infant Death Syndrome risk factors with regards to sleep position, sleep surface, and co-sleeping. Alexander RT, Radisch D. JFS 2005 Jan; 50(1): 147-151. See below under “cosleeping.”

Incongruent cerebral growth in sudden infant death syndrome. Kadhim H, Sebire G, Khalifa M, et al., J Child Neurol 2005; 20: 244-246. [See also the same authors' 2003 article on cytokine profiles, above].

These authors measured the brain weights of 97 SIDS infants (age range 1 to 10.5 months; median age 3.25 months) and 23 infants who died of other unspecified conditions (age range 1 to 7 months; median age 3 months). They found that the SIDS brains were heavier than the control brains. All of the SIDS brains were above the 50th percentile (using ranges derived by Shankle et al. from the data of Coppoletta & Wolbach), and 57% of them were above the 99th percentile. As for the control brains, 74% of them were above the 50th percentile but only one (4%) was above the 99th percentile. The authors compared the brain weight findings with head size and with the weights of the other body organs. They found that the SIDS infants did not have larger heads than the control infants, but they did have larger organs, and their organs were above 50th percentiles for weight, while apparently those of the controls were not. The brain weights in both groups correlated closely with body weight. (Weight norms were appropriately controlled for prematurity.) All brains had complete neuropathologic examinations, and had no neuropathologic findings that would explain their differential weights.

The authors concluded that their data suggest disproportionate brain growth in a subset of SIDS victims. They do not pretend to know why this occurs, or how it might contribute to the occurrence of SIDS, although they offer a speculation relating to a different cerebral cytokine profile in SIDS (upregulated interleukin 1-beta) causing increased nerve growth factor.

There are numerous problems with this article which render it useless.

1. The authors do not state what their control infants died of. Coming from a university hospital, one might assume that many the control infants may have died of disease. If so, that by itself could explain their having smaller brains than SIDS infants, who are by definition healthy. I note that the SIDS infants also had above-average body organ weights, while the control infants did not. This supports the suggestion that the controls may have been chronically ill. If so, the authors' data simply show that healthy infants have larger organs than sick infants -- a finding that will surprise no one. (The authors implicitly admit this defect in their discussion, pointing out that truly "normal" organ weights are not available, and that SIDS organs may in fact be the only "normal" organs that are available.)
2. It is not surprising that nearly all the brains of both the SIDS group and the control group were heavier than the Coppoletta & Wolbach data from 1933. As pediatric pathologists know, Coppoletta & Wolbach's data were drawn from a population of foundlings and chronically ill inpatients in Boston City Hospital. That population was in no way comparable, genetically, nutritionally, or clinically, to a modern infant population in the advanced countries. The Coppoletta & Wolbach organ weights are grossly small by comparison with what is found in infants today. Little wonder that the authors found that both their SIDS brains and their control brains were above the C&W norms.
3. Although the authors performed a chi-square correlation of the brain weights with the organ weights and the body weights within each group, surprisingly they did not perform such a correlation between the two groups. So we don't know what was the statistical significance of the difference in brain weights which the authors observed. From looking at the scatterplots of their data which the authors have kindly provided, it appears that there is a great deal of overlap between the two groups.
4. Even if the authors' data are taken to demonstrate a statistically significant difference in brain weight between SIDS infants and other equally healthy infants, the meaning of such a finding is unclear. The authors note that their finding is contradicted by another study which found no difference in brain weights between SIDS and controls. Given that the SIDS brains are more likely to be normal in weight than hospital controls, it makes no sense to conclude that the heavier weight of the SIDS brains means that there is something abnormal about SIDS brains. The authors themselves demonstrated, by means of complete and detailed neuropathologic study of all the brains, that there was absolutely nothing abnormal about them. The article can be taken as establishing that SIDS babies have normal brains. We know this.

Probability of coincident vaccination in the 24 or 48 hours preceding sudden infant death syndrome in Australia. Brotherton JL, Hull BP, Hayen A, Gidding HF, Burgess MA. Pediatrics 2005; 115: 643-646. From Dr Reece's review in the Autumn 2005 Quarterly. He notes that SIDS peaks during the same months of life that infants are receiving their vaccinations. These authors did a statistical study on their SIDS cases

and found that the probability of a SIDS infant's having been vaccinated in the past 24 hours was 1.3%, and in the past 48 hours was 2.6%. The authors state that "Vaccination does not cause SIDS."

Long QT syndrome and other channelopathies -- testing for. FAMILION test. Genaissance Pharmaceuticals, Five Science Park, New Haven, CT 06511. 203-773-1450. <http://www.genaissance.com> For more information about the Familion test see <http://www.familion.com>. Or call the sales rep at 866-FAMILION. The Familion test looks for mutations in any of five cardiac ion channel genes. According to Jill Hennessey, M.S., Familion product manager, up to 30% of cases of SCD in children and adults with negative autopsy results may be caused by long QT syndrome or a related disorder. The test can use either postmortem blood or tissue. The cost is \$5,400.

Tissue AND BLOOD samples should be **frozen** at autopsy. They say freeze the blood immediately, because the cells and the DNA lyse. Need either of these samples:

Two purple-top tubes of blood, frozen immediately

or

25-50 milligrams of tissue, preferably heart, frozen

As of 9/21/05 they are working on a method to do it on formalin-fixed heart tissue.

See also the Death Genomics Laboratory at the Mayo Clinic, Michael J. Ackerman, MD, 507-284-0101, ackerman.michael@mayo.edu

Techniques and criteria in pathologic and forensic-medical diagnostics in sudden unexpected infant and perinatal death. Maturri L, Ottaviana G Lavezzi AM. Am J Clin P 2005; 124: 1-10. These Italian pathologists did histology on the cardiac conducting systems and autonomic nervous systems of 100 SIDS infants, 52 stillborns (sudden intrauterine death), and 8 newborns, all of whom died suddenly and unexpectedly. They also did 25 controls who died of hypertrophic cardiomyopathy, pneumonia, sepsis, biliary atresia, and other natural diseases. As to the CNS, they sectioned the spinal cord, cerebellum, stellate ganglion and superior cervical ganglion, the carotid bifurcation, and they sectioned the entire brain stem, dividing it into four levels. (This led to an average of 360 brainstem sections per baby; they did special stains also.) As to the conducting system, they made two blocks: block 1 was the sinoatrial node and its approaches and ganglion; block 2 was the AV node with its atrial approaches. Plus lungs, plus the aorticopulmonary ganglia. They refer to their earlier work showing hypoplasia of the arcuate nucleus in stillborns and SIDS; they give photomicrographs of hypoplastic arcuate nuclei (bilateral or unilateral), and refer to agenesis of this nucleus seen in cases, and describe an association with pulmonary hypoplasia, and hypoplasia of the reticular respiratory formation and the parabrachial Koelliker-Fuse complex (which is in the brachium of the rostral pons. Its function is to prevent the fetus from breathing during labor & delivery and then start it breathing upon delivery.) Four of their SIDS cases had viral encephalitis involving or close to the ventrolateral reticular formation. The peripheral autonomic nervous system showed neuronal immaturity of the cervical sympathetic ganglia in 9 cases, hyperplasia of the aorticopulmonary paraganglia in 24 cases (SIDS cases). As to the conducting system, they observed "areas of resorptive degeneration" in 97% of SIDS cases and 76% of control cases. This involved growth of young fibroblasts with collagen deposition. Dispersion or septation of the AV node and/or the bundle of His was seen in 41% of SIDS and 16% of controls, not statistically significant. They give an interesting discussion of the possible dysfunction of the Koelliker-Fuse complex. They give a good brief review of the various neurological theories of SIDS.

Subtle autonomic and respiratory dysfunction in sudden infant death syndrome associated with serotonergic brainstem abnormalities: a case report. Kinney HC, Myers MM, Belliveau RA, et al. J Neurop Exp Neurol 2005 Aug; 64(8): 689-694. From the abstract: The medullary 5-HT system is composed of 5-HT neurons in the raphe, extra-raphé, and arcuate nuclei at the ventral surface. This system is thought to modulate respiratory and autonomic function, and thus abnormality within it could potentially lead to imbalances in sympathetic and parasymp tone. We report the case of a full-term American Indian boy who died of SIDS at 2 postnatal weeks, and who had subtle respiratory and autonomic dysfunction measured prospectively on the

second postnatal day. Heart rate variability sugg [elev parasymp tone] in active sleep and lower in quiet sleep. At autopsy, arcuate nucleus hypoplasia and 5-HT receptor-binding abn in the arcuate nucleus and other components of the medullary 5-HT system were found. So monitoring of these physiologic tests cd be shown to be useful in prevention if reproduced in a large pop.

The mother G3 was an alcoholic since age 14 and drank during the pregnancy. Also smoked. Maternal drinking and smoking during pregnancy are major risk factors for SIDS. Baby was born at 38 weeks w bw 3402 gm, Apgars 8/9 w thin mec. Three days in the nbn. No medical problems. Had physiologic studies at 2 d of life. This consisted of cardiorespiratory monitoring during both active and quiet sleep as part of a research program for high-risk newborns. Autopsy showed typical SIDS findings. Rel heavy brain wt 430 gm (exp 382) cw agonal cong. Moderate gliosis of the cerebral wm and inferior olive, a focal microscar of the cerebellar cortex, thot to reflect perin hypoxia.

Most infants in that age have a higher heart rate in active sleep than in quiet sleep, but this was reversed in the present infant.

Gives autoradiographs of this medulla compared to two control medullae, showing a complete lack of uptake of tritiated LSD in the normal location of the inferior arcuate n of this infant, indicating absence of the nucleus, which was confirmed histologically; the infant had severe hypoplasia (almost complete absence) of the arcuate nucleus.

AAP revises SIDS prevention recommendations. News release from the AAP dated Oct 10th, 2005.

1. The AAP no longer recognizes side sleeping as acceptable.
2. Bedsharing is not recommended during sleep.
3. Pacifiers reduce the risk of SIDS.
4. The following have been consistently identified as rsk factors for SIDS:

- prone sleep
- soft sleep surface
- maternal smoking during pregnancy
- overheating
- late or no prenatal care
- young maternal age
- preterm or low birth weight
- male sex
- blacks -- 2x-3x
- Indians -- 2x-3x

AAP Policy Statement: The changing concept of sudden infant death syndrome: diagnostic coding shifts, controversies regarding the sleeping environment, and new variables to consider in reducing risk. Task force on SIDS, 2005-2006: John Kattwinkel MD, Fern R Hauck MD, Maurice E Keenan MD, Michael Malloy MD, Rachel Y Moon MD, Consultant Marian Willinger PhD. Pediatrics 2005 Nov; 116(5): 1245-1255. From the abstract: There has been a major decrease in the incidence of SIDS since the AAP released its recommendation in 1992... Some of the recent decrease may be a result of coding shifts to other causes of unexpected infant deaths. Since the last st in 2000, several issues have become relevant, including the significant risk of side sleeping. The AAP also stresses the need to avoid redundant soft bedding and soft objects in the infant's sleeping env, the hazards of adults sleeping with an infant in the same bed, the SIDS risk reduction associated with having infants sleep in the same room as adults and with using pacifiers at the time of sleep, the importance of educating secondary caregivers and neon practitioners on the importance of "back to sleep," and strategies to reduce the incidence of positionnal plagiocephaly associ w supine...

1. Epidemiology. They note that while the SIDS incidence has gone down, the incidence of other causes of unexpected infant death has gone up. Also, the total infant mortality has not changed. "These observations increase the likelihood that some deaths previously classified as SIDS are now being classified in other categories and the true SIDS rate since 1999 may be static." The "other categories" referred to in ICD-9 or 10 are "unknown or unspecified causes" ICD-10 799.9/R99, "suffocation in bed" ICD9 E913[0] and "suffocation -- other" ICD9 E913[1] or ICD10 W76-7 and W81-4. Regular SIDS has the ICD-10 code 7980/R95.

a. Epidemiology of prone positioning. The rate of prone positioning among blacks was 21% versus 11% among whites; the rate of SIDS among blacks was 2.5X that of whites.

b. There has been a decrease in seasonality.

2. Sleep position. A California study done since back-to-sleep showed that side sleep and prone sleep conferred the same risk. (Li, Pettitti et al., 2003). Also, "The California study extended two previous observations that infants unaccustomed to the prone position and placed prone for sleep were at greater risk than those usually placed prone."

3. Bedding. The soft pillows and bedding business goes back to a study by Abramson (1944), but was confirmed by a case-control study in the US (Hauck et al, 2003).

4. Cosleeping. It can be hazardous under certain conditions. (Citing Scheers et al., 2003; Unger et al., 2003; Kemp et al., 2000; Drago & Dannenberg, 1999) Plus others. Some of these studies have found the correlation between death and bed sharing to reach statistical significance only among mothers who smoked. However the European study found that bed sharing with mothers who did not smoke was a significant risk factor up to 8 weeks of age. Scotland found greatest risk under 11 weeks. The risk of SIDS seemed to be particularly high when there are multiple bed sharers, and may also be increased when the bed sharer has consumed alcohol or is overtired. It is extremely hazardous on a couch.

5. Pacifiers. A protective effect. Several studies.

6. Day care. 20% of SIDS deaths occur in secondary care. Many of these were associated with unaccustomed prone sleeping. This is "particularly concerning."

7. Home monitoring. Not be used for SIDS prevention, but may be indicated for some infants who have had an ALTE.

8. Immunization. Studies refute the association. (citing Griffin et al., 1988; Hoffman et al., 1987; Jonville-Bera et al., 2001; MacIntyre et al., 2003) "Still, of 100 deaths reported to the federally administered VAERS from 1997 to 1998, approximately half were attributed to SIDS."

9. Breastfeeding. Breastfed infants are more rousable during sleep than bottle-fed ones. But epid studies have not clearly shown any protective effect re SIDS. However, there is a protective effect re all causes of postneonatal mortality.

10. Positional plagiocephaly. Also called the syndrome of plagiocephaly without synostosis (PWS) Supposedly this has dramatically increased. Congenital plagiocephaly is caused by intrapartum molding. Acquired plagiocephaly is caused by supine sleeping. No tummy time.

11. NICU veterans re sleep position. Often get in the habit of placing prone.

12. Recurrent SIDS and infanticide. Mentions the papers of Southall et al. (covert video, 1997), Meadow, 1999; and Meadow, 2002. "However, on the basis of an in-depth review of recurrent SIDS among families that had experienced one SIDS death, Carpenter et al. (Lancet, 2005) calculated an 87% probability that a second SIDS death within a family would be of natural cause." Calculations of the number of SIDS that might be covert homicide range from 6% to 10%, with the recurrence risk at 2% to 6%. (citing Oyen et al., 1996; Levene et al., 2004). "Therefore, the task force supports the position that the vast majority of either initial or second SIDS within a family seem to be natural rather than attributable to abuse, neglect, or homicide. However, the task force maintains that a complete autopsy etc. are necessary..." [This paragraph was largely stimulated by the 2005 Lancet article on repeat SIDS by Carpenter et al. out of the UK. But, unbeknownst to the AAP, the data in Carpenter's paper had been doctored in such a way as to markedly lower the proportion of repeat SIDS that were considered "unnatural" deaths. In fact, all the deaths that were not clearcut filicide, and which in the earlier work of the same research group had been categorized as "unnatural," were recategorized to "natural" in the course of preparing the 2005 paper. This act lowered the "unnatural" rate from 34.5% to 13%. See Gornall, 2006, under "SIDS versus suffocation," above. --JKR]

13. The cause of SIDS. "The predominant hypothesis regarding the etiology of SIDS remains that certain infants, for reasons yet to be determined, may have a maldevelopment or delay in maturation of the brainstem neural network that is responsible for arousal and affects the physiologic responses to life-threatening challenges during sleep." [Life-threatening challenges in a crib??? --JKR]

I find it odd, given the work of Ackerman and the Italian researchers, that the Statement never mentions long QT, especially in the context of recurrent SIDS. --JKR

As to plagiocephaly, see Lynne Hutchison et al., Pediatrics 2004 Oct., below under "Unclassifiable."

[See also the AAP paper, "Changing concepts of SIDS: implications for infant sleeping environment and sleep position," 2001, above. -- JKR]

Sudden unexpected death in infancy and childhood due to undiagnosed neoplasia: an autopsy study.

Somers GR, Smith CR, Perrin DG, Wilson GJ, Taylor GP. Am J Forens Med P 2006 Mar; 27(1): 64-69. Found eight cases over twenty years in the autopsy files of the Hospital for Sick Children:

<u>Tumor</u>	<u>Age</u>
ALL	1 mo
AML infiltrating myocardium	2 mo
The others were outside the SIDS age range; see under "Unclassifiable," below.	

Some cardiologists recommend routine ECG screening of newborns. Laurie Barclay MD. Medscape Medical News 2006. These cardiologists at Pavia say "The availability of very effective therapies makes it very difficult to justify not proceeding in this direction. One important additional benefit is that the identification of the infants affected by LQTS will allow the identification of other affected family members and their protection as well." Cost effective. In the January 2, 2001 issue of *Circulation*, a Norwegian study reported that **10% of SIDS had genes for LQTS**. ECG could also pick up other CHD.

Sudden infant death syndrome. Hunt CE and Hauck FE. Canadian Medical Association Journal 2006 Jun; 174(13): 1861-1869. (Dr Hunt is at the National Heart, Lung, and Blood Institute.) The topic headings in this review article are:

- Introduction
- Pathophysiology
- Risk factors
 - Sociodemographic
 - Pregnancy-related
 - Maternal substance use
 - Infant sleep practices and environment
 - Infant feeding practices and exposures
- Genetic risk factors
- Interactions between genetic and environmental risk factors
- Infants at increased risk of SIDS
- Clinical strategies
 - Intervention
 - Reducing the risk of SIDS

Introduction states that SIDS is still the most common cause of postneonatal infant death. It accounts for about 25% of all the deaths between one month and one year of age. Its incidence has declined by more than 50%, largely because of safe-sleep campaigns. "There is evidence in some countries, however, that this remarkable progress is reaching a plateau. Changes in the classification of SIDS on infants by medical examiners...may be falsely reducing SIDS rates..." Further reduction will depend on more robust campaigns to reduce the modifiable risk factors.

Pathophysiology. Petechiae occur in 68%-95% of cases and are more extensive than in explained causes of infant death. The authors cite their own chapters in *Avery's Neonatology* and *Nelson's Pediatrics* to the effect that in research autopsies, large proportions (2/3) of SIDS infants had identifiable changes in lungs, other organs, and brainstem structure and function suggestive of chronic hypoxia. These include increased fascicular endothelial growth factor (VEGF) in the CSF at autopsy (citing Krous et al., 2003), structural and neurotransmitter alterations in the brainstem including increases in dendritic spines (indicative of delayed maturation of neurons), delayed maturation of synapses in the medullary respiratory centers, decreased tyrosine hydroxylase immunoreactivity in catecholaminergic neurons, and decreases in serotonin receptor immunoreactivity. 60% showed hypoplasia of the arcuate nucleus. Decreases in binding to kainate, muscarinic, and 5-HT receptors.

Risk factors. Box 1 lists all the environmental risk factors -- antenatal and postnatal:

Antenatal/ maternal

Postnatal

Smoking	Age 2 – 4 mos (but may be decreasing)
Alcohol	Male
Drug abuse esp. opiates	Black/ AI race
Inadequate prenatal care	No pacifier at bedtime
Lower class mother	Prematurity
Younger mother	Prone or side
Uneducated mother	Recent febrile illness
Single mother	Tobacco smoke
Multiparity	Soft bedding
Short interpregnancy interval	Overheating
Intrauterine hypoxia history	Face covered by bedding
IUGR	Cosleeping
	Sleeping in a separate room
	Winter season

The text divides the risk factor discussion into subheadings:

Sociodemographic. Mentions that in some countries the age for SIDS has been decreasing, with a narrowing of the age distribution into the earliest months. Also the seasonal predominance has been flattening out. The male sex continues to have a 30 – 50% higher incidence.

Pregnancy-related. Factors suggest a suboptimal in-utero environment.

Maternal substance abuse. A major association with cigarettes. (See Surgeon General's report, below). Infants of smoking mothers are 5X more likely to die of SIDS, and the risk is dose-related. There may be a small independent effect of paternal smoking. As to illegal drugs used prenatally, studies have shown a 2X to 15X increase in risk of SIDS for opiate use, but have NOT found an association with alcohol use, either prenatal or postnatal, except in certain special situations, namely,

periconceptional/ first trimester binge drinking in Indians (Iyasu et al., 2002)
alcohol use in the last 24 hours before the baby died (Netherlands study, 1998)
fetal alcohol syndrome 8X increased risk (Burd et al, 2004)

Sleep practices. As prone sleeping has become less common, the odds ratio for SIDS if they do sleep prone has been increasing; por ejemplo, from 2X to 11X in Norway. A special fatal situation for “unaccustomed prone,” that is, infants who were placed prone for the first time in their lives, or “secondary prone,” that is, infants who for the first time were found prone even though not placed that way. The unaccustomed prone is more apt to occur in out-of-home care settings or with new caregivers. Side sleep is no good: it gives a 2X greater risk than supine. (Li, Pettitti, Willinger et al., 2003). Discusses retraining hospital nursery staff on this. Soft bedding and beanbag pillows give a 2 – 3X greater risk. If you combine the risks, e.g. prone sleeping + soft bedding, you get 20X. Heavy comforters. Overheating + prone = 6 – 10X. But hot weather does not increase risk (!) As to cosleeping, several studies have implicated it as an independent risk factor for SIDS. Two studies (England and NZ) showed risk only if mother smoked. (Blair, Fleming et al., 1999; Scragg et al., 1993). But more recent studies have found no smoking factor, but have found a risk in nursing. (Venneman et al., 2005; Carpenter et al., 2004; Tappin et al., 2005) “Bed sharing has been found to be extremely hazardous when other children are in the same bed, when the parent is sleeping with an infant on a couch or other soft or confining sleep surface, and when the infant is less than 4 months of age. (Citing Carpenter et al., 2004; Hauck et al., 2003; Blair, Fleming et al., 1999; Tappin et al., 2005; McGarvey et al., 2003). Risk is also increased with longer duration of bedsharing during the night...” There is some evidence that sleeping separately in the parents' room is actually protective.

Feeding practices. Some studies found that breastfeeding is protective, but it may be merely a marker for upper social class status. [See “On the job, nursing mothers find a 2-class system,” New York Times, September 1, 2006] As to pacifiers, they have been associated with a significantly lower risk of SIDS across all social classes. Why is this? The author suggests that maybe pacifier use and dislodgement increases arousability.. Goes on to discuss ancillary issues, such as whether pacifiers interfere with breastfeeding. As to URI's they have NOT been shown to be an independent risk factor. As to vaccination, “Fewer SIDS infants than control infants were found to have been immunized. However,

among immunized infants, no temporal relation between vaccine administration and death has been identified. Parents should be reassured that immunization does not present a risk for SIDS.” (Citing *Task force on SIDS. The changing concept of sudden infant death syndrome: diagnostic coding shifts, controversies regarding the sleeping environment, and new variables to consider in reducing risk.* Pediatrics 2005; 116: 1245-1255.)

Genetic risk factors. “Overall it is estimated that **5% - 10% of SIDS cases are associated with a defective cardiac ion channel and hence an increased potential for a lethal arrhythmia.**” Also, effective embryologic development of the autonomic nervous system. Five genes. The ability to shorten the QT interval as the heart rate increases appears to have been impaired in some SIDS infants.” (Citing Franco et al., 1999, from that Italian group.)

Interactions between genetic and environmental risk factors. Gives a diagram, in which most of the arrows from both sides point to “impaired autonomic regulation” as the final common pathway to a SIDS death. On the sides are many of the risk factors we have mentioned above.

Infants at increased risk of SIDS. Infants at increased risk of SIDS include those who have had an ALTE, siblings of prior SIDS infants, and preterm infants. A history of ALTE is present in 5% - 9% of SIDS infants, and the risk of SIDS may be 5X greater in infants with such a history. As to sibling risk, it has been found that siblings of infants dying of any natural cause, including SIDS, are at significantly increased risk of dying from the same cause. 5 – 13X for natural diseases and 5 – 6X for SIDS. But the difficulty of ruling out intentional suffocation confounds the data and makes it difficult to tell a definite risk. “However, there are now substantial data in support of genetic risk factors for recurrent SIDS, and recent epid data confirm that second infant deaths in families are not rare and that at least 80% - 90% are natural. Recurrent infant death from SIDS in subsequent siblings is 6 times more likely than from homicide.” (Citing Carpenter, Waite et al., Repeat sudden unexpected and unexplained infant deaths: natural or unnatural? Lancet 2005; 365: 29-35). As to birth weight and gestational age, there is an inverse relation between birth weight/ gestational age and incidence of SIDS.

Quotes the AAP guidelines to reduce the risk of SIDS, in a separate box.

Smoking report 2006. The health consequences of involuntary exposure to tobacco smoke: a report of the Surgeon General, available at http://www.cdc.gov/Tobacco/sgr/sgr_2006/index.htm Thanks to Dr Keens for making us aware of this report. Dr Keens advises us, in a memorandum submitted to the August 10th, 2006 meeting of the Southern California Regional SIDS Council, that Chapter 5 of this report is entitled “Reproductive and developmental effects from exposure to secondhand smoke,” and that it provides a comprehensive review of epidemiological studies of this. Nearly all the studies “have an odds ratio greater than one...” Dr Keens summarizes the argument as (1) multiple epid studies consistently show an increased risk of SIDS. (2) Some studies show a dose-response relationship. Therefore “a level of risk that is almost certainly causal.” The report concludes that “The ev is sufficient to infer a causal rel between exposure to secondhand smoke and SIDS.” Dr Keens comments that the wording of this conclusion differs from the wording seen elsewhere in the report, where it is merely stated that smoke “increases the risk” of SIDS. Dr Keens says, “There is no claim that biological studies have now demonstrated a cause for SIDS that we did not previously know. Obviously, many infants who die from SIDS were not exposed to environmental tobacco smoke, and so it is difficult to see that it is a cause. (It would not satisfy Koch’s postulates).” [Getting back to the triple-risk hypothesis, this risk fits nicely. –JKR] [Well, Tom, take a look at the report of Shah et al., 2006, below. –JKR] The causality statements in this report have become controversial. See the Surgeon General’s report at <http://www.surgeongeneral.gov/library/secondhandsmoke/> and the Surgeon General’s press conference of June 27th, 2006 at <http://www.surgeongeneral.gov/news/speeches/06272006a.html>

Effect of prone and supine position on sleep, apneas, and arousal in preterm infants. Bhat RY, Hannam S, Pressler R, Rafferty GF, Peacock JL, Greenough A. Pediatrics 2006; 118(1): 101-107. As reviewed by William T. Basco, Jr., MD, FAAP. Medscape Pediatrics 2006; 8(2), posted 10/3/2006. 24 infants of less than 33 weeks gestation were monitored with video-polysomnography for two 3-hour periods on 2 consecutive days; the first day they were prone, the second day they were supine. Found that prone position was associated with more sleep and more efficient sleep. Slept for 146 minutes in the prone position and only 116 minutes in the supine position. Prone spent more time in quiet sleep, with fewer awakenings and arousals. However, central apneas were much more common among infants sleeping in the prone position (5.6 on average) than among infants sleeping in the supine position (2.2).

Multiple serotonergic brainstem abnormalities in sudden infant death syndrome. (Preliminary Communication). Paterson DS, Trachtenberg FL, Thompson EG, Belliveau RA, Beggs AH, Darnall R, Chadwick AE, Krous HF, Kinney HC. JAMA 2006 Nov; 296(17): 2124-2132, with accompanying editorial by Dr Weese-Mayer. [SO! I see that Dr Chadwick's daughter is now a medical student! I can't say I'm surprised!] Froze medullae from 31 SIDS cases and 10 control infants obtained from the San Diego Medical Examiner's Office under the blanket-consent provisions of the California Government Code regarding Coroners in SIDS and nonSIDS cases, Government Code Sec. 27491.4 and 27491.45a. Used autoradiography to determine the following four items in the raphe obscurus:

- 5-HT neuron count (elevated in SIDS)
- 5-HT neuron density (elevated in SIDS)
- 5-HT receptor binding density (absolutely reduced in SIDS) (the 5-HT-1A receptor)
- 5-HTT binding density (relatively reduced in SIDS) (5-HT transporter protein)

The structures analyzed were: In the rostral medulla: the midline raphe, lateral extraraphe, and ventral surface. In the midmedulla: same

Says the serotonergic system of the medulla oblongata consists of 5-HT neurons located in the midline raphe, lateral extraraphe, and ventral surface, and helps regulate autonomic and respiratory function. (citing their own chapter in a 2004 textbook). These medullary nuclei are interconnected (citing their own 2001 art) and project extensively to nuclei in the brainstem and spinal cord that influence respiratory drive, blood pressure regulation, thermoregulation, upper airway reflexes, and arousal. located in the midmedulla and rostral medulla. Discusses the genetics of these receptors. Refers to their 2005 case report of one male neonate who displayed altered autonomic and respiratory function at birth and 5-HT receptor binding abn at autopsy 2 weeks after birth. The pathogenesis of this abn remains to be determined. There are up to seven subtypes of 5-HT receptors; they chose the 1A subtype because it is found in high density in regions of the medulla in which binding was most severely reduced in their previous studies and because it is a somatodendritic autoreceptor that plays important roles in cardiorespiratory control. [Speaking of density, their writing is so dense that I almost can't condense it.] Their controls were age- and race- congruent and were 2 drownings, 1 plastic bag, 2 pneumonia, 1 GBS, 1 unsuspected congenital heart disease, 2 known congenital heart disease, and 1 inborn error of metabolism. They located the populations of serotonergic neurons in the chosen levels of the rostral medulla and midmedulla by first immunostaining frozen postfixed sections for tryptophane hydroxylase. They counted the + immunostained neurons by computed morphometry. Then they did quantitative autoradiographic densitometry on adjacent sections for the 5-HT subtype 1A receptor and the 5-HT transporter protein.

The receptor binding was significantly lower in male SIDS (n=6) than in female SIDS (n=10) (P=.04). It was significantly higher in both male and female controls than it was in male or female SIDS cases. (P=.02 and .05 respectively). But there was overlap.

The lesser receptor binding in male SIDS "may help explain why males are more vulnerable to SIDS."

The abnormalities found did not correlate with any SIDS risk factors, including sleep position and cosleeping. Therefore, it is an independent variable, a common underlying vulnerability, which fits with the triple-risk hypothesis. The authors propose that "The increased risk of SIDS in the prone or facedown position may reflect the infants' inability to respond to the asphyxial or hypercarbic challenge in the facedown position, due to the abn in the mdullary 5-HT system that compromise protective reflexes, including arousal and head turning."

The authors say that before the nature and pathogenesis of the medullary 5-HT dysfunction in SIDS can be determined, it will be necessary to do two things: (a) elucidate the pathways and mechanisms regulating the expression of the markers they analyzed here, and (b) determine the medullary 5-HT level in SIDS.

The editorial comments that while these are elegant studies, the study population is nonrepresentative of SIDS because it did not include any blacks.

Sudden infant death syndrome and reported maternal smoking during pregnancy. Shah T, Sullivan K, Carter J. Am J Pub Health 2006 Oct; 96(10): 1757-1759. Smoking gave an odds ratio for SIDS of 2.6.

The authors estimate that 21% of SIDS cases could have been prevented if women had not smoked during pregnancy. Also that “61.3% of the SIDS cases in children born to women who smoked during pregnancy were a *result* of smoking.” [emphasis added --JKR] “A primary modifiable risk factor.”

Caregivers in death, dying, and bereavement. International Working Group on Death, Dying, and Bereavement. *Death Studies* 2006; 30: 649-663. How do caregivers get into this field and how do they manage?

Meaning-making in the aftermath of sudden infant death syndrome. Guenther Krueger. *Nursing Inquiry* 2006; 13: 163-171. There are current theories of bereavement. Parental bereavement is now described by Wheeler (2001) as a search for meaning, and as an attempt to reconstruct a shattered world. Restructuring a new reality. This differs from the trajectory of healing described in older theories of grief.

Extramedullary hematopoiesis in liver of sudden infant death cases. Klara Toro, Martha Hubay, and Eva Keller (Semmelweis University, Budapest). *FSI* 2006. Did 51 SIDS cases and 102 nonSIDS cases. Used H&E and anti-Hemoglobin A immunohistochemistry. Found a higher freq of EMH in SIDS livers than in controls. $P=.0474$. This may be a consequence of anemia associated with intrauterine hypoxia, or infections. Complicated theory. Hypoxanthine increases with acute hypoxia, as shown by Rognum in vitreous humor in 1988.

Sudden infant death syndrome(SIDS) -- standardised investigations and classification: recommendations. Bajanowski T, Vege A, Byard RW, Krous HF et al. *FSI* 2006. An international consortium pleads for standardized autopsy methods and diagnostic definitions. Incorporates the 2004 San Diego definitions.

The role of beta-amyloid precursor protein staining in the neuropathologic evaluation of sudden infant death and in the initiation of clinical investigations of subsequent siblings. Byard RW, Blumbergs P, Kennedy JD, Riches KJ, Martin J, Thompson GN. *Am J Forens Med P* 2006 Dec; 27(4): 340-344. Presents the case of a SIDS infant in whose corpus callosum and internal capsule the authors discovered linear and granular BAPP positivity, not matching either the pattern of TAI or of vascular axonal injury (VAI). Having ruled out those two possible causes also by history (there was no hint of trauma or vascular events in the prenatal or postnatal background), the authors ascribe this BAPP + to occult previous episodes of hypoxia due to central apnea. When a subsequent sibling was born, the authors studied him and found by polysomnography that he DID suffer from spontaneous central apnea with desaturations. Hence a familial disorder. The authors take this opportunity and use this case to demonstrate that in order to properly evaluate a SIDS brain, you have to do specialized studies such as BAPP. They decry the practice of not having formal NP done and even worse the “unfortunate situation” of the brain sometimes not being dissected at all. “[T]his report demonstrates that there is a distinct role for formal neuropathology, and in particular immunoh staining of brain tissue for BAPP, in infants who present with sudden and unexpected death...”

Are the risk factors for SIDS different for preterm and term infants? Thompson JMD, Mitchell EA for the New Zealand Cot Death Study Group. *Arch Dis Child* 2006; 91: 107-111. From Dr Reece’s review in the *Quarterly*, all the risk factors were the same, except for parity, which was a risk factor for terms but not for preterms. Preterm itself is a risk factor.

SIDS & Infant Death: Program Manual and Trainer’s Guide, 2006 (CD). Put out by the US Department of Health & Human Services, Health Resources & Services Administration. Includes a 175 page manual and a trainer’s guide in the form of slides. The manual covers the topics, 1. What is SIDS? 2. Risk reduction education 3. Bereavement support services 4. Training 5. Program expansion 6. Research & statistics 7. Partnerships & collaborations 8. Public relations and the media 9. Fundraising & development. Appendix A: History of SIDS. Appendix B: Glossary. Appendix C: Bibliography. As to topics 7 and 8, compare to the comparable parts of Victor Vieth’s agenda for ending child abuse in 120 years (below under “Unclassifiable.”) Regarding the need for propaganda, bureaucracy, lobbying.

Sudden infant death syndrome: a subject of medicolegal research. (review article). Sharma BR (India). *Am J Forens Med P* 2007 Mar; 28(1): 69-72. Reviews the basics from 1969. Refers to the views of

Huber and Rambaud that the entity should not be recognized because it serves no useful purpose. Refers to the 40% decline and back-to-sleep. Infanticide possibility makes scene and records investigation mandatory. Under “factors associated with SIDS,” discusses

bedsharing
thermal stress
bedding material
prone sleeping
sleep apnea

As to bedsharing, references five studies that cosleeping increases the risk of SIDS. (Bass, Kravath, NEJM 1986; Luke, JFS 1977; Norvenius, Scand 1987; Scragg, BMJ, 1993; Fleming Blair, 1996). Overlaying hypothesis. Controversial. “Some studies have postulated that bedsharing may reduce the risk of SIDS by potentiating the bonding process and prompting breastfeeding.” (citing McKenna, Mosko, 1997). Mentions rebreathing maternal carbon dioxide hypothesis. Notes that the combination of bedsharing with maternal smoking greatly increases the risk, as reported by many researchers.

As to thermal stress, says infant’s inadequate thermoregulatory mechanism could lead to hyperthermia in the presence of copious bedding and high room temperature, a hypothesis that cannot be tested by autopsy.

As to bedding material, refers to excessive softness and tendency to trap carbon dioxide in the case of prone sleeping -- essentially a suffocation mechanism, or mechanical asphyxia. “Bedding may play a role in some infant deaths...” Citing Kemp et al., 1998. But supine sleeping is not exempt from bedding: bedding that covers the face or entire head poses a potential danger of causing “asphyxia stress.” 2% of newborns have no CO2 response. (Citing Enid Barness et al., Am J Forens Med P 1991).

As to prone sleeping, hypotheses to explain its risk include mechanical asphyxia, rebreathing, airway obstruction, and backpressure on the mandible. (Citing Waters et al., face straight-down sleeping, 1996). “The prone position has also been associated with decreased arousal, increased sleep duration, and an increased amount of non-REM sleep.” (Citing Sleep, 1996).

As to prolonged sleep apnea, associated with young age, prematurity, and infection, suggested that respiratory viruses may trigger apnea. Also cow milk allergy, mites, botulism, spinal hemorrhages (?), selenium deficiency, vitamin E deficiency, metabolic disease are “frequently reported factors” associated with SIDS. (no citation)

Forensic investigation of SIDS: mentions lack of cooperation among agencies. Then goes through what things should be looked for at the death scene, autopsy samples.

Air pollution and infant death in southern California 1989-2000. Ritz B, Wilhelm M, Zhao Y. Pediatrics 2006 Aug; 118(2): 493-502. Correlated the amount of NO, O3, CO2, and particulates in the air with paired birth and death certificates for all infant deaths (not just SIDS) compared with living controls. Found increases in the risk of respiratory death and for SIDS with increasing levels of pollutants. For example, the risk of respiratory death increased by 20 to 35% for each 1 ppm increase in the level of CO, SIDS risk increased 25-19% for each 1 ppm increase in NO.

The continuing decline in SIDS mortality. Mitchell EA, Hutchison L, Stewart AW. Arch Dis Chil 2007 Apr 3; <http://adc.bmj.com/cgi/content/abstract/adc.2007.116194v1> There was a rapid decline in the early 90’s. Prone sleeping fell to less than 4% and stayed there. This can account for the fact that SIDS has declined by 63%. “The most likely explanation for this decline has been the decrease in infants sleep[ing] on their side. This change wd result in a 39% reduction in SIDS. Side sleeping position doubles the risk of SIDS compared with the supine pos.

SIDS GENETICS

[Speaking of genetics, there is a nice CD put out in March 2007 by the American College of Medical Genetics that gives an introductory education in medical genetics calibrated to pediatricians.]

<http://www.acmg.net>

A serotonin malfunction hypothesis. Okado N, Narita N, Narita N. Medical Hypotheses 2002 Mar; 58(3): 232-236. 5-HT has an excitatory influence on the respiratory center. If 5-HT reaching this center is reduced, the symptoms of SIDS could be explained by that. We found that the L and XL alleles of the 5-HT transporter gene were more frequent and the S allele less frequent than in controls. Many of the other risk factors for SIDS (other than prone position) change the serotonin levels in the brain, so this gene product could be a common pathway for all these risk factors to cause the symptoms.

Possible role of mtDNA mutations in sudden infant death. Opdal SH, Vege A, Egeland T, musse MA, Rognum TO. (Oslo) Pediatric Neurology 2002 Jul; 27(1): 23-29. Studied a hypervariable region and a coding region in the gene for mtDNA in 257 sudden-death infants (some SIDS, some naturals due to infection) and 102 living infants. Found, as to substitutions in the hypervariable region and point mutations in the coding region:

	Avg # of substitutions	point mutations
SIDS	2.58	5.6% had
Borderline SIDS	2.63	2.9% “
Infections	3.28	11.1% “
Controls	2.02	2.9% “

“The results indicate that increased levels of HVR-1 substitutions may be an indicator of mtDNA instability. Furthermore, mtDNA mutations may play a role in some patents with SIDS that was unexplained or thought to be caused by infection.” Note by JKR: why did the infectious deaths have a higher rate of mutations? Apparently these mutations are more of a risk factor for infection than they are for sudden death as such.

Evidence for a genetic component in SIDS. Gordon AE, MacKenzie DA, El Ahmer OR, Al Madani OM, Braun JM, Weir DM, Busuttill A, Blackwell CC. (Edinburgh) Child Care Health Dev 2002 Sep; 28 Suppl 1: 27-29. Because there is “increasing evidence” that inflammatory responses have been present in some SIDS, these authors tested 41 SIDS parents and 61 adult controls for blood cytokine (interleukin) response to antigenic stimulation with Staphylococcal toxin and E. coli LPS. Found that the SIDS parents produced higher levels of IL-1. Then they did DNA testing of a small number of persons (10 SIDS parents, 10 local controls, and 10 exotics), and found that there was a polymorphism in the gene for IL-1 as follows:

SIDS parents	40%
Local controls	15%
Exotics	0%

Suggest that SIDS infants may have higher IL-1 levels due to a genetic polymorphism, and therefore react more strongly to toxins such as cigarette smoke. Also mention that this particular genetic polymorphism seems to be racially associated, so that some racial groups would be at higher risk than others (e.g. the Bangladeshi exotics tested.)

Interaction between apnea, prone sleep position, and gliosis in the brainstems of victims of SIDS. Sawaguchi T, Franco P, Kato I, et al. FSI 2002 Sep; 130 Suppl: S44-S52. A prospective study of 27,000 infants' sleep-wake behavior. Had 26 SIDS deaths and 12 natural-disease deaths. Analyzed any apneas recorded during the weeks leading up to the deaths. Then studied the brainstems for gliosis in the nucleus ambiguus, nucleus solitarius, and other structures related to cardiorespiratory control and sleep-wake function. Computed the density of GFAP-reactive astrocytes. All correlations were negative except for a correlation between obstructive apnea, prone position, and gliosis in the raphe nucleus of the midbrain. No relationship was found between the findings and the epidemiology of SIDS.

Medial smooth muscle thickness in small pulmonary arteries in SIDS revisited. Krous HF, Floyd CW, Nadeau JM, Silva PD, Blackbourne BD, Langston C. *Pediatr Dev Pathol* 2002 Jul-Aug; 5(4): 375-385. Found no thickening. Also noted that there was no RVH. This is evidence against a long-term-hypoxia theory of SIDS, such as that of Kinney et al.

Brain stem lesions in the SIDS: variability in the hypoplasia of the arcuate nucleus. Matturi L, Biondo B, Suarez-Mier MP, Rossi L. (Milan). *Acta Neuropathologica* (Berlin) 2002 Jul; 104(1): 12-20. Studied 62 cases of SIDS (mean age 3 ½ months) and 25 controls using morphometry. Divided their SIDS brainstems into four morphologic subcategories:

SIDS A	histologically well-developed arcuate nuclei
SIDS B	severe bilateral hypoplasia of the arcuate nuclei
SIDS C	partial bilateral hypoplasia
SIDS D	right unilateral hypoplasia

SIDS A was 27 cases, in other words, almost half the SIDS cases had normal arcuate nuclei. The morphometry of these was the same as controls. The other groups had decreased numbers of neurons.

SIDS: association with a promoter polymorphism of the serotonin transporter gene. Weese-Mayer DE, Berry-Kravis EM, Maher BS, Silvestri JM, Curran ME, Marazita ML. *Am J Med Genet* 2003 Mar 15; 117(A)(3): 268-274. From the abstract: Serotergic receptor binding in the arcuate n., n. raphe obscurus, and other medullary regions is decreased in SIDS brains. Further, a variable tandem repeat sequence polymorphism in the promoter region of the serotonin transporter protein (5-HTT) gene has recently been associated with risk of SIDS in a Japanese cohort. This polymorphism differentially regulates 5-HTT expression... We therefore investigated the 5-HTT promoter polymorphism in a cohort of 87 SIDS cases (43 AA and 44 cauc) and matched controls. Positive associations were found between SIDS and the 5-HTT genotype distribution, specifically for the L/L (long allele) genotype... There was also negative association between SIDS and the S/S (short allele) genotype. This could be used to identify at-risk individuals.

Hoppenbrouwers T and Hodgman J. *SIDS*. Women's and Children's Hospital, Newborn Division, Rm L919, 1240 North Mission Road, Los Angeles 90033, Attn: Dr. Toke Hoppenbrouwers. ISBN 09742663-0-2. Published 2003. Available by pdf at <http://www.hoppenbrouwers.net/toke/sids.asp>

Sudden Death in Infancy, Childhood, and Adolescence. Roger W. Byard. West Nyack, NY: Cambridge University Press, 2004. <http://www.cambridge.org>

The sudden infant death syndrome gene: does it exist? Opdal SH, Rognum TO. *Pediatrics* 2004; 114(4): e506-e512. A review article. Distinguishes between lethal mutations and predisposing mutations. Says lethal mutations like MCAD and LQT would account for only about 2% of SIDS deaths. But, harking back to the trivalent hypothesis,

Research review: Sudden infant death syndrome: review of implicated genetic factors. Weese-Mayer DE, Ackerman MJ, Marazita ML, Berry-Kravis EM. *Am J Med Genet Part A* 143a: 771-788 (2007). This paper is too dense to be summarized. The topics addressed are:

- genes for ion channel proteins
 - the Schwartz-QT hypothesis
 - molecular evidence linking the Schwartz-QT hypoth with LQTS
- genes for serotonin transporter
 - rationale for studying serotonin genes
 - neuropathological data
 - transporter gene (5-HTT; SLC6A4)
 - 5-HTT polymorphisms in SIDS
 - 5-HTT promoter
 - 5-HTT intron 2 VNTR

promoter and intron 2 combined analyses
5-HTTT 3' untranslated region and SIDS
genes pertinent to the early embryology of the autonomic nervous system
nicotine metabolizing genes
genes regulating inflammation, energy production, hypoglycemia, and thermoregulation

“As this review clearly indicates, a number of genetically controlled pathways appear to be involved in at least some cases of SIDS. Given the diversity of results to date, genetic studies support the clinical impression that SIDS is heterogeneous with more than one entity and with more than one possible genetic etiology.”

SIDS EPIDEMIOLOGY (see also TWIN SIDS, below)

Studies of the sudden infant death syndrome in King County, Washington. III. Epidemiology. Bergman AB, Ray CG, Pomeroy MA et al. Pediatrics 1972; 49: 860-870. Reported “risk factors” similar to the Sheffield study of the 1980’s. Study of 1065 infants classed as SIDS in the 1960’s, finding a more than random incidence of low birth weight, single mother, etc. But this work was later revised, when it was noticed that the risk factors in fact only occurred in a small proportion of the infants, all of whom had positive findings at autopsy, and thus were distinguishable on both epidemiologic and anatomic grounds from “true SIDS.” See “Wrong turns in SIDS research,” 1997.

Death-scene investigation in sudden infant death. Bass M, Kravath RE, Glass L. NE J Med 1986 Jul 10; 315(2): 100-105. They did death-scene invest in 26 presumptive SIDS. In 24 out of 26 the scene investigation revealed possible causes of death such as asphyxiation by object in crib, overlaying, hyperthermia, SBS. Reached two conclusions:

1. A high SIDS rate in a lower-class population should be questioned.
2. Scene investigation will reveal a definable cause of death in many cases.

Socioeconomic differentials and neonatal mortality: racial comparison of California singletons. Gould JB, Davey B, LeRoy S. Pediatrics 1989 Feb; 83(2): 181-186

SIDS epidemiology: a review and update. Little RE and Peterson DR. Epidemiologic Reviews 1990; 12: 241-246. Argues basically for a stratification of SIDS for further research. Says that the definition by mere exclusion is resulting in a mixture of cases that are not comparable. “There has been no systematic categorization of SIDS by subtype.” “Case definition based on diagnosis by inclusion from objective, reproducible evidence, rather than by exclusion as is currently practiced, holds the greatest promise for telling advances in future epidemiologic research.” 245.

Epidemiology of SIDS: maternal, neonatal, and postneonatal risk factors. Hoffman HJ, Hillman LS. Clin Perin 1992; 19(4): 717-737. Including the maternal factors of young age, multiparity, low education, no PNC or late PNC, and unmarried. Cited by the CDC in “Guidelines for Death Scene Investigation of Sudden Unexplained Infant Deaths” (1996).

Wrong turns in SIDS research. Abraham B. Bergman. Pediatrics 1997 Jan; 99(1): 119-121. “Speaking of being wrong, our Seattle group was among the first to identify the epidemiologic risk factors associated with SIDS. (citing Pediatrics, 1972) As the years went by, we were struck by the lack of risk factors in the vast majority of the SIDS victims we studied. This clinical impression led our pathologists and clinicians to reexamine and reclassify the 1065 cases previously diagnosed as SIDS, and compare these findings

with birth certificate information. As suspected, we found that the social and/or demographic risk factors clustered in a relatively small proportion of infants, most of them with anatomic findings at autopsy, and that a significant proportion of classic-SIDS infants could not be distinguished from control infants. Two lessons emerged from this study: (a) the need for precise and uniformly applied diagnostic criteria in the postmortem examination, and (b) the need in SIDS research to concentrate on as homogeneous a population as possible, i.e., the classic SIDS cases, rather than on the relatively small proportion of 'outliers.' ” For further work along this line by the Seattle group, see Bass et al., NEJMed, 1986.

Facts about SIDS deaths in California (from the California Department of Health Services, Vital Statistics and Death Records, 1998. Courtesy of Grant Neie, RN.

1. Overall US SIDS rate 149/100,000 LB 1996
2. x
3. SIDS is the leading cause of death among infants from one month to one year
4. Male:female = 63% : 37%
5. California SIDS deaths 250 per year 1996
6. Maternal age at birth under 20, 12% of live births, 22% of SIDS 147/100,000 LB
7. Birth weight under 2500 grams SIDS rate 281/100,000 LB Calif
Birth weight under 2500 grams, mother under 20 SIDS rate 668/100,000 LB Calif
8. SIDS rate by race, 1998

White	56
Black	201
Asian	33
Hispanic	28
7. 93% of SIDS happened before 6 months.

Epidemiology of the SIDS: maternal, neonatal, and postneonatal risk factors. Hoffman HJ and Hillman LS. Clin Perin 1992; 19(4): 717-737.

MMWR: Sudden infant death syndrome -- United States, 1983-1994. MMWR Morb Mortal Wkly Rep 1996 Oct 11; 45(40): 859-863. The leading cause of postneonatal mortality in the US, accounting for 1/3 of all postneonatal deaths. Analyzes age-specific and race-specific trends for 1983-1994. The rates of SIDS declined three times faster during 1990-94 than they had during 1983-89.

The changing epidemiology of SIDS following the national risk-reduction campaigns. Mitchell EA. Pediatr Pulmonol Suppl 1997; 16: 117-19. The mechanism by which the prone sleeping position causes SIDS is in some way related to temperature, because the effect of prone sleeping position is different depending on season, latitude, illness, thermal insulation, and sheepskins.

Mortality within the first 2 years in infants exposed to cocaine, opiate, or cannabinoid during gestation. Ostrea EM Jr, Ostrea AR, Simpson PM. Pediatrics 1997 Jul; 100(1): 79-83. Conclude that prenatal drug exposure in infants, although associated with a high perinatal morbidity, is not associated with an overall increase in their mortality rate or incidence of SIDS during the first 2 years of life. However, a significantly higher mortality rate was observed among low birth wt infants who were + for both cocaine and opiate.

Intrauterine cocaine exposure and the risk for sudden infant death syndrome: a meta-analysis. Fares I, McCulloch KM, Raju TN. J Perin 1997 May-Jun; 17(3): 179-182. The increase in risk for SIDS was found not to be specific to cocaine but to intrauterine exposure to illicit drugs in general. Meta-analysis of ten published studies. **The odds ratio versus drug-free infants was 4.1**, but there was a problem with the confounding variable of polydrug exposure.

Abandoning prone sleeping: effect on the risk of SIDS. Skadberg BT, Morild I and Markestad T. J Pediatr 1998; 132: 340-343 Norwegian study: the incidence of SIDS dropped from 3.5 per thousand LB to 0.3 per thousand LB. The rate of prone sleeping fell from 64% to nearly zero. Of SIDS victims, 60% were placed prone for their final sleep. Smoking rates of mothers did not change during the study period.

Problems in the diagnosis of SIDS. Hata K, Funayama M, Tokudame S, Morita M. (Sapporo Univ). Acta Paediatrica Japonica 1997 Oct; 39(5): 559-565. There are no clear criteria differentiating SIDS from other causes of SUDS. Medical examiners can be divided into three groups: SIDS tolerationists, who think all SUDS should be dx'd as SIDS unless a clearcut cause of death appears, SIDS exclusionists, who think SIDS is ruled out by any positive microscopic findings or peculiar scene findings and a middle group. Thus the content of a SIDS dx will be different depending on the philosophy of the examiner who rendered it and possibly on the region where given philosophies prevail.

Modifiable risk factors for SIDS: when will we ever learn? J Pediatr 1998; 132: 197-198

Prevalence and predictors of the prone sleep position among inner-city infants. Brenner RA, Simons-Morton BG, Bhaskar B et al. JAMA 1998 Jul; 280: 341-346. These NICHD scientists found that 40% of these low-income inner-city infants were placed for sleep in the prone position. They found that this prevalence is higher than what occurs in general population. Also that a predictor of prone was the fact that the mother had seen nurses in the hospital placing her baby in the prone position to sleep; 93% of the mothers who had seen this placed their infants prone to sleep. Also the presence of a grandmother in the home was a predictor. Relative risks: poverty 1.8, black 2.0, grandmother 1.8.

Fact sheet from California Department of Health Services, 1998:

1. SIDS is the leading COD for infants 1 mo to 1 yr.
2. Males 65% females 35% of SIDS.
3. 300 babies a year in California
4. SIDS rate among mothers under 20 = 147 per 100,000 LB, 2X normal.
5. Rates by race in California: W 64, B 230, H 34, A36 per 100,000 LB
6. November through March
7. 93% under 6 mos. (graph)
8. Calif rate fallen by 40% since 1988. (graph)

Changes in the epidemiologic profile of SIDS as rates decline among California infants: 1990-1995.

Adams EJ, Chavez GF, Steen D, Shah R, Iyasu S, Krous HF. Pediatrics 1998 Dec; 102(6): 1445-1451. The rate among Blacks declined less than in the other racial groups. Risk-reduction messages need to be reformulated to reach this population. Also different risk factors or risk-factor weightings may exist. "Differences in SIDS reduction between groups may be related ... to differences in the distribution of risk factors between groups. As shifts to nonprone sleep positions occur, the relative importance of other risk factors can be expected to increase. Additional work is needed to reevaluate the most important indicators of risk for each group..."

SIDS: risk factor profiles for distinct subgroups. Kohlendorfer U, Kiechl S, Sperl W (Innsbruck). Am J Epidemiol 1998 May; 147(10): 960-968. 99 SIDS and 136 controls in Tyrol. They identified two subgroups of SIDS who had different risk conditions and ages at death -- a clearcut bimodal distribution of deaths breaking out at age 4 mos which had different risk factors. "Early SIDS" and "late SIDS." The risk factors broke out as follows:

Early SIDS: prematurity, apnea, LBW, previous +FH of SIDS, prenatal smoking
 Late SIDS : seasonality
 Both SIDS: inadequate PNC, lower class, prone sleeping

This article is commented on by Kenner T, Einspieler C, Haidmayer R, letter, Am J Epidemiol 1999 Apr; 149(8): 785-786.

Abandoning prone sleeping: effect on the risk of SIDS. Skadberg BT et al. J Pediatr 1998 Feb; 132(2): 340-343. Before the campaign, 64% of infants slept prone and the SIDS rate was 3.5 per thousand LB. After the campaign 1.4% of infants slept prone and the SIDS rate was 0.3 per thousand. Other sleep positions noted. Conclusion: SIDS is rare when prone sleeping is avoided. Side sleeping bad.

Sociodemographic risk factors for SIDS: associations with other risk factors. The Nordic Epidemiological SIDS Study. Acta Paediatrica 1998 Mar; 87(3): 284-290 Low maternal age, high birth order, single mother, low maternal education, low paternal education, maternal unemployment, paternal unemployment. Odds ratios computed. Smoking was an independent variable. "Sociodemographic differences remain a major concern in SIDS in a low-incidence situation and even in an affluent pop with adequate health services" (meaning Scandinavia).

Influence of increased survival in very low birth weight, low birth weight, and normal birth weight infants on the incidence of SIDS in the US 1985-1991. Bigger HR et al. J Pediatr 1998 Jul; 133(1): 73-78. The marked increase in the survival of VLBW infants increased the pool of babies at potential risk for SIDS. VLBW infants SIDS rates have not changed, while those for LBW and NBW infants have fallen.

Reducing the risk of SIDS: a review of the scientific literature. Henderson-Smart DJ, Ponsonby AL, Murphy E. J Paediatr Child Health 1998 Jun; 34(3): 213-219 See above under SIDS Biology.

Sudden unexplained deaths in infancy: what are the causes? Cote A, Russo P, Michaud J. J Pediatr 1999 Oct; 135(4): 437-443. They reviewed all 623 cases of SIDS in Quebec. Overall 80% went out as SIDS. The percentage of non-SIDS was higher in specialized pediatric autopsy centers (19% vs. 15%). They recommend that as the SIDS rate declines, SIDS autopsies should be done in specialized centers.

The effect of the weekend on the risk of SIDS. Spiers PS, Guntheroth WG. (Seattle). Pediatrics 1999 Nov; 104(5): e58. They compared two groups: upper-class infants going to downclass for day care during the week and coming home for the weekend, versus working-class infants going upclass for day care during the week and coming home for the weekend. Then counted how many of each group died of SIDS on the weekend as compared with during the week. They were testing the hypothesis that going downclass should increase risk for SIDS. And it did. The overall weekend-to-week ratio for all infants was 1:1. But for working-class infants coming downclass for the weekend the weekend-to-week ratio was 1.13. For upper-class infants coming upclass for the weekend the weekend-to-week ratio was 0.55. See also the Swedish paper Feb. 2001 below, noting a weekend propensity. See also Mitchell, 1999, below.

[SIDS and unaccustomed prone sleep position] Mitchell EA et al. Arch Ped Adol med 1999 Nov; 153: 1136-1141. Basically about day care. Cf Spiers & Guntheroth, 1999, above.

Usual sleep	Day care sleep	# SIDS	# controls	odds ratio
Supine	supine	125	1026	1
Prone	supine	12	32	3
Supine	prone	31	14	19
Prone	prone	218	507	4

Non-lethal and subtle injury and unexpected infant death. Byard RW, Donald T, Chivell W. J Law Med 1999; 7: 47-52.

Bruce Beckwith: "At the 1989 SIDS definition conference sponsored by the NIH, and the 1992 International SIDS Conference in Sydney, Australia, I tried without success to introduce language that would distinguish "classical" SIDS from cases that fit part of the profile but not all of it. A diagnosis such as "Category 1 SIDS," for example, could be used for a clearcut case, and "Category 2 SIDS" for cases that some experts might consider acceptable and others would not. I proposed criteria for Category 1, including age 3 weeks to 8 months, found dead after presumed or observed sleep, lack of evidence of neglect or abuse, negative history of similar deaths in sibs, other close relatives, or in custody of the same caregiver, and absence of histological markers of significant stress. Cases that fail to meet any one or more of these criteria might, when appropriate, be designated as SIDS, but with a qualifying adjective in the term." "More rigorous criteria for true SIDS than we proposed in 1969 would have prevented many infanticides, facilitated the recognition of lethal genetic disorders mistakenly called SIDS, and prevented the misuse of this term to cover up medical negligence, etc." Source: PEDPATH 8/7/99.

Mild and moderate preterm birth an important cause of infant mortality. Reuters Medical News from Medscape, summarizing an article in the Aug 16 2000 issue of JAMA to the effect that overall infant mortality in US among moderate preterm infants (32-33 weeks) was 6.6X term rate. Among mild preterm (34-36 weeks) it was 2.9X. (relative risk) JAMA 2000 Aug 16; 284: 843-849.

Racial disparities found in well-child care. Reuters Medical News on Netscape, citing Ronsaville DS and Hakim RB, Am J Pub H 2000 Sep; 90: 1436-1443 to the effect that only 35% of Black infants of all social classes received adequate well-child care under the AAP criteria, compared with 53% of white and 37% of Hispanic. Was true across class lines, so was a true racial disparity, rather than a class disparity. A later art shows that the working poor have inadequate access to health care. And indeed most of our SIDS cases come from the working poor.

A clinical comparison of SIDS and unexplained sudden infant deaths: how healthy and how normal? Platt MW, Blair PS, Fleming PJ, Smith IJ, Cole TJ, Leach CE, Berry PJ, Golding J. (Newcastle). Arch Dis Child 2000 Feb; 82(2): 98-106. Population-based case-control study compared the clinical histories of SIDS and SUDS by doing parental interviews and chart reviews with four controls for each affected infant. Had 325 SIDS, 72 SUDs, 1,588 controls. They used illness markers in the last 24 hours before death, and also previous ALTE. Previous ALTE did not differ between SIDS and SUDS but they both were increased above controls. Acute illness markers were elevated over controls in both index groups. Their acute illness scoring system is called "Baby Check," which is designed to mark for possible indicia of serious illness. Since it did score significantly higher for SIDS/SUDS than for controls, they suggest that it might identify babies at risk of sudden death, particularly if its screening use is applied to a high-risk subpopulation. Since the back-to-sleep campaign, SUDS cases are largely coming from poverty homes where social and demographic factors may contribute to death. These researchers used family interviews and parent questionnaires to compare 325 SIDS families and 72 sudden explained death families to 1588 case controls with living children families matched for age, four control families for each SIDS family. The explained-death cases were caused by infection (46%), accidents (15%), congenital anomalies (14%), and NAI (13%), and other conditions such as metabolic disease, BPD, etc.. Did univariate and multivariate analysis. Found that SIDS compared to case controls and explained deaths had shorter gestations, lower birth weight, more need for neonatal resuscitation and NICU care, more colic, poor weight gain, infections, ALTE's, congenital anomalies and symptoms during the final 24 hours. So the SIDS infants were less healthy. Supports the idea of a population of at-risk infants who are unhealthy, i.e. atypical SIDS.

SIDS in child care settings. Moon RY, Patel KM, Shaefer SJM. Pediatrics 2000 Aug; 106(2): 295-300. If we apply the current incidence of SIDS to census data on use of day care centers and family child-care homes, we would expect that about 7% of SIDS should happen in such settings. But our study in two states shows that 20% occurred there. Factors associated with SIDS death in child care settings were older age, race, and highly educated parents. Retrospective study for 1995-97 from SIDS databases at the state level in eleven states. 1,916 SIDS cases. In many cases the child slept on his back at home in his white upper-class home and was placed prone for the first time in the usually working-class "family" child care setting.

Changing trends in the diagnosis of sudden infant death. Mitchell E, Krous HF, Donald T, Byard R. Am J Forens Med Path 2000 Dec; 21(4): 311-314. 114 consecutive cases of SUDS in South Australia over a five year period from 1994 to 1998. 45 SIDS, 19 NAT, 21 ACC, 5 HOMI, 24 UNDET. Noted an increase in the dx of accidental asphyxia and an increase of "cases in which the family background and autopsy findings suggested more complex mechanisms." Believes that more thorough examination has resulted in identifying these factors which probably went undiscovered in earlier years due to less thorough investigation in those days. **The increase in UNDETS was due to better investigation**, not due to a real increase in such deaths. "Now that additional information is available on social background, it appears that a significant percentage of families in which infants die unexpectedly have chaotic lifestyles with poor living circumstances, multiple partners, frequent changes of address, intravenous drug use, and histories of domestic violence. This, in combination with apparently 'minor' but nonetheless significant nonlethal inflicted injuries, precludes the use of SIDS as a diagnostic category." (citing Byard et al., J Law Med 1999.)

Education about supine sleeping has reduced SIDS deaths in Sweden. Reuters Health on Medscape, 2001 Feb. Arch Dis Child 2001; 84: 24-30. SIDS declined from 1.1 per thousand in 1991 to 0.3 in 1999. The age at death fell from 90 days to 60 days. The high incidence during weekends persisted. Seasonality was unchanged. The odds ratios almost doubled for prematurity and smoking during pregnancy. They said that to reduce it further, they will have to reduce smoking and also improve "the quality of maternal care." In other words, they think the supine campaign has gone about as far as it can go.

Maternal and paternal recreational drug use and sudden infant death syndrome. Klonoff-Cohen H, Lam-Kruglick P (UCSD) Arch Ped Adol Med 2001 Jul; 155(7): 765-770. Finding that maternal drug use during pregnancy was not associated but paternal marijuana use was.

Increased risk of SIDS in younger siblings tied to genetics. Reuters Health on Medscape, Aug. 23, 2001. Citing a study by Dr. Carl E. Hunt, director of the National Center on Sleep Disorders Research in Bethesda, MD, in the American Journal of Respiratory and Critical Care Medicine, August 2001. The SIDS risk he says is 5X that of the general pop, but "the risk remains so low that fewer than one in 200 younger siblings will succumb to SIDS." His meta-analysis of 143 articles found evidence that genes controlling development of the brain stem "are at least partly responsible for impaired neural control." Nearly half the studies of SIDS siblings and a somewhat smaller percentage of parents found brainstem autonomic abnormalities. Deficiency in arousal responsiveness. Familial aggregation of deficient hypoxic responsiveness in asymptomatic adult family members. He acknowledges that the older SIDS-sibling studies "have been discredited," but says that this pattern continues in the more recent studies in which ascertainment of SIDS was tighter.

Reducing the incidence of SIDS in the delta region of Mississippi: a three-pronged approach. Kum-Nji P, Mangrem CL, Wells PJ. Southern Medical Journal 2001; 94(7): 704-771. (have) Gives a good summary of the state of knowledge about SIDS to 2001, and a useful graph of all causes of infant death. Current US incidence 3,000 deaths per year or about 1/3 of all infant deaths, or 0.77 per 1,000 live births (citing CDC figures for 1983-1997). By 6 to 8 months of age, more than 95% of cases have occurred. Graph shows that in rural Mississippi complications of LBW are a much more common cause of death than SIDS (46% as opposed to 20%). In third place is congenital heart diseases (8%). Child abuse is way down there (2%). Gives tables summarizing (A) the ten etiologic hypotheses and (B) the fifteen risk factors. As follows:

Etiologic hypotheses:

1. Thermal stress due to overbundling results in severe hyperthermia
2. Prone causes CO₂ rebreathing which depresses the cardiorespiratory center.
3. Premature infants forget to breathe during nonREM, which they hv more of.
4. Sudden flood of bacterial toxins
5. Bottle feeding low in immunity and PUFA
6. Tobacco smoke toxins cause brain damage w/wo bacterial toxin interaction
7. Soft bedding traps CO₂ for rebreathing
8. Cosleeping might give CO₂ from adults. But might be good also bc nursing.
9. Long QT syndrome
10. Maternal substance abuse (MSA) during gestation cause brain damage.

Risk factors:

- A. Modifiable:
 1. Prone
 2. Smoke
 3. Bottle
 4. Hot
 5. Bedding
 6. Cosleeping
 7. PNC
 8. URI
- B. Non-modifiable
 1. Maternal age
 2. Maternal education
 3. Race

4. Prematurity
5. Birth weight
6. Class
7. Season

Discusses three brainstem hypotheses:

- I. Sleep-wake regulation. Lack adequate arousal response to hypoxia and/or CO₂.
- II. Temperature regulation. Allow themselves to get too hot. Possible dysautonomia.
- III. Cardiorespiratory regulation. Poor chemoreceptors for hypoxia and CO₂.

Each of these malfunctions could lead to prolonged apne-brady. They intersect with the other risk factors. (Multifactorial hypothesis: see Hannah Kinney).

In the Mississippi delta, which is mostly very poor rural Blacks whose SIDS rate has failed to decline with the rest of the US and is twice the national rate, these doctors have gotten a grant to try to modify three modifiable risk factors: prone, smoking, and bottlefeeding. Interesting is their justification for why they chose these three factors and not others.

As to prone, these are 2X to 3X more likely to die of SIDS, so the statistical effect can be expected to be substantial. Cite studies for the fact that in inner-city Black populations, prone sleeping is much more common, and their own unpublished data showing that in the Delta, only 9% of mothers knew that prone was a SIDS risk factor. Studies showing that educational programs can substantially reduce the practice of prone in inner-city Black populations.

As to smoking, it is “one of the most significant predictors of SIDS” and also of infant respiratory tract infections including otitis media (!) (citing multiple refs) A dose-response relationship has been shown. In the Delta, 72% of mothers smoke regularly, compared to 19% for the state as a whole. Why does smoking potentiate SIDS? Three hypotheses:

1. Damages the cardiorespiratory center in the developing brain
2. Potentiates low levels of bacterial toxins
3. Impairs catabolism of catecholamines in the brain

As to bottlefeeding, these are 2X to 3X more likely to die of SIDS. Discusses hypotheses as to why: immunity, PUFA's, and nocturnal tactile stimulation/ arousal. Minorities do less breastfeeding. In rural Mississippi 19% for Blacks versus 44% for Whites. “Other studies show that social support systems, such as those at the workplace and in the hospitals, are necessary for a successful breast-feeding program.”

Numbers of SIDS deaths in California, 1990-2000

Numbers of SIDS deaths in Los Angeles County, 1990-2000

Grant Neie, personal communication May 9, 2002.

	1990	1991	1992	1993	1994	1995	1996	1997	1998	1999	2000
CA	717	725	568	545	486	397	311	329	259	237	222
LAC	252	208	177	176	120	107	82	87	69	46	34

SIDS risk factors as of 2001, as per Dr Ramanathan. Cardiorespiratory events recorded on home monitors: comparison of healthy infants with those at increased risk for SIDS. Ramanathan R et al. JAMA 2001 May 2; 285(17): 2199-2207.

Prone
Soft surface
Overheating
Maternal smoking during pregn
Maternal smoking with cosl
Late or no PNC
Young maternal age
Male

Prematurity
LBW
Black
American Indian

This article showing that in the CHIME study of 1079 infants (one group of 306 healthy term infants and six groups totalling 773 high-risk infants (152 with ALTE, 178 SIDS siblings, 443 preemies), there was no difference in the number of “conventional” apne-bradies or “extreme” apne-bradies. Accordingly, Dr Ramanathan does not prescribe home monitors for his patients. Ramanathan, R, personal communication, December 12, 2002.

Prevalence of retinal hemorrhages and child abuse in children who present with an apparent life-threatening event. Pitetti RD, Maffei F, Chang K, Hickey R, Berger R, Pierce MC. Pediatrics 2002 Sep; 110(3): 557-562. Prospective study of 128 ER admissions under 24 months with ALTE, covering a two-year study period. Found child abuse in 3 pts and RH in 1. Found that 117 had a family history of SIDS. Interestingly, the authors do no comment on the epidemiologic implications of this background. See under “RH -- In General.”

[Nighttime child care centers] Inadequate SIDS risk factor knowledge. Moon RY, Weese-Mayer DE, Silvestri JM. Pediatrics 2003 Apr; 111: i4: p. 795(5). (have) Found that these nighttime child care centers used by mothers who work night shifts have inadequate practices.

Racial disparity and modifiable risk factors among infants dying suddenly and unexpectedly. Unger B, Kemp JS, Wilkins D et al. Pediatrics 2003; 111: e127-e131. From the review by Dr Reece in the *Quarterly* for July 03: This population-based study in St Louis 1994-1997 included not only SIDS but also UNDET and Accidental Suffocations. Included bedsharing and sleep surface data. **FOUND: an increase in the racial disparity of all three types of deaths since Back to Sleep.** Also, the ratio of prone to nonprone deaths did not differ between races, so the disparity cannot be attributed to sleep position or failure of back-to-sleep to reach the black community. The authors opine that high levels of **high-risk bedsharing** in the AA community could explain a great deal of the disparity in all three types of deaths. Bedsharing was 67% in AA versus 35% in non-AA. Deaths on “makeshift bedding” were 12% of AA, 0% of non-AA. All three types of death were more common in AA:

<u>Type</u>	<u>AA</u>	<u>non-AA</u>	<u>Multiple</u>
SIDS	58	32	1.8
Acc suffoc	13	3	4.3
UNDET			

Death rates per 100/000 LB:

<u>Type</u>	<u>AA</u>	<u>non-AA</u>	<u>Multiple</u>
SIDS	2.08	0.65	3.2
Acc suffoc	0.47	0.06	7.8
UNDET	0.36	0.06	6.0

Multiple SIDS -- coincidence or beyond coincidence? Hill R. Paediatr Perin Epid 2004; 18: 320-326. Uses probability theory on the CESDI database of 500,000 live births in England to show that having two SIDS deaths is more probable than having two homicides. Therefore questions the Meadow rule that “two is suspicious.”

SIDS and unclassified sudden infant deaths. Krous HF, Beckwith JB, Byard RW et al., Pediatrics 2004 Jul; 114(1): 234-238. The new San Diego stratified definition of SIDS, intended to capture the findings that

lead to Undet diagnoses and rope them within the definition of SIDS. See above under “SIDS versus suffocation” for details.

SIDS Deaths in California, 2004 California Department of Health Services official print

This only became available in 2006, courtesy of the State Health Department.

SIDS rate overall for the state: 31.4 per 100,000 LB

SIDS rates by race per 100,000LB: Hisp 23.6, White 38.3, AA 83.7.

171 SIDS in California in 2004

SIDS was the second leading cause of postneonatal death in infants.

Males 2/3, females 1/3

No seasonal pattern observed in 2004 (a new finding)

Age pattern is distinctive: 81% died before the age of 5 months.

ICD-10 was introduced in 1999. Under it, SIDS death rates cannot be compared to earlier years because the definition changed.

Notwithstanding the above, California’s SIDS rate continued to decline through 2004. “But some of the decrease may be due to counties using different definitions of SIDS.”

Sudden Infant Death Syndrome risk factors with regards to sleep position, sleep surface, and co-sleeping.

Alexander RT, Radisch D. JFS 2005 Jan; 50(1): 147-151. See below under “cosleeping.”

Stable prevalence but changing risk factors for SIDS in child care settings in 2001. Moon RY, Sprague BM, Patel KM. Pediatrics 2005; 116: 972-977. A followon to the same authors’ earlier study (1995-1997) of the same thing. In that study, they found that 20% of SIDS occurred in child care settings. This time it was 16%. They note that with 55% of mothers in the workforce, half of all infants are in day care. Obviously, these day-care SIDS cases occur during the workday, giving them a different time of day from regular SIDS cases. This time, there was no demographic difference from the regular SIDS cases.

How reliable are SIDS rates? Sheehan KM, Devaney DM, Matthews T. Arch Dis Chil 2005; 90: 1082-1083. According to Dr Krous’s review in the Spring 2006 edition of the *Quarterly*, this study from Ireland showed that using more recent forensic criteria for Undet to rediagnose old cases originally diagnosed as SIDS increased the Undet rate from .03 to .64, and reduced the SIDS rate from .71 to .08. Dr Krous comments that obviously, any time you change the diagnostic criteria for any disease, you will change the incidence. For purposes of this article, the Undet criteria were seven criteria basically amounting to “worrying parental practices or additional pathology findings.” Dr Krous refers to the new San Diego stratified definition (Pediatrics July 2004) and Fleming’s version of a new stratified definition, as trying to take account of all these factors.

Widening social inequalities in risk for SIDS. Pickett KE, Luo Y, Lauderdale DS. Am J Pub H 2005; 95: 1976-1980. As reviewed by Dr Wendy Lane in the Spring 2006 issue of the *Quarterly*, found that Back to Sleep has increased disparity as to maternal education across all races. As Dr Lane writes, “While the risk for SIDS declined for all levels of maternal education, it declined the most for highly educated women.”

AAP Policy Statement: The changing concept of sudden infant death syndrome: diagnostic coding shifts, controversies regarding the sleeping environment, and new variables to consider in reducing risk. Pediatrics 2005 Nov; 116(5): 1245-1255. From the abstract: “There has been a major decrease in the incidence of SIDS since [back-to-sleep, 1992]... Some of the decrease...may be a result of coding shifts to other causes of unexpected infant deaths.” Issues discussed:

- Side sleeping is no longer recognized by the AAP as safe.
- Soft bedding and soft objects in the infant’s sleeping environment
- Hazards of bedsharing “under certain conditions”
- Risk reduction from roomsharing
- Risk reduction from pacifiers
- Back-to-sleep education of neonatal nurses etc.
- Reducing positional plagiocephaly

Mentions racial disparity in prone positioning -- 11% in white vs. 21% in black. Correlates with the racial disparity in SIDS. Decrease in the seasonality of SIDS. As to bedding, says the soft bedding or "burrowing" concept goes back to Abramson, 1944. A case control study in the US (Hauck et al., 2003) has confirmed the strong association of SIDS and soft bedding -- adjusted odds ratio of 21. As to cosleeping, says this is "a highly controversial topic." It facilitates bonding and nursing, but it can be hazardous "under certain conditions." (Citing Scheers et al., 2003, Unger et al., 2003, Kemp et al., 2000, Drago et al., 1999). And what conditions would those be? One is a smoking mother. Some of the studies have found a correlation only among mothers who smoked. (Blair et al., 1999, Scragg et al., 1993). But the European study and the Scottish study found that it didn't matter if the mother smoked. Another condition is very young infant -- up to 8 weeks in the European study and up to 11 weeks in the Scottish study. Another condition is multiple cosleeping. (Hauck et al. 2003). Another is alcohol or overtired. Another is longer duration of bedsharing during the night. Another is couch. There is growing ev that roomsharing reduces risk. Pacifiers. Secondary caregivers: 2/3 of US infants are in nonparental child care. In the US, 20% of SIDS deaths occurred in nonparental care. Many of these were prone deaths, especially when the child is unused to sleeping prone. "This is particularly concerning, because unaccustomed prone sleep increases the risk of SIDS by as much as 18-fold." (Citing Mitchell et al., 1999, L'hoir et al., 1998). As to home monitors and ALTE,

The etiology of pediatric out-of-hospital cardiac arrest by coroner's diagnosis. Ong, Marcus E.H., Siell, Ian, Osmond, Martin H, Nesbitt, Lisa, Gerein, Rick, Campbell, Starla, McLellan, Barry. Resuscitation 2006; 68: 335-342. The authors note that population-based studies of the etiology of pediatric "out-of-hospital cardiac arrest" have been previously performed by Sirbaugh et al. (1999) and Young et al. (2004). But these studies were based on hospital and paramedics' records. The authors note that the autopsy, not the hospital record, is considered the "gold standard" of etiology. Their study is autopsy-based. They used the records of the provincial coroners' offices in Ontario over the eleven-year period 1992 to 2002 for this prospective cohort study. They acquired their cases from ambulance call records referring to persons aged 0 to 18 years who were documented by the emergency medical services as being pulseless, apneic, and requiring chest compressions in the field. There were 474 such young people. Of these, 465 died, for a mortality rate of 98%. Of these 465 fatal outcomes, 439 (95%) matched to provincial coroners' records. The autopsy rate, apparently of the 465 deaths, (not clear) was 84%. This implies that the authors attained the "gold standard" of etiology in about 82% of their 474 out-of-hospital cardiac arrests.

The breakdown of the 474 out-of-hospital cardiac arrests by age showed that 43% of them occurred in infants under one year. The annual incidence rates by age group were as follows:

age 1-4	175 per million population
age 5-14	33 per million population
age 15-18	62 per million population

The preponderance of infants under one year, plus the above age-group distribution, shows the overwhelming preponderance of infants and toddlers in the cardiac-arrest population. That same preponderance is evidenced by the authors' finding that SIDS alone constituted the etiology of 14% of all the cardiac arrests in all age groups combined.

The objective of the study was to obtain an autopsy-based catalogue of the etiology of pediatric cardiac arrests, and also to find out how many of these cardiac arrests were due to preventable forms of injury. The coroners' records broke down the fatal outcomes by manner of death (natural, accidental, suicidal, homicidal, and undetermined). That broke out this way:

natural	47%
accidental	31%
suicidal	7%
homicidal	3%
undetermined	11%

The causes of death are given by the authors in a detailed two-page table. This shows that SIDS was the commonest natural cause (30% of all naturals), drowning was the commonest accidental cause (27% of all accidentals). There were 15 homicides -- 6 penetrating injury, 5 blunt force injury, 1 neglect, 1 strangulation, and 1 by fire. As to Undetermined manner of death, there were 49. Overall, the authors found that 52% of the cardiac arrests were potentially preventable.

The authors state that their findings will be useful in planning preventive strategies for the preventable forms of pediatric sudden death -- homicide, suicide, and accident. But to this reviewer, it is not clear how this information, which largely duplicates what is already available, will be used. Maybe to persuade government and the public to undertake targeted prevention programs aimed at drownings and suicides? The authors remain studiously vague on exactly how this information is to be used.

Multiple serotonergic brainstem abnormalities in sudden infant death syndrome. (Preliminary Communication). Paterson DS, Trachtenberg FL, Thompson EG, Belliveau RA, Beggs AH, Darnall R, Chadwick AE, Krous HF, Kinney HC. JAMA 2006 Nov; 296(17): 2124-2132. See above under "SIDS biology." The authors did genotyping of their subjects for polymorphisms in the promoter region (5HTTLPR) and in intron 2 of SLC6A4, the gene for the 5-HT transporter.

LA County Presumptive SIDS referral report as of November 7, 2006. Prepared by LA County Department of Public Health: Maternal, Child, and Adol Health: Fetal & Infant Health Programs: SIDS Program.

Total presumptive SIDS referrals	53
Final diagnosis (Coroner)	
Undet	11
SIDS	4
Natural not SIDS	3
Pending	35
Cosleeping	
yes	27
no	20

Gives a chart of positions: position put to sleep versus position found.

Cases by race:

White	5	(9%)
Black	17	(32%)
Hisp	29	(55%)
Other	2	(4%)

Gives a bar chart of manner of death dxd by the LA County Coroner by year, 2000 to 2004: Shows UNDET rising from 28% to 48%, NAT declining from 54% to 31%, HOMI remaining steady at 7%.

Do risk factors differ between explained sudden unexpected death in infancy (SUDI) and sudden infant death syndrome? Vennemann M, Bajanowski T, Butterfass-Bahloul T, Sauerland C, Jorch G, Brinkmann B, Mitchell EA. Arch Dis Chil (UK) 2007 Jan; 92: 133-136. German researchers did a 3-year population-based case-control study 1998-2001. Found that the only risk factors that differed between SUDI and SIDS were prone sleeping and lack of breastfeeding in the first two weeks of life. These were risk factors for SIDS but not for SUDI. Maternal smoking and lower class status were risk factors for both syndromes, and offer avenues for targeted prevention measures.

TWIN SIDS

From Reece & Ludwig, *Child Abuse: Medical Diagnosis and Treatment, Second ed., 2001*: "In most studies, SIDS is seen to be 2.5 times more common in multiple births ...than in singleton births. This is probably due to the lower mean birth weight of twins... Sudden unexpected death in both twins has been

the subject of numerous articles... The authors [of Malloy & Freeman's 1999 study of 767 sets of twins] concluded that, independent of birth weight, twins do not appear to be at greater risk for SIDS compared with singleton births." p. 520.

Beal, Med J Aust 1973; 1: 1146-1148

Cooke, BMJ 1964; 2: 1549

Simultaneous sudden infant death syndrome in twins. Smialek JE. Pediatrics 1986 Jun; 77(6): 816-821. Case report of nine cases of simultaneous deaths in twin infants personally autopsied by the author.

Sudden infant death syndrome in twins. Beal S. Pediatrics 1989 Dec; 84(6): 1038-1044. Literature review. See also 1973.

SIDS among twins. Malloy MH, Freeman DH Jr. Arch Pediatr Adol Med 1999 Jul; 153(7): 736-740. In the five year period 1987-1991 the twin-sids rate was 2.5/1000 LB. The singleton sids rate was 1.2/1000. So the crude relative risk was 2:1. Twin sids was associated with Black race, uneducated mother, and LBW. They used a mainframe to find the co-twins of twins who died of SIDS. 23,464 singleton SIDS and 1,056 twin SIDS. Among the 767 twin pregnancies in which one or both members died of SIDS, there were only 7 in which both twins died of SIDS. They calculated relative risk on a crude basis and on an adjusted basis, adjusted for birth weight, race, and class. The adjusted relative SIDS risk for any one twin compared to a singleton was 1.13. The adjusted relative risk for the second twin was 8, but the 90% confidence interval for that was 1.18 – 56.67. (The interval is so wide because even with a huge study the absolute numbers are small.) They conclude that that a twin is not at greater risk for SIDS than a singleton infant is. See the discussion in Reece & Ludwig, 2001, above.

The epidemiology of SIDS. Platt MJ, Pharoah PO. (Liverpool) Arch Dis Chil 2003 Jan; 88(1): 27-29. A birth certificate/death certificate study. States as background that "Twins compared to singletons are at increased risk of SIDS." [This is true, see above. But it is true because twins have lower birth weights. – JKR] The crude relative risk of SIDS in twins was twice that in singletons, but the birthweight-specific risk was greater in singletons, except in those of birthweight over 3,000 grams. (SIDS risk increased with decreasing birthweight for both twins and singletons.) Lack of a sex correlation suggests that zygosity is not a factor.

AAP National Conference and Exhibition, New Orleans, 2003. Paper by Richard J Cartie, MD of UT, examined the San Antonio Medical Examiners records for 1988-2001 and found that the absolute number of infant deaths stayed the same, while the "SIDS" component decreased from 82% to 36% and the UNDET component increased from 18% to 64%. Concluded that the actual incidence of SIDS may not be decreasing at all, just changing its name to UNDET.

Simultaneous sudden infant death syndrome. Balci Y, Tok M, Kocaturk K, Yenilmez C, Yorulmaz C. J Clin Foren Med 2006. From Dr Byard's review in the Quarterly for summer 2006, this family had a strong history of twinning and a strong family history of infant death, yet no genetic testing was done. Therefore, Dr Byard says it would have been better to diagnose the COD as "unclassified sudden infant death" (USID).

COSLEEPING

My mnemonic for remembering the high-risk forms of cosleeping. (August 2005, updated May 2006):
"LUVSMI"

Large adult

Unsafe sleep surface
 Very small infant
 Smoking
 Multiple cosleeping
 Intoxication

Does bed-sharing affect the risk of SIDS? AAP Policy statement RE 9725. Pediatrics 1997 Aug; 100(2): 272. By mother is ok if no soft bedding. By others may incr risk of SIDS. Unclear.

Infant arousals during mother-infant bed sharing: implications for infant sleep and sudden infant death syndrome. Mosko, Richard, McKenna. Pediatrics 1997 Nov; 100(5): 841-849. This very interesting sleep study (similar work was done at CHOC) showing how the infant manipulates his mother's sleep pattern to satisfy his own needs. Discussed by Keens, 2003, below.

Reducing the risk of SIDS: a review of the scientific literature. Henderson-Smart DJ, Ponsonby AL, Murphy E. J Paediatr Child Health 1998 Jun; 34(3): 213-219. Australia. Covering baby's head is strongly related to SIDS. Bedsharing among smokers.

Risk factors in cosleeping. Jem Berry, MD. Jem.berry@bristol.ac.uk unpublished observations, 1999. Most cosleeping may be safe, but there are circumstances when it may be less safe. These include:

Very large adult
 Very small baby
 Age under 12 weeks
 Newborn
 Alcohol use
 Drug use
 Tobacco use
 Sofas = 40x increased risk

[CESDI Study] Blair PS et al. BMJ 1999; 319: 457-462. The following taken from Dr Keens' discussion (Keens, 2003): Most of the SIDS-cosleeping deaths were on sofas. Bedsharing did not increase the SIDS risk for infants over 4 months or parents who did not smoke. See also

Review of hazards associated with children placed in adult beds. Suad Nakamura, Wind M, Danello MA. Arch Ped Adol Med 1999 Oct; 153(10): 1019-1023. Retrospective review of CPSC data from 1990-1997. Eight years of data collected by the CPSC on deaths of children younger than 2 yrs in adult beds, daybeds, and waterbeds. Had reports of 515 deaths. Apparently not broken down as to cosleeping / not cosleeping

551 deaths
 121 overlaying
 77% were under 3 months, with the overwhelming majority
 under 1 month
 11% were on water beds
 394 entrapment

No data collected on alcohol use. Conclusion: adult beds are hazardous with or without cosleeping. "Placing children younger than 2 years to sleep in adult beds exposes them to potentially fatal hazards that are generally not recognized by the parent or caregiver."

[Accidental causes of 2,178 infant deaths.] Drago DA and Dannenberg AL. Pediatrics 1999; 103(5): e59. Discussed by Keens, 2003, from which I have taken the following data:

Wedging	879
Facial occlusion	512
Overlaying	180
Entrapment with suspension	145

Hanging	111
Entanglement	91
Compression (collapsed crib)	55
Positional asphyxia (hamper, hammock)	33
Entrapment without suspension	30
Unknown	142

Dr Keens makes the point that only 24% were associated with bedsharing, while the other 76% were sleeping alone. So according to these data, it's safer to cosleep than to sleep alone.

Babies in adult beds, by Suad Nakamura, PhD, Directorate for Health Sciences, CPSC. Consumer Product Safety Review, Winter 2000, p.5. (have) "Placing babies to sleep in adult beds puts infants at risk of suffocation or strangulation, according to a CPSC study in the Arch Ped Adol Med. (See below) The study revealed an average 64 deaths per year to babies under the age of 2 years placed to sleep in adult beds, including waterbeds and daybeds. Medical authorities, such as the AAP and the SIDS alliance, also have concerns about placing babies to sleep in adult beds. AAP believes that bedsharing or co-sleeping may be hazardous under certain conditions....(no ref) The SIDS Alliance recently issued a statement that stated, '...bedsharing...can, under a number of conditions, actually be hazardous.' A CPSC review of incident data from Jan 1990 to Dec 1997 linked adult beds to at least 515 baby deaths. Analysis of the deaths revealed four major hazard patterns, These included:

- n suffocation associated with [cosleeping]
- n suffocation [due to wedging] between the mattress and another object
- n suffocation due to [face down on waterbed]
- n strangulation in rails or openings on beds that [allow a baby's body but not head]"

CPSC's study is the first to quantify the number of fatalities resulting from the practice of co-sleeping with babies. But it actually deals with adult beds, with or without cosleeping. Of the 515 deaths, 121 were [due to overlaying.] More than ¾ of these occurred to infants younger than 3 months. The other 394 deaths resulted from suffocation or [strangulation/entrapment.] ... CPSC is working with the bedrail industry...

Bed sharing and infant mortality: guilt by association? J Hum Lact 2000 Aug; 16(3): 189-91. The breastfeeding community protests.

Bed sharing is not a "consumer product." McAfee T. Arch Ped Adol Med 2000 May; 154(5): 530-531.

SIDS and cosleeping. Rosenberg KD. Arch Ped Adol Med 2000 May; 154(5): 529-30. Cited by Dr Ramanathan as a review of four large studies showing no increase of SIDS risk from cosleeping unless the mother smokes. The four studies were New Z 1993; CA 1995; England 1996; New Z Maori 1997.

Unsafe sleep practices and an analysis of **bedsharing** among infants dying suddenly and unexpectedly: results of a four-year, population-based, death scene investigation study of sudden infant death syndrome and related deaths. Kemp JS, Unger B, Wilkins D, Psara RM, Ledbetter TL, Graham MA, Case M, Thach BT. Pediatrics electronic pages 2000 Sep; 106(3): e41. They retrospectively reviewed the death-scene data from St. Louis area in 119 infant deaths. 88 SIDS, 16 accidental suff, 15 undet. In 75% of the cases, the infant was sleeping on a surface not designed for infants. Cosleeping in 47%. Head covered by bedding in 30%. Only 8% of the deaths (10 infants) involved an infant sleeping alone on its back without bedding over the head and face.

Position:

	<u>Prone</u>	<u>Side</u>	<u>Supine</u>	<u>Unk</u>
Total	66	10	31	12
SIDS	55	6	20	

Surface:

	<u>Crib</u>	<u>Pen</u>	<u>Bed</u>	<u>Sofa</u>
Total	29	6	50	19
SIDS				14

Seven cases were entrapped by a bedmate. Preventable deaths: 84% were potentially preventable. Not counting three deaths that were made UNDET because of possible but unprovable foul play.

“the impact of bedsharing remains controversial. (discussion) In the three case-control studies, the risk (apparently of both SIDS and nonSIDS) is lessened when smoking is eliminated. But the Chicago Infant Mortality Study suggests an effect of bedsharing independent of smoking.

Comment by JKR: Unsafe sleep arrangements and cosleeping may be covariables with lower class status and chaotic living conditions.

This article is discussed in Keens, 2003, below.

Changing concepts of SIDS: implications for infant sleeping environment and sleep position. AAP Policy Statement (RE9946). Pediatrics 2000 Mar; 105(3): 650-656. (have) See above under “SIDS Biology.”

SIDS in South Australia, 1968-1997. Part 3: Is bed sharing safe for infants? Beal SM, Byard RW. Reviewed all SIDS for 17 year period. As a result, they re-did the “probable” causes of death for these, and found that ACCI was the most likely manner of death for the following percentages of infants in the following sleeping circumstances:

Designated infant containers	5%
Sharing a bed or couch	24%
Placed alone on a bed or couch	72%

AAP Policy Statement February 2001: Distinguishing SIDS from Child Abuse Fatalities. (RE 0036). AAP Committee on CAN. Pediatrics 2001 Feb; 107(2): 437-441. <http://www.aap.org/policy/re0036.html> (have) This latest attempt by Dr. Reece and the AAP. This article qualifiedly limits the diagnosis of SIDS to the first six months of life: “Approximately 90% of SIDS deaths occur before the age of 6 months. (citing Peterson, 1988). SIDS is suspected when a previously healthy infant, usually younger than 6 months, is found dead in bed...” Appears to make the diagnostic criteria of SIDS more conservative: “Only on completion of a thorough and negative case investigation (including performance of a complete autopsy, examination of the death scene, and review of the clinical history) should a definitive diagnosis of SIDS be assigned as the cause of death.” “Cases that are autopsied and carefully investigated but reveal **substantial and reasonable uncertainty** regarding the cause or manner of death may be designated as undetermined.” A young infant’s death should be ruled as attributable to SIDS [only] when all of the following are true:

7. [A]utopsy findings are compatible with SIDS.
8. There is no gross or microscopic evidence of trauma or significant disease process.
9. There is no evidence of trauma on skeletal survey.
10. Other causes of death are adequately ruled out. (giving a list of examples)
11. There is no evidence of current alcohol, drug, or toxic exposure, and
12. Thorough death scene investigation and review of the clinical history are negative.”
(Negative for what?)

Later, says that “Pathologists establish the diagnosis of SIDS by exclusion when they are unable to identify other specific causes for a child’s death.” And the death “remains completely unexplained.” (citing DiMaio, *Forensic Pathology*, 1989.) “Cases that are autopsied and carefully investigated but reveal substantial and reasonable uncertainty regarding the cause or manner of death may be designated as undetermined. Examples of undetermined cases include suspected (but unproven) infant death attributable to infection, metabolic disease, accidental asphyxiation, or child abuse.”

SIDS, bedsharing, parental weight, and age at death. Carroll-Pankhurst C, Mortimer EA Jr. Pediatrics 2001 Mar; 107(3): 530-536. Went through the Coroner’s files in Cleveland for 1992-1996, found 84 SIDS cases, compared age, maternal weight, other risk factors, and bedsharing. 30 bedsharing, 54 non-bedsharing. Age for bedsharing was 9 weeks, for non-bedsharing was 12 weeks. Mean pregravid weight

of bedsharing mothers of deceased infants was 84 kg vs 67 kg for nonbedsharing mothers of deceased infants (no P value given in the abstract).

Category of SIDS	Mean age at death
Nonbedsharing infants	12 wks
Bedsharing w thin mother	10 wks
Bedsharing w fat mother	7 wks

Among an urban pop at high risk for SIDS, bedsharing was strongly associated with a younger age at death, regardless of any other factors. In this subpop, bedsharing was an independent risk factor for sudden death, particularly if the parent was large. [But with such small numbers, these figures cannot possibly be statistically significant. –JKR Also, does not consider the UNDETS in the same pop.] See also Betty Spivack's commentary in Child Abuse Quarterly Jul 2001.

Comment on Carroll-Pankhurst article by Betty Spivack, March 12, 2001. Until we have reliable data on the frequency of cosleeping at various stages of infancy in various socioeconomic groups and stratified for other SIDS risk factors, we cannot make a decision as to whether the practice is an additional risk or protective. Until data of this sort comes out, we won't get beyond the well-demonstrated fact that cosleeping occurs in 20-40% of infants dying of SIDS. The significance of that fact will remain elusive. Additional risk: Carroll-Pankhurst art and Ped 1992; 90: 905-908, BMJ 1990; 301: 85-89, BMJ 1999; 319: 1457-1460. Protective: BMJ 1995; 311: 1269-1272, J Behav Med 1993; 16: 589-610, Sleep 1993; 16: 263-282.

Mother-infant cosleeping: toward a new scientific beginning. McKenna JJ and Mosko S. In: Byard RW and Krous HF, eds., *Sudden Infant Death Syndrome: Problems, Progress & Possibilities*. New York: OUP, 2001, pp. 258-274. A sophisticated scholarly discussion of cosleeping including nursing and infant physiologic responses. Nursing cosleeping is very common and nonpathologic. Result: there is cosleeping and cosleeping. It depends on who, the surface (sofa, etc.) the family environment. "...must recognize that under specific conditions, especially among the urban underclass where most of the bed-sharing deaths occur, specific types of cosleeping can be dangerous... Hence, there are dangerous conditions -- not necessarily dangerous parental bodies." No recommendation for or against cosleeping as such is appropriate at this time.

Association between sudden infant death syndrome and prone sleep position, bed sharing, and sleeping outside an infant crib in Alaska. Gessner BD et al. Pediatrics 2001 Oct; 108(4): 923-927. Reviewed all SIDS 1992-1997: 130 cases, of which 115 had sleep location known. Of the 115, 98% were one or more of the following:

Prone
Not in a crib
Cosleeping

Of 40 deceased infants who coslept, all were either prone or with a drug-intoxicated parent or on a water bed.

Sleep position and bed-sharing in SIDS deaths: an examination of autopsy findings. Thogmartin JR, Siebert CF, Pellan WA. (Palm Beach County ME's Office) J Pediatr 2001- Feb; 138(2): 212-217. Significant autopsy findings that could explain the death were found in 47% of the supine solitary infants and only 16% of the prone solitary infants. Among bedsharing infants it was 18% versus 9%. So supine deaths (either bedsharing or not) more often had findings. (1) non-prone deaths were more frequently explained by illness; (2) bedsharing increased the proportion of illness-negative deaths regardless of sleep position.

Cosleeping and sudden unexpected death in infancy. Person TLA, Lavezzi WA, Wolf BC. Arch Path Lab Med 2002; 126: 343-345. Retrospective review of the investigative files in 58 SUDS cases.

58 SUDS

27 cosleepings

11 cosleepings had been dx'd as SIDS

7 cosleepings had parental intoxication

31 not cosleeping

See Henry Krous's review in *Child Abuse Quarterly* for July 2002. Dr Krous summarizes that the authors conclude that cosleeping is potentially dangerous. Further, that some cosleeping deaths are either overlayings or "lethal rebreathing in compressible bedding materials."

Specific dangers associated with infants sleeping on sofas. Byard RW, Beal S, Blackbourne B, Nadeau JM, Krous HF. *J Paediatr Child Health* 2001; 37; 476-478. Found ten cases of SUDS on sofas with complete scene investigations from 1991 to 1998. Four were cosleeping, six were not.

4	overlayings/ wedgings involving a cosleeping adult
2	spontaneous wedgings

Conclusion: sofas are unsafe both for sleeping and cosleeping.

Cosleeping in young Korean children. Yang C-K and Hahn H-M. *Dev Behav Ped* 2002; 23: 151-157. From Dr Krous's review in *Quarterly*: 88% coslept: 40% bedsharing and 40% roomsharing. All of the children were over six months of age during the time of sleeping studied.

Trends in infant bed sharing in the United States, 1993-2000: the national infant sleep position study. Willinger M, Ko CW, Hoffman HJ, Kessler RC, Corwin MJ. *Arch Peds* 2003 Jan; 157(1): 43-49. From the NIH/NICHD press release: The National Infant Sleep Position Study (NISP), the first study of how many US infants cosleep with an adult, showed that the proportion of infants sharing an adult bed at night increased from 5% to 12% between 1993 and 2000. Nearly 50 percent of infants in the study spent at least some time sleeping on an adult bed at night, and 20% do so half the time or more. AA infants were four times more likely to bedshare as white, and Asian/Other infants three times as likely. Both studies suggest that bedsharing is widespread and strongly infl by cultural factors. These were two studies: a telephone survey of 8,400 families throughout the 48 states, and an interview survey of 369 AA mothers in Washington DC. Found that mother under 18 were 2X as likely, low income 1.5X more likely. The second study found that 48% of AA infants bedshared, more likely to bedshare if the mothers were single or if they had moved at least once since the birth. Goes on to briefly discuss bedsharing: "a controversial and poorly understood practice. "Studies that directly assessed the risk of SIDS associated with bedsharing have not found bedsharing to be protective. Some studies have shown an increased risk for SIDS as w bedsharing under the fol dconditions: maternal cigarette smoking, recent maternal alcohol consumption, infant covered by a duvet (quilt or comforter), and parental fatigue.

Racial disparity and modifiable risk factors among infants dying suddenly and unexpectedly. Unger B, Kemp JS, Wilkins D, Psara R, Ledbetter T, Graham M, Case M, Thach BT. (St. Louis). *Pediatrics* 2003 Feb; 111(2): e127-131. Black and nonBlack infants did not differ in sleep position, but they did differ in cosleeping, particularly on unsafe surfaces. Suggests a targeted public information campaign on bedsharing in AA. Studied the death scenes and autopsy results of 119 infants in St Louis, 81 African-American and 38 non-African-American (both SIDS and nonSIDS). "The objective...was to compare death rates attributable to SIDS and related causes of death (accidental suffocation and Undetermined) in African-American and non-African-American infants and the prevalence of unsafe sleep practices at the time of death." Sleep surfaces other than those specifically designed for infants were termed "nonstandard" sleep surfaces.

Found: The rates of all three types of infant death (SIDS, AS, and UNDET) were increased for AA infants:

Type	AA rate/1000 LB	non-AA rate/1000 LB
------	-----------------	---------------------

SIDS	2.08	.65
AS	.47	.06
UNDET	.36	.06

Sleepsurface-associated deaths were nearly twice as common in AA:

Bedsharing	67% of the deaths`	35% of the deaths
Nonstandard SS	79%	46%

Found position on back or side was (+) associated with bedsharing:

Bedsharing found on back or side	49%
Not bedsharing found on back or side	20%

Found position was NOT associated with race

NonProne	43%	38%
----------	-----	-----

And neither was unsafe cosleeping (couch, sofa, or all-night bedsharing), but unsafe cosleeping was present in all or many deaths in all races.

Infant-parent bedsharing in an inner-city population. Brenner RA, Simons-Morton BG, Bhaskar B, Revenis M, Daas A, Clemens JD. (NIH) Arch Ped Adol Med 2003 Jan; 157(1): 33-39. Bedsharing is controversial and poorly understood. "Proponents cite potential advantages such as increases in bonding and facilitation of breastfeeding, whereas opponents cite potential increases in risks of suffocation and SIDS, particularly among mothers who smoke." We interviewed 369 inner-city mothers at birth and again at 3-7 months, and found that 48% of their infants usually slept in a bed with a parent or other adult. Statistically associated things were single marital status of the mother and one or more moves. Breastfeeding and crowding were not statistically associated with bedsharing. Conclusions: Bedsharing was common and stable.

Bedsharing and the risk of sudden unexpected infant death. Thomas G. Keens, MD. (PowerPoint Presentation, to the 16th Annual Conference of the Association of SIDS and Infant Mortality Programs, Washington, DC, March 14th, 2003. Printed text kindly provided by Dr. Keens. Includes discussion of the question, "Is bedsharing protective?" Under this head, Dr Keens makes the following points:

- Considerable maternal-infant behavioral interaction may affect development.
- More frequent breastfeeding is promoted.
- Maternal breathing may entrain the infant's breathing, reducing apnea
- Exhaled CO₂ from the mother may stimulate the infant's breathing.

Cites sleep laboratory data by Richard et al., J Appl Physiol 1998; 84: 1374-1380. But concludes that there is no proof of any decrease in apnea, or any stimulation of breathing.

Are there any long-term benefits to the infant from bedsharing? Yes: higher self-esteem in young adulthood (citing Lewis RH and Landa LH, Arch Sex Behav 1988; 17: 349-363, and Crawford M., Ethos 1994; 22: 42-82, and Forbes JF et al., Military Medicine 1992; 157: 196-200.)

Do we know the cause of infant deaths associated with bedsharing? No. The studies have problems. In any particular case it is difficult to determine (a) whether bedsharing occurred or not, (b) whether the death was a SIDS or an overlaying, (c) whether death occurred before or after overlaying.

How big is the problem? Poor epidemiologic data is a hindrance to knowing this. Data on sleeping arrangements were missing from 31% of the California Department of Health Services case reports. These reports (1992-1996) found that out of 2,399 SUDS cases,

- 8.7% were found in bed with a parent
- 25.6% were found alone in an adult bed
- 7.6% were found on a sofa or couch with or without an adult

So the crude death rates per 1,000 LB under these circumstances were:

All SUDS	.84
SIDS	.81
Alone in adult bed	.22
Bedsharing	.07
Sofa or couch	.06

Does bedsharing increase the risk of SIDS or other SUDS? Some bedsharing is safe, some is not; it depends on the circumstances. Specific bedsharing risks can be identified. (Citing McKenna & Mosko in Byard & Krous, eds., *SIDS*, 2001.)

Maternal: obesity, smoking, drug or alcohol use, prone infant, overwrapping, prematurity.

Chaotic household: young, unsupported, little prenatal care, little education

Dangerous beds: soft mattress, lots of pillows, duvets, covering the infant's head.

Reviews the data by Drago & Dannenberg, 1999 (see above) to the effect that of 2,718 accidental infant deaths, only 24% were associated with bedsharing, while 76% were sleeping alone. Therefore one could argue that cosleeping is safer than sleeping alone(!) Discusses Nakamura's work on 515 cosleeping deaths, of which 121 were overlayings, 394 entrapments; no data on alcohol use. Discusses the British CESDI study on SIDS risk with cosleeping (Blair et al, BMJ 1999). [See also Emery's work on suffocation, above under "SIDS v. suffocation," which also cites CESDI. --JKR]

What do the experts advise? CPSC, AAP.

What should we recommend? Discourage unsafe forms of bedsharing (above). Probably discourage it for infants under 4 months. "The relationship between SIDS and bedsharing is complex, and not amenable to one blanket recommendation."

What about roomsharing?

Tempelman, a Scottish police surgeon of 1892, who was first to draw attention to the role of alcohol in overlaying, reporting that of 258 infant suffocation cases, more than half occurred on Saturday night.

Factors relating to the infant's last sleep environment in sudden infant death syndrome in the Republic of Ireland. McGarvey C, McDonnell M, Chong A, O'Regan M, Matthews T. Arch Dis Child 2003; 88: 1058-1064. A five-year case-control study of 203 SIDS infants compared with 622 healthy controls. Data on class and heredity, pregnancy history, birth history, postnatal medical history, circumstances of death. Cosleeping and prone sleeping both increased the risk of SIDS by multivariate analysis in infants under 5 months birth age. This important study is cited by the 2005 AAP task force on SIDS Policy Statement (below) as finding increased risk of SIDS with cosleeping regardless of whether the mother smokes, and in settings of very young infant and unsafe sleep surfaces i.e. couch).

Cosleeping: benefits, risks, and cautions. Berkowitz CD. Adv Pediatr 2004; 51: 329-349.

SIDS in 20 regions of Europe: case control study. Carpenter RG, Irgens LM, Blair PS, England PD, Fleming P, Huber J et al. Lancet 2004; 363: 185-191. This is the ECAS study. Cited by Alexander & Radisch (2005) and Lahr & Rosenberg (letters, 2006) as an important study finding that cosleeping is unsafe. Cited extensively in the 2005 AAP Policy Statement as showing that cosleeping definitely increases the risk of SIDS under certain circumstances, including smoking mother, nonsmoking mother, unsafe sleep surface, age less than 8 weeks.

AAP Task Force on SIDS. Policy Statement: The changing concept of SIDS: Diagnostic coding shifts, controversies regarding sleeping environment, and new variables to consider in reducing risk. Pediatrics

2005 Nov; 116(5): 1245-1255. One paragraph is given over to a pithy review of the literature, noting that the ECAS authors and the Scottish authors have endorse the United Kingdom Department of Health's advice that the safest place for an infant to sleep in the first six months is in a crib in the paretns' room ("roomsharing.") States that this is a highly controversial area, because cosleeping also has been shown to have benefits in terms of bonding and breastfeeding. Cites Scheers, Rutherford, Kemp, 2003, Unger, Kemp et al, 2003, Kemp, Unger et al., 2000, Drago, Dannenber, 1999 as case series either demonstrating overlaying hazard or Undet deaths increase with cosleeping. Cites the following case-control studies as having been done specifically on SIDS and cosleeping:

Fleming, Blair et al, BMJ, 1996
 Hauck, Herman et al., 2003
 McGarvey et al., 2003
 Blair, Fleming et al., BMJ 1999
 ECAS Study, 2004
 Mitchell, Thompson, 2005 (In Rognum's book)
 Klonoff, Cohen et al. 1995
 Luke, JFS 1978
 Matthews et al., 2004
 Scragg et al., 1993
 Tappin et al. (Scotland), 2005

"Some of these studies" (Blair, Fleming et al., BMJ 1999 and Scragg et al., BMJ 1993) found hazard only with mothers who smoked. But ECAS and Scotland found hazard even with nonsmoking mothers, up to ages 8 weeks and 11 weeks, respectively. Says that the literature finds increased SIDS risk in the following circumstances:

multiple cosleeping (Hauck, Herman et al., 2003)
 alcohol (ECAS, 2004)
 overtired (Scragg et al., 1993)
 very young infant (McGarvey et al., 2003, Blair, Fleming et al., BMJ 1999, ECAS, 2004)
 unsafe sleep surface, e.g. couch (Blair, Fleming et al., 1999, Hauck, herman et al., 2003, McGarvey et al., 2003, Tappin et al. Scotland, 2005)
 greater duration of cosleeping (Fleming, Blair et al., 1996, Blair, Fleming et al, 1999, Scragg et al., BMJ 1993)

Cited by Alexander & Radisch (2005) for the proposition that cosleeping is hazardous and that infants should sleep alone, supine, and on a firm sleep surface lacking soft objects and loose bedding.

Sudden Infant Death Syndrome risk factors with regards to sleep position, sleep surface, and co-sleeping.

Alexander RT, Radisch D. JFS 2005 Jan; 50(1): 147-151. Retrospective review of the autopsy reports on 102 SIDS deaths from the North Carolina ME's Office, which comprised "all infant deaths in North Carolina during 1999 and 2000 that could possibly be considered SIDS." Studied the following particular risk factors:

prone	61%
cosleeping	47%
not in a crib	65%

Broke them down by under four months of age and over four months of age. Found that 92% had one or more of these three risk factors. Did not stratify by race or class. Lists the risk factors for SIDS as follows:

prone
 side
 not in crib
 unsafe bedding
 smoking

drug abuse
 prematurity
 male sex
 low birth weight
 young maternal age
 infection
 lower class
 no prenatal care

In a later exchange of letters (see below), the authors clarify the main point of this article. **The MAIN POINT is the statistical finding that “SIDS is rare in the setting of an infant sleeping alone, supine, and in a crib.”**

The literature support for the above **Main Point**, aside from the present authors’ findings, is the ECAS study of 2004, the Scottish study of 2005 (Tappin et al.), and the AAP article of 2005.

States, in briefly summarizing the risk-factor literature, that “Often a combination of risk factors is present.” (Citing Fleming et al., BMJ 1996; Kemp et al., 1998; Mitchell et al., 1998; Kemp, Unger et al., 2000; Gessner et al., 2001; Hauck et al., 2003; Gilbert-Barness et al., 1991; Leach et al., 1999.) The authors note that adult beds, couches, and waterbeds “are *independent* risk factors for *infant death* [note, not for SIDS --JKR] due to the potential for entrapment, wedging, and smothering.” [emphasis added --JKR]

Found the following sleep surfaces in use:

adult mattress	36%
crib/bassinette	34%
couch/sofa	13%
water bed	2%
other	13%

Position found:

prone	62%
supine	28%
side	8%
other	2%

Also detailed data on cosleeping -- who, what, and where.

Includes data on incidental autopsy findings.

pulmonary congestion, hemorrhage, or edema	44%
mediastinal petechiae	44%
visceral congestion	13%
cardiac malformation	12%
pulmonary inflammation	11%
tracheitis	5%
neuropathology	5%
aspiration	2%
fatty liver	2%
hepatitis	1%
fluid third-spacing (effusions)	1%

As to bronchopneumonia, the authors’ discussion says, “Although tracheal and pulmonary inflammation were not very common in the SIDS deaths (4.9% and 10.8% of cases, respectively), the significance of these lesions is debatable. A case-control study concluded that upper respiratory and pulmonary

inflammation are not a cause of death in SIDS (citing Krous, Blackbourne, et al., 2003). Others, however, have considered acute bronchopneumonia or bronchiolitis to fall into a 'gray zone;' these lesions may be considered lethal if sufficiently extensive and severe. (citing Valdes-Dapena's Histopathology Atlas, 1993.) These distinctions seem to fall into the category of semantics; most pathologists would probably be hesitant in diagnosing SIDS in a case of diffuse severe acute bronchopneumonia..."

As to fatty liver, states that "A fatty liver is not a sensitive screen for fatty acid oxidation disorders since a third or more of cases may have mild or absent steatosis." (citing Boles et al., 1998; MMWR, 2003)

As to limitations of this study, the authors note that there were no controls. That is, they documented the frequency of sleep risk factors in death infants, but not in the normal population. These limitations preclude them from making any recommendation about sleeping arrangements, including as regards cosleeping. "Whether or not there is an increased SIDS risk from co-sleeping with an unimpaired parent on an adult mattress requires additional study."

There was an exchange of letters on this article. Lahr & Rosenberg wrote in from the Oregon Department of Human Services (JFS 2006 Mar; 51(2): 427) to say that this article "adds nothing to the debate on the safety of maternal-infant bedsharing." Why? Number one, because there is no denominator; the authors do not know the incidence of bedsharing in the population. But these guys DO know it, from original research in Oregon: 35% of mothers always coslept and 41% sometimes did. And in a nationwide study, 19% of mothers did at least half the time and another 27% sometimes did it. But you have to know the incidence in your study population. Number two, they assumed that cosleeping with a nonsmoking mother is a risk factor for SIDS, even though that has never been demonstrated. Number three, they lumped unsafe sleep surfaces in with beds. The authors reply to point 1 that they never pretended to comment on whether cosleeping is or is not to be recommended. They acknowledged in their paper that they had no denominator; it was a purely statistical report. As to point 2, obviously the risk is controversial, but does that mean that you can't do research on it? As to number 3, Lahr & Rosenberg's own earlier population-based study lumped unsafe sleep surfaces in with beds and had no denominator! So how can they object to this? Also, while Lahr & Rosenberg characterize maternal cosleeping as an "acceptable option," recent ECAS study showed that maternal cosleeping increased the death risk in under 8 weeks infants significantly; are Lahr & R unaware of that study?? The authors emphasize their statistical finding that SIDS is rare in the context of an infant sleeping alone supine in a crib, and reiterate that the literature (Kemp, Unger et al, Unsafe sleep practices, Pediatrics 2000, and Gessner et al, Alaska, Pediatrics 2001) confirms that SIDS is rare outside the context of a sleep-related risk factor. Lahr & Rosenberg fire back that a European study (Tappin et al., 2005, below) showing risk came out while their letter was under submission, and they have now changed their opinion...

But the point is, literature on the risk of cosleeping is developing.

Bedsharing, roomsharing, and SIDS in Scotland: a case-control study. Tappin D, Ecob R, Brooke H. J Pediatr 2005; 147: 32-37. Cited by Lahr & Rosenberg, *supra*, as "an important paper that replicated the findings of the European Concerted Action on SIDS (ECAS) study of 2004..." to the effect that cosleeping with a nonsmoking mother is hazardous under 8 weeks.

Cosleeping and sudden unexpected infant deaths in Kentucky: a 10-year retrospective case review. Knight LD, Hunsaker DM, Corey TS. Am J Forens Med P 2005 Mar; 26(1): 28-32. These authors used a meta-analysis of four published studies to derive a presumed normal rate of cosleeping in the general infant population, which they found to be 24%. In their own ten-year retrospective review of 697 SIDS victims, the cosleeping rate was 36%. Therefore, the relative risk of death when cosleeping was 1.74. And when you took out the cases where sleeping arrangement was unknown, it was 2.05. Therefore, cosleeping represents a risk factor for sudden death. However, the authors also collected information on other aspects of the sleeping arrangement, such as couch, small bed, softness of bed, number of cosleepers, relationship of cosleepers. They noted that most of the cosleeping deaths occurred on surfaces not intended for infants. "Therefore, the actual hazard may also be related to confounding factors which create an unsafe cosleeping environment, such as cosleeping on couches and other unsafe surfaces (including beds too small for the number of individuals sleeping); soft bedding, pillows and comforters, parental intoxication or exhaustion;

cosleeping with adults who smoke; and cosleeping with noncaregivers or nonelective cosleeping with a disinterested caregiver due to socioeconomic factors. ...[P]erhaps it is not even cosleeping in itself that is dangerous, but the environment and circumstance in which cosleeping occurs.” Incidentally found petechiae:

thymic	47%
epicardial	39%
cutaneous or scleral	4%

Sleep arrangements and behavior of bedsharing families in the home setting. Baddock SA, Galland BC, Taylor BJ, Bolton DPG. Pediatrics 2007 Jan; 119(1): e200-e207. These researchers set up cams in the homes of 40 infants who were known to cosleep. Their cams showed that the infants slept facing the breast-level area of their mothers and the mothers slept facing the infants. Nursing occurred for average 40 minutes. Head-covering episodes occurred frequently because of changes in the mother's position. Infants were not exposed to maternal expired air. Lower room temperature caused an increase in infant head-covering instances. There were no deaths.

Benefits and harms associated with the practice of bed sharing. Horsley T, Clifford T, Barrowman N, Bennett S, Yazdi F, Sampson M, Moher D, Dingwall O, Schachter H, Cote A. Arch Ped Adol Med 2007 Mar; 161(3): 237-245. This is a meta-analysis of existing research. They found forty papers. “Evidence consistently suggests that there may be an association between bed sharing and sudden infant death syndrome among smokers, but the ev is not as consistent among nonsmokers. This does not mean that no association between bed sharing and SIDS exists among nonsmokers, but that existing data do not convincingly establish such an assoc. Data also suggest that bed sharing may be more strongly assoc w SIDS in younger infants. A positive association between bed sharing and breastfeeding was identified. Current data could not establish causality. It is possible that women who are most likely to practice prolonged breastfeeding also prefer to bed share.” Conclusion: need prospective cohort studies to define the benefits and harms. They give a nice chart showing all the factors studied, who studied them, and what conclusion was reached if any as to each factor. The factors and findings are:

<u>Factor studied</u>	<u># of studies</u>	<u>association (+ or -)</u>	<u>no association</u>
maternal alcohol consumption	4		Iyasu 2002 Klonoff-Cohen 1995 Scragg 1993 Klonoff-Cohen 1995
maternal recr drug consumption	1		
age of infant	3	Carpenter 2004 (-) Blair 1999 (-) McGarvey 2003 (-)	
birth weight	1		Arnestad 2001
death during daytime sleep	1	Williams 2002 (-)	
death on a weekend	1		Mitchell 1992
death away from home	1		Schluter 1998
> two layers of clothing	1		Iyasu 2002
>2 layers of covers	1		Iyasu 2002
duvets	1		McGarvey 2003
insulating property of bedding	1		McGarvey 2003
pillows	1		McGarvey 2003
found prone	1		McGarvey 2003
no pacifier	1		McGarvey 2003
breastfeeding started at birth	1	McGarvey 2003 (-)	
history of illness since birth	1	McGarvey 2003 (+)	
symptoms in the 48 hrs before death	1		McGarvey 2003
social disadvantage	1		McGarvey 2003
surface softness	1		McGarvey 2003
sofa sharing	1	Blair 1999 found unclear association	

The purported benefits of bedsharing are: (1) breastfeeding, (2) bonding, (3) better sleep.
The purported harms of bedsharing are: (1) SIDS.

PACIFIERS

AAP Policy Statement 2005: The changing concept of sudden infant death syndrome: diagnostic coding shifts, controversies regarding the sleeping environment, and new variables to consider in reducing risk. Task Force on Sudden Infant Death Syndrome. Pediatrics 2005 Nov; 116(5): 1245-1255. This thing recommends, "Consider offering a pacifier at nap time and bedtime: although the mech is not known, the reduced risk of SIDS assoc w pacifier use during sleep is compelling, and the ev that pacifier use inhibits breastfeeding or causes later dental complic is not. Until ev dictates otherwise, the task forced recommends use of a pacifieer throughout the first year of life acc to the following procedures:

The pacifier should be used when placing the infant down for sleep and not be reinserted once the infant falls asleep...

Pacifiers should not be coated in any sweet solution

Pacifiers should be cleaned often

For breastfed infants, delay pacifier introduction until 1 month of age to ensure tha breastfeeding is firmly established."

Cites Mitchell et al., 1993, Fleming et al., 1999, L'Hoir et al, 1999, Tappin et al., 2002, Hauck et al., 2003, Carpenter et al., 2004, McGarvery et al, 2004 as having reported a protective effect. The mechanism is unclear, maybe a lower arousal threshold. Gives a graphic presentation of the data from the above papers. giving the odds ratios they found, which are .44, .62, .16, .55, .33, .47, and .34, respectively.

Do pacifiers reduce the risk of SIDS? A meta-analysis. Hauck FR, Olanrewaju OO, Siadaty MS.

Pediatrics 2005 Nov; e716. <http://www.pediatrics.org/cgi/doi/10.1542/peds.2004-2631>

Conclusions: "Published case-control studies demonstrate a significant reduced risk of SIDS with pacifier use, particularly when placed for sleep. The studies reviewed attempted to control for social class, along with maternal age, parity, birth weight, tobacco, class, covered face, sleep[position, bedsharing, age, sex, breastfed, gestational term, etc."

Use of a dummy (pacifier) during sleep and risk of sudden infant death syndrome (SIDS): population-based case-control study. Li DK, Willinger M, Pettiti D et al. BMJ 2006; 332: 18-22. According to Dr Krous's review in the summer 2006 Quarterly, this paper showed a dramatic 90% reduction in SIDS risk. But pacifiers should only be introduced after one month of age and after breastfeeding has been established. The AAP has already opined as of 2005 (above), and Dr Krous says that this work supports those recs.

BLOOD IN THE NOSE/ FACE

The mistaken diagnosis of child abuse: a form of medical abuse. Kirschner RH and Stein RJ. Am J Dis Chil 1985; 139: 873-875. Presents 10 cases of erroneous diagnosis. 5 were SIDS. In some there was serosanguinous drainage from the nose and mouth, a common finding in SIDS.

Covert video recordings of life-threatening child abuse: lessons for child protection. Southall DP, Plunkett MD, Banks MW, Falkov AF, Samuels MP. Pediatrics 1997; 100(5): 735-760. Revealed attempted suff in 33 of 39 suspected cases. 0 in 46 controls. See editorial, "Unimaginable images: seeing is believing," by Richard D. Krugman, Pediatrics 1997 Nov; 100(5): 890-891, saying that it teaches that "(T)here are several straightforward clues that appear in these cases that should alert us..." (1) nasal or oral bleeding, (2) other siblings have died.

SIDS or murder? (letter). Becroft DM and Lockett BK. Pediatrics 1998 May; 101(5): 953-955
See above under "SIDS vs. suffoc." "**Bleeding from the mouth or nose** was observed during 6 of 10 previous ALTEs suffered by these children and three surviving infants who had been in the same care."

Unnatural sudden infant death. Meadow R. Arch Dis Child 1999 Jan; 80(1): 7-14. Retrospective review of the records and findings of 81 children judged by the courts to have been killed by their parents. Factors found:

4. Most homes were disadvantaged
5. Half the perps had a history of somatizing or factitious disorder.
6. 43% of the children had facial bruises, facial petechiae, or blood on the face.

AAP Policy Statement February 2001: Distinguishing SIDS from Child Abuse Fatalities. (RE 0036). AAP Committee on CAN. Pediatrics 2001 Feb; 107(2): 437-441. <http://www.aap.org/policy/re0036.html>
...Certain circumstances should indicate the possibility of intentional suffocation, including:

7. Previous recurrent cyanosis, apnea, or ALTE while in the care of the same person
8. Age at death older than 6 months
9. Previous unexpected or unexplained deaths of 1 or more siblings
10. Simultaneous or nearly simultaneous death of twins
11. Previous death of infants under the care of the same unrelated person
12. Discovery of blood on the infant's nose or mouth in association with ALTE's.

Nasal and intrapulmonary haemorrhage in SIDS. Becroft DMO et al. Arch Dis Child (Br.) 2001; 85: 116-120. Fresh intrapulmonary and nasal hemorrhage in SIDS as a ?marker for suffoc? Dr Krous has kindly provided the abstract:

"BACKGROUND: Fresh intrapulmonary and oronasal haemorrhages in cases of sudden infant death syndrome (IDS) might be markers for accidental or intentional smothering inappropriately diagnosed as SIDS.

AIM: To compare the incidence, epidemiological association, and inter-relation of nasal haemorrhage, intrapulmonary haemorrhage, and intrathoracic petechiae in infant deaths certified as SIDS.

METHODS: In SIDS cases from a large nationwide case-control study, a wide range of variables were compared in cases with and without report nasal haemorrhages and, in a sub group of cases, in those with and without pathologically significant intrapulmonary haemorrhage.

RESULTS: Nasal haemorrhage was reported on 60 of 385 cases (15%) whose parents were interviewed. Pathologically significant ontra-alveolar pulmonary haemorrhage was found in 47% of 115 case studied, but was severe in only 7%. Infants with nasal haemorrhage had more haemorrhage into alveoli and air passages than age matched cases without nasal haemorrhage. In multivariate analysis, nasal haemorrhage was associated with younger infant age, bed sharing, and the infant being placed non-prone to sleep.

Intrapulmonary haemorrhage was associated with the same three factors in univariate analysis, but in multivariate analysis only younger infant age remained statistically significant. There was no significant association between nasal or intra-alveolar haemorrhages and intrathoracic petechiae.

CONCLUSIONS: Nasal and intrapulmonary haemorrhages have common associations not shared with intrathoracic petechiae. Smothering is a possible common factor although is unlikely to be the cause in most cases presenting as SIDS."

Oronasal blood in sudden infant death. Krous HF, Nadeau JM, Byard RW, Blackbourne BD. Am J Forens Med Path 2001 Dec; 22(4): 346-351. Says the literature on this is limited; "little published information is available." Citing Beckwith, 1973; Krous, 1984; Krous, 1988; Meadow, 1990; Southall et al (covert video), 1997. They reviewed their database on 406 cases of SUDS, in 155 of which some type of oronasal

secretion was described. Found 40 cases (26%) in which the secretion was described as sanguinous. In 28 cases (18%) it was described as "blood." Analyzing these 28 cases, they found that half the time (14 cases) the blood was seen before CPR. Of these 14 cases,

10	SIDS
2	Acc suff
3	Undet

Of these ten SIDS cases, 4 had mod to severe lung hem, 5 had mild, 1 had no. Two of the 28 inf with blood had superficial oronasal lacerations possibly attributable to resusc. In 23 the frenula were recorded and were recorded as normal; 5 had no report on the frenula.

They conclude that oronasal blood, if observed before CPR, may be a sign of accidental or inflicted suffocation. As support for this conclusion, they note their findings that

4. Of the 14 non-CPR blood cases, all but 3 were cosleeping and only 2 were supine in a safe crib.
5. Blood was reported in 14% of all acc suff cases, as compared with 1% of SIDS cases.
6. Among the 10 non-CPR SIDS cases, the CPS history was only known in 3, but one of these three had multiple CPS referrals for abuse. That's a rate of 33%.

The authors advise:

3. Mucoïd or frothy bloody fluid expelled from pulmonary hemorrhage should be distinguished from frank blood.
4. An otoscope should be used to examine the oronasal skin and mucosa in cases where possible blood is seen. Frank blood is usually from local oronasal injuries. The cause of these is either attempted resuscitation or other physical injury to the face, accidental or inflicted.

Considering suffocatory abuse and Munchhausen syndrome by proxy in the evaluation of children experiencing apparent life-threatening events and sudden infant death syndrome. Truman TL, Ayoub CC. Child Maltr 2002; 7: 138-148. See under "MSBP." The risk factors for inflicted suffoc were:

1. Recurrent, poorly explained ALTE's
2. The same caregiver is the witness to most of the ALTE's
3. Blood in the mouth or nose
4. Bruising inconsistent with resusc
5. Siblings with other "medical problems." (Especially SIDS or ALTE).

Oral bleeding: child abuse alert. Stricker T, Lips U, Sennhauser FH. (Zurich). J Paediatr Child Health 2002 Oct; 38(5): 528-529. Two cases of child abuse in which oral bleeding was the presenting symptom. In both case, the dx was missed and the child represented with more serious injuries. Injuries to the oral cavity and oral bleeding of uncertain origin in infants should be carefully assessed in rel to the adequacy of the history to explain the injury.

PETECHIAE

The Pathology of Homicide. Lester Adelson, (1974):
"Asphyxial stigmata"

"The combination of such postmortem findings as (1) cyanosis, (2) persistent fluidity (or liquidity) of the blood, (3) facial, palpebral, bulbar, subpleural, and subepicardial petechiae, (4) right-sided cardiac

dilatation, and (5) visceral congestion was formerly accepted by some investigators as proof that death resulted from mechanical asphyxia...

“Ab initio it should be stated that the coexistence of these findings, in and of themselves, does not prove that death resulted from mechanical asphyxia.

All these phenomena are non-specific and in no way peculiar to this mode of death. Inasmuch as they are observed frequently in deaths arising from unquestioned natural causes, they have no value in proving that death resulted from mechanical asphyxia.” p. 527

A necropsy study of distribution of petechiae in non-sudden infant death syndrome. Krous HF, Jordan J. Arch Path Lab med 1984; 108: 75-76. Cited by Kim Collins, 2001, for the proposition that petechiae from neck compression are less frequent in infants.

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, below.)

Intrathoracic petechial hemorrhages: a clue to the mechanism of death in sudden infant death syndrome? Beckwith JB. Ann N Y Acad Sci 1988; 533: 37-47. I think this is the article where it is discussed whether the thymic petechiae are on the intrathoracic or the extrathoracic portion of the gland. Cf the photographs in Gilbert-Barness E., ed., *Potter's Pathology of the Fetus and Infant*. St. Louis: Mosby-Year Book, Inc., 1997, p. 439. Compare Kim Collins, 2001 and discussion in Kohr, 2003.

The forensic significance of conjunctival petechiae. Rao VJ, Wetli CW. Am J Forens Med P 1988; 9: 32-34. Cited by Kim Collins, 2001, for the proposition that petechiae from neck compression are less frequent in infants.

Hanging deaths in children. Cooke CT, Cadden GA, Hilton JMN. Am J Med Sci 1989; 10: 98-104. Cited by Kim Collins, 2001, for the proposition that “Previous researchers have found few hanged or strangled infants to have intrathoracic petechiae.”

Masque ecchymotique: specific or nonspecific indicator for abuse. Perrot J. Am J Forens Med Path 1989; 10: 95-97.

DiMaio DJ, DiMaio VJM, *Forensic Pathology*. New York: Elsevier, 1989. Cited by Kim Collins, 2001, for the proposition that petechiae from neck compression are less frequent in infants.

Suffocation, recurrent apnea, and sudden infant death. Meadow R. J Pediatr 1990 Sep; 117(3): 351-357. 27 babies who were suffocated by their mothers, 9 fatal and 18 nonfatal. Median age 9 mos, range 2-48 mos. 15 (55%) were over 6 mos. 5 had petechiae about the face or mouth.

Pathological findings in hanging and wedging deaths in infants and young children. Moore L, Byard RW. Am J Forens Med Path 1993; 14: 296-302. See under “Strangulation,” below. 14 cases of hangings and wedgings. See SIDS book, 2001, below, to the effect that oculofacial petechiae in an infant come only from compression asphyxia.

Histopathology Atlas for the Sudden Infant Death Syndrome. Valdes-Dapena MA, McFeeley PA, Hoffman HJ et al. Washington: Armed Forces Institute of Pathology, 1993:

Pleural petechiae: “Petechiae are encountered commonly in the pleura in both gross and microscopic examination in cases of sudden infant death. They probably represent agonal events and can be expected in 38% of microscopic sections of lung in SIDS cases. p. 103

Thymic petechiae: “Although not always present, thymic petechiae are a recognized hallmark of SIDS... Although characteristic, they are not diagnostic for SIDS and are often found in other types of deaths such as asphyxia... [T]hymic petechiae were noted, either in the gross autopsy or microscopically, in 69% of SIDS cases and 38% of explained deaths.” p. 111

Skin and other locations: “In contrast to the petechiae found often on the surfaces of the thoracic organs in SIDS, petechiae in other places, e.g. conjunctivae, oral mucous membranes, and epiglottis, suggest suffocation or some other asphyxial mechanism of death.” p. 270

Spitz and Fisher’s Medicolegal Investigation of Death, Third Edition (1993):

“However, it has been shown that petechial hemorrhages are by no means conclusive evidence of death by suffocation. Identical hemorrhages, especially of the conjunctivae and skin, are often found in cases of natural death...” p. 460

Forensic Pathology. Dominick J. DiMaio and Vincent J. M. DiMaio, (1993):

“Asphyxia”

“The classical signs of asphyxia are visceral congestion, cyanosis, petechiae, and fluidity of blood. These are nonspecific, however, and can occur in deaths from other causes... Injury to the walls of the capillaries may result in development of petechiae. These are most prominent in the visceral pleura and epicardium. Petechiae are nonspecific.” p. 207

“Sudden infant death syndrome”

“Petechiae of the thymus, epicardium, and pleural surfaces of the lungs are common. these, like the edema and congestion, are nonspecific and may be absent in obvious cases of SIDS and present in non-SIDS cases. The petechiae are due to nonspecific agonal anoxia.” p. 291

Petechial hemorrhages. Jaffe FA. Am J Forens Med P 1994; 15: 203-207. Cited by Kim Collins, 2001, for the proposition that petechiae from neck compression are less frequent in infants.

Intrathoracic and subconjunctival petechiae in sudden infant death syndrome (SIDS). Kleeman WJ, Wiechern V, Schuck M, Troger HD. FSI 1995; 72: 49-52. Said to be the only article showing oculofacial petechiae in an innocent setting. Questioned by Byard & Krous, 2001, below.

Knight’s Forensic Pathology, 2d ed. New York: Oxford University Press, 1996. Cited by Kim Collins, 2001, for the proposition that petechiae from neck compression are less frequent in infants. See the third edition, below.

A contribution to possible differentiation between SIDS and asphyxiation. Betz P, Hausmann R and Eisenmenger W. FSI 1998; 91: 147-152. Uses conjunctival petechiae and lung hyperinflation (emphysema) as indicia of suffocation, which were found exclusively in the suffocation group. See above under SIDS vs. Suffocation.

Petechial hemorrhages and unexpected infant death. Byard RW, Krous HF. Legal Medicine (Tokyo) 1999 Dec; 1(4): 193-197. From the abstract: “The significance of petechial hemorrhages in cases of unexpected infant death remains uncertain. While intrathoracic petechiae occur in the majority of cases of sudden infant death syndrome (SIDS), their relationship to terminal mechanisms has been debated. Facial, conjunctival, and external upper chest petechiae are not a feature of SIDS and raise the possibility of underlying illness such as sepsis, or of forceful coughing or vomiting. Alternatively the presence of cutaneous or conjunctival petechiae may suggest trauma or asphyxia due to chest or neck compression. Given the possibility of alternative mechanisms of death it is preferable to designate the cause and manner of death as ‘undetermined,’ rather than SIDS, when petechiae are found in unusual locations.”

Petechiae of the baby’s skin as differentiation symptom of infanticide versus SIDS. (Germany).

Oehmichen M, Gerling I, Meissner C. JFS 2000 May; 45(3): 602-607. Three siblings. The oldest one and last to die was 3 years old and was found to have obvious oral-nasal-throat injuries and facial petechiae, and the mother confessed to having suffocated her smaller two infants earlier. Upon review, these infants, aged 1 year and 3 months, were found to have facial petechiae, upper thorax, and intraoral, consistent with the SVC distribution. Mother confessed on all three.

Asphyxial deaths and petechiae: a review. Ely SF and Hirsch CS. JFS 2000; 45(6): 1274-1277. Pets are caused by impaired venous drainage, a purely mechanical event. Forms of asphyxia that don't involve compression rarely have pets. "[N]o relationship exists between the development of petechiae and the presence or absence of _____." (p. 1276)

Cindy Christian and Lynn Douglas Mouden, Maxillofacial, Neck, and Dental Manifestations of Child Abuse. In: *Child Abuse: Medical Diagnosis and Management, 2d ed.* Reece RM and Ludwig S, eds. Philadelphia: Lipincott Williams & Wilkins (2001), pp. 112-113: "Although strangulation or suffocation of a child may cause petechial hemorrhages of the face or neck, **this is not a reliable finding.** In a series of 14 pts who were intentionally suffocated during covert video surveillance, no child had facial markings that lasted more than 30 to 60 seconds after the attempted suffoc. (citing Samuels et al., 1992). Meadow reviewed the records of 81 children who were smothered to death. Blood in the mouth, nose, or on the face was reported in 39% of the chil, and only 10 chil had either bruises or petechiae on the face or neck. More than half of the victims had neither bruises, petechiae, nor a history or finding of bleeding."

Robert M. Reece and Henry F. Krous, Fatal Child Abuse and Sudden Infant Death Syndrome. In: Reece Rm and Ludwig S, eds., *Child Abuse: Medical Diagnosis and Management, Second Edition.* Philadelphia: Lipincott Williams & Wilkins (2001). "Intrathoracic petechiae, the most common abnormality seen with the naked eye, are identified in about 80% of SIDS cases... Experimental evidence and observations in human postmortem examinations suggest petechiae limited to the thorax can result by breathing against an obstructed upper airway during the moments preceding death. Alternatively, it also has been suggested that bronchiolar obstruction could cause the same finding. More recently, Poets et al identified intrathoracic petechiae in infants shown by monitoring to be gasping deeply before dying of SIDS." p. 528

Subconjunctival haemorrhage and respiratory distress. Chiu C-H et al. Lancet 2001 Sep 1; 358: 724. Case report of periorbital ecchymosis and subconjunctival hemorrhages from adenovirus.

Specific pathologic problems and possible solutions. Byard RW and Krous HF. In: *Sudden Infant Death Syndrome: Problems, Progress & Possibilities.* New York: Oxford University Press, 2001, pp. 230-231. Discuss petechial hemorrhages: Intrathoracic in 68% to 95% of cases. "By way of contrast, petechial hemorrhages on the conjunctiva and face are unusual in infants, and are generally seen only in cases of hanging or crush asphyxia. (citing Moore & Byard, Pathologic findings in hanging and wedging deaths in infants and young children, 1993). Although they have been described very rarely in cases where the death was attributed to SIDS (citing Kleeman et al., 1995), this has not been a general experience; for example, it has been stated that petechiae 'are never present on the conjunctiva, eyelids or on or in other soft tissues of the head or neck in SIDS.' " (citing Hilton, 1989). Further: "If facial or conjunctival petechiae are noted, the history should be carefully checked for forceful coughing or vomiting. **Pertussis** may result in petechiae of the face and conjunctiva (citing Nelson's Pediatrics, 1979)... External petechiae in cases of sudden and unexpected infant death are of greater importance than similar lesions in adults and must be regarded as a significant finding that requires explanation. If no other plausible explanation can be elucidated, mechanical asphyxia, whether induced or accidental, must be seriously considered.... require extremely careful investigation, with close collaboration among the various agencies involved..."

Death by overlaying and wedging. Collins KA. Am J Forens Med P 2001 Jun; 22(2): 155-159. See above under "SIDS v. suffocation." Found 32 cases diagnosed as either overlaying, SIDS versus overlaying, wedging, and other accidental asphyxia. Found

"asphyxia due to overlaying"	11
"SIDS v. overlaying"	10
wedging	8
other accidental asphyxias	<u>3</u>
total	32

Of the 21 overlaying and SIDS v. overlaying cases, the ages were 6 days to 11 months. Twelve of them were cosleeping with more than one other person. Only two adults admitted drinking. Of these 21 cases, found:

ocular petechiae	3
facial petechiae	1
intrathoracic petechiae	19

“With neck compression of strangulation, one may see ocular and facial petechiae as venules rupture secondary to the increased pressure as veins are occluded and arterial patency maintained. However, this has been noted to occur less frequently in infants and young children.” (citing Knight, 1996; Byard & Cohle, 1994; Rao & Wetli on conjunctival petechiae, 1988; Jaffe on petechiae, 1994; DiMaio’s book; Krous & Jordan, necropsy study of petechiae, 1984.) As to intrathoracic petechiae, they may be caused by forceful respiratory efforts against an occluded airway; almost never seen in hangings and strangulations. “Like SIDS victims, overlaying and wedging victims usually have a completely negative autopsy. The presence of intrathoracic petechiae supports the theory of mechanical asphyxia.” This article is cited by Kohr, 2003, below, for the proposition that in eleven overlaying victims they found only two who had ocular petechiae and one who had facial petechiae, while nine had intrathoracic petechiae.

Intrathoracic petechiae in sudden infant death syndrome: relationship to face position when found. Krous HF, Nadeau JM, Silva PD, Blackbourne BD. *Pediatr Dev Pathol* 2001 Mar-Apr; 4(2): 160-166. The authors distinguish the “face-down position” from the “prone” position. The “prone” position would be either face-down or face-to-the side. In fact, 37% of the prone were face down. 199 SIDS cases. 142 found prone. 74 found prone and face down. Found:

	face down	not face down	
had intrathoracic petechiae	39%	61%	=100%
did not have intrathoracic petechiae	9%	91%	=100%

The P value for the above was $P=.057$, which the authors comment that “The wide confidence interval yielded by our analysis limits our ability to clarify the precise pathophysiologic role of external oronasal obstruction in SIDS. While it remains possible that a subset of SIDS cases occur as a result of external obstruction, we are unable to generalize its importance. Internal airway obstruction and rebreathing with terminal gasping, both of which have been documented in sudden infant death, remain other possible scenarios leading to the production of intrathoracic petechiae.”

Cases were also grouped by the severity of the thymic petechiae. Found that the face-down position was not associated with any difference in the severity of thymic petechiae.

Environmental hyperthermic infant and early childhood death: circumstances, pathologic changes, and manner of death. Krous HF, Nadeau JM, Fukumoto RI, Blackbourne BD, Byard RW. *Am J Forens Med Path* 2001 Dec; 22(4): 374-382. Ten documented cases -- eight in vehicles and 2 in beds. Age range 53 days to 9 years. Living or longer-surviving victims had centrilobular necrosis, myonecrosis or rhabdomyolysis, and DIC. Lung disease renders them more vulnerable. All 8 children discovered in vehicles had intrathoracic petechiae, while one of the two children found in bed had them. Gives an extensive discussion of intrathoracic petechiae. Since SIDS also frequently have intrathoracic petechiae, and SIDS have been shown by Guntheroth & Spiers to be associated with thermal stress, these may be the extreme end of a heat continuum that includes SIDS. In that case, meticulous scene investigation is required to differentiate heat exposure. Says that as to homicide versus accidental manner of death, “The manner of death was considered accidental when the investigation indicated miscommunication among the caretakers, or if the child entered the vehicle without the caretaker’s knowledge.” (p. 381)... The manner of death was homicide in 5 cases on the basis of inflicted trauma and circumstances indicating neglectful parenting... Verification of the caretakers’ understanding of their responsibilities regarding the child’s welfare is critical.” The autopsy findings mainly vary by the duration of survival *after* the heat exposure.

Inflicted compressional asphyxia of a child. Kohr RM. JFS 2003 Sep; 48(5): 1148-1150. (Terre Haute). Case report of a two year old in a day care center who refused to go to sleep, so she was taken out in the hall and placed face down on a floor mat, and a 130-lb. adult female attendant with a leg cast. Had no petechiae and no findings except for a pattern abrasion on the back that matched the leg cast. Discusses the lack of findings in slow compression. See under "SIDS v. suffocation, above. Cites Kim Collins, Death by overlaying, Am J Forens Med P 2001 for the lack of petechiae in overlaying.

Knight's Forensic Pathology, Third Edition. Pekka Saukko and Bernard Knight. London: Arnold, 2004. "Petechiae in the eyes and intra-ocular haemorrhages can occur after CPR, as well as after violent sneezing or coughing; they are well known to occur during whooping cough." p. 40

"The autopsy diagnosis of 'asphyxia'"

"Only by a careful assessment of the history and circumstances of the death, exclusion of other causes, and a cautious evaluation of the signs described above, can any conclusion be reached. Most important of all is the finding of a cause for airway obstruction or other local trauma,, such as prolonged pressure on the neck or chest, obstruction of the airways, postural causes, or occlusion of the external respiratory orifices."

"It cannot be emphasized too strongly that the mere finding of any of the non-specific features, such as congestion and petechiae, without firm circumstantial or preferably physical evidence of mechanical obstruction of respiration, is quite insufficient to warrant a speculative diagnosis of asphyxia. If such collateral evidence is not forthcoming, then the cause of death must be left undetermined." p. 357

Cosleeping and sudden unexpected infant deaths in Kentucky: a 10-year retrospective case review. Knight LD, Hunsaker DM, Corey TS. Am J Forens Med P 2005 Mar; 26(1): 28-32. Reviewed 697 SUDS autopsies over a ten year period looking for cosleeping effects. Incidentally found petechiae:

thymic	47%
epicardial	39%
cutaneous or scleral	4%

Three subsequent infanticides covered up as SIDS. Bohnert M, Perdekamp MG, Pollak S. Int J Legal Med 2004; 119: 31-34. This family had three SIDS in nine years. Cases had marked pulmonary hemorrhage and pulmonary interstitial emphysema and visceral petechiae.

How useful is the presence of petechiae in distinguishing non-accidental from accidental injury? Nayak K, Spencer N, Shenoy M et al. Child Abuse & Neglect 2006; 30: 549-555. Quite useful. Larry Ricci points out in his review for the Autumn 2006 Quarterly that the usefulness of petechiae depends on the pre-test prevalence of abuse in the subpopulation being studied. In a general population with a pre-test probability of 6.8%, the post-test probability with petechiae rises to 33.8% -- significant enough to warrant further investigation of these pts. In an abuse clinic population with a pre-test probability of 58%, the post-test probability of abuse rises to 99%. The article's authors found that in a well-child clinic the prevalence of petechiae was 1.5%, while in a child abuse clinic it was 16%. Also, 21% of all abused chil had petechiae, while only 2.3% of the accidental group. The SENS of petechiae was 22%, SPEC 98%, PPV 80%.

PULMONARY SIDEROPHAGES

Total differential diagnosis collected from all sources

Infection
 Blood dyscrasias such as thalassemia
 Heart failure
 Pulmonary hypertension
 Idiopathic pulmonary hemosiderosis
 Fungal infection, past or present
 Acute pulmonary hemorrhage of infancy
 Bone marrow transplantation
 Immunodeficiency disorders
 Heiner's syndrome
 Celiac sprue
 Vasculitis (Henoch, SLE, IgA, PAN, JRA, ITP, WG, Behcet's, drug rxn)
 Goodpasture's syndrome
 Prematurity
 Abruptio placentae
 NICU
 Trauma
 Near-miss SIDS
 Past episodes of hypoxia
 Imposed suffocation
 Accidental suffocation
 Physical abuse

Time course of hemosiderin production and clearance by human pulmonary macrophages. Sherman JM, Winnie G, Thomassen MJ et al. Chest 1984; 6: 409-411. Cited by Schluckebier et al. (2002) for the proposition that "[I]t takes macrophages 33 to 48 hours to break down hemoglobin. Siderophages can remain in the lungs for as long as two weeks, if not longer." Discussed by Forbes & Acland (2004) in their literature review to the effect that the authors studied hemosiderin formation and clearance in four infants with known acute pulmonary hemorrhage, found that one case remained negative, three cases showed hemosiderin at earliest 50 hours. Also did an experiment with human alveolar macrophages in vitro to sheep erythrocytes, finding that they formed HS at earliest 72 hours.

Interstitial haemosiderin in the lungs of SIDS: a histological hallmark of 'near-miss' episodes? Stewart S, Fawcett J, Jacobson W. J Pathol 1985; 145(1): 53-58. The well-known presence of pleural petechiae in SIDS cases stimulated these authors to do iron stains on 24 consecutive sudden infant deaths. (Judging from the article's title, apparently all were diagnosed as SIDS.) Found:

Hemosiderin macrophages	13 babies	54%
No hemosiderin macrophages	11 babies	46%

Ten out of the thirteen positive cases were in the classic SIDS age range of one to three months and had clean lungs free of hemorrhage or inflammation. All of the negative cases were older and had lung inflammation. Because pleural petechiae are thought to be associated with sudden apnea, therefore hemosiderin (assumed to represent previous pleural petechiae) could represent previous episodes of sudden apnea. From the abstract: "The demonstration of previous hemorrhagic foci in babies showing no other pathological abnormality may represent a histological hallmark of previous 'near-miss' episodes of hypoxaemia from whatever cause."

If you follow that syllogism, then 54% of SIDS could have had previous (unrecognized or unreported) episodes of apnea of hypoxia "from whatever cause." This logic could be used as an argument for home apnea monitoring. This article is cited along with Becroft & Lockett, 1997, by Schluckebier et al. (2002) for the proposition that "Several studies have evaluated whether the presence of hemosiderin-laden macrophages in the lungs of infants who die unexpectedly may indicate previous hypoxic events" and along with Becroft & Lockett and Hanzlick, Delaney, 2000 for the proposition that "Some authors have suggested their presence in large numbers should preclude a diagnosis of SIDS." But it's not clear that the

article says that. Forbes & Acland in their 2004 review describe this article as the first study to link pulmonary hemosiderin with infant hypoxia. Say that the authors suggest that the hemosiderin macrophages result from earlier petechiae, which themselves could have resulted from near-miss hypoxic attacks. Forbes & Acland point out an overlooked finding in this work -- the finding that all but one of the cases that had interstitial hemosiderin macrophages also had alveolar hemosiderin macrophages.

Neonatal and infantile pulmonary hemorrhage: an autopsy study with clinical correlation. Coffin CM, Schechtman K, Cole FS, Dehner LP. *Pede Path* 1993 Sep-Oct; 13(5): 583-589. Found pulmonary hemorrhage in 24% of stillborns and 74% of liveborns. Reviewed past literature to find hemosiderin in 7-18% of infant lungs.

The postmortem examination. Valdes-Dapena MA. *Pediatr Ann* 1995; 24: 365-372. Agonal alveolar hemorrhage may occur in two-thirds of infant autopsies.

Miller RR. Pulmonary disease in the immunocompromised host. In: Thurlbeck WM, Churg AM, eds. *Pathology of the Lung, 2d ed.* New York: Thieme Medical Publishers, 1995: 349-364. Cited by MMWR, 2004 (below) as stating that hemosiderin in lung macrophages indicates degradation products of hemoglobin.

Intra-alveolar pulmonary siderophages in sudden infant death: a marker for previous imposed suffocation. *Pathology* 1997; 29(1): 60-63 Becroft DM and Lockett BK. Iron stains showed previously overlooked alveolar siderophages widely distributed in the lungs of two pairs of siblings who had hospital admissions for multiple ALTE one to three weeks before dying suddenly at home, plus one additional infant who was suffocated at home. (Total four out of five babies, which the authors describe as “known” asphyxic homicide.) The fifth baby had negative lungs and also had no history of previous ALTE. [In a later report (see *letter*, 1998, below), they report a third pair of dead siblings dying under suspicious circumstances and having intraalveolar siderophages.] They also reviewed retrospectively 158 SIDS infants and found

158 SIDS

143 with no ALTE history

7 had alveolar siderophages (5%)

15 with ALTE history

1 had alveolar siderophages (7%)

seven who had diffuse alveolar siderophages. But Forbes & Acland (2004) point out that four of these seven were NICU graduates. They caution that this is different from *interstitial* siderophages, which are nonspecific, citing Byard RW et al., Assessment of pulmonary and intrathymic hemosiderin deposition in SIDS. *Ped Pathol Lab Med* 1997; 17: 351-357; cf. Stewart S et al, 1985, above. Bleeding from the mouth or nose was observed during six of ten previous ALTEs suffered by these children and three unrelated infants in the same care. “Such external hemorrhage is well described in imposed infant suffocation...” See Letter, below. This article is cited along with Stewart, 1985 by Schluckebier et al. (2002) for the proposition that “Several studies have evaluated whether the presence of hemosiderin-laden macrophages in the lungs of infants who die unexpectedly may indicate previous hypoxic events.” This article is discussed by Forbes & Acland in their 2004 review to the effect that it was Becroft & Lockett who first suggested that alveolar hemosiderin macrophages could be considered grounds for suspicion of previous imposed suffocation. Forbes & Acland comment that if alveolar siderophages really indicated previous imposed suffocation, “it might be expected that a greater proportion of hte cases with reported ALTE’s would give a positive stain.”

Assessment of pulmonary and intrathymic hemosiderin deposition in SIDS. Byard RW, Stewart WA, Telfer S, Beal SM. (Adelaide). *Ped Pathol Lab Med* 1997 Mar-Apr; 17(2); 275-282. Looked for pulmonary *interstitial* hemosiderin in 12 SIDS infants with histories of ALTE’s and 22 SIDS infants without histories of ALTE’s.

Previous history of ALTE

4/12 = 33%

No previous hx of ALTE

4/22 = 18%

All thymuses were negative. Noted that 66% of the SIDS with hx of ALTE stained negative, so this finding was non-diagnostic. Compare Becroft & Lockett (above), who found that three pairs of suffocated siblings had *alveolar* hemosiderin, which they feel has a different significance from interstitial hemos. cf. Stewart et al, 1995.

Update: pulmonary hemorrhage/hemosiderosis among infants -- Cleveland, Ohio, 1993-1996. MMWR January 17, 1997; 46(02); 33-35 (have). Rainbow Babies studied 10 cases and 30 controls. Acute idiopathic diffuse pulmonary hemorrhage/hemosiderosis. Pulmonary hemorrhages recurred in five of the infants after they returned to their homes. One infant died. The cases were all clustered in the same neighborhood. Nine of the affected infants and seven of the controls lived in water-damaged homes. The air concentration of *Stachybotrys atra* was significantly higher in the homes of case infants. Then they found an additional 11 cases, of which two died. So then with three deaths the Coroner reviewed all 172 infant deaths seen during the period 1993-95. He found that nine infants (5%) had +lung hemosiderosis. Two of these were homicides and one had a past history of child abuse. No etiol id's for the other six. Two had had symptoms (epistaxis, hemoptysis, and four had cough or melena). Editorial: "The review by the Coroner indicated that some infant deaths initially attributed to SIDS actually resulted from pulmonary hemorrhage. Agonal alveolar hemorrhage may occur in approximately two thirds of infant autopsies. [citing Valdes-Dapena, *supra*]. However, the presence of extensive hemosiderin-laden macrophages within the alveoli indicates major predeath pathologic processes... macro require approx 48 h to convert blood into hemos. DDX wd be (a) cardiac, (b) trauma, (c) pn, (d) perhaps suffoc. See Letter, next below, which comments on this report. For further on *S. atra*, see *California Morbidity*, April 1998, below. According to Schluckebier et al. (2002, *infra*), he following other refs have supported *Stachybotrys* as a cause of infant pulmonary hemorrhage: Coffin, 1993; Pappin, 1994; Goretsky, 1996; Pappas, 1996; Etzel, 1998; Elidemir, 1999; Stocker & Dehner, 2001.

Boat TF. Pulmonary hemorrhage and hemoptysis. In: Chernick V, Boat TF, Kendig E., eds. *Kendig's Disorders of the Respiratory Tract in Children*, 6th ed. Philadelphia: W. B. Saunders, 1998: 623-633. Cited by MMWR, 2004 (below) for the proposition that Hemosiderin is a pathologic state indicative of bleeding of any type into the lungs.

Alveolar hemorrhage in 200 babies. Jem Berry, MD, (personal communication, 1999). Observations from 200 cases. *Extensive* lung hemorrhage is more often seen in very young babies. Is seen more often in suffocations (both accidental and intentional), and this association becomes stronger as the hemorrhage becomes more extensive. But is nonspecific. [Henry Krous reports that lung hemorrhages are very common in SIDS.] Forbes & Acland (2004) suggest that very young age (under 3 mos or premature) could be an independent variable explaining alveolar hemosiderin in infants.

Intra-alveolar haemorrhage in sudden infant death syndrome: a cause for concern? (editorial) J Clin Pathol 1999; 52: 553-554.

Prognosis in pediatric idiopathic pulmonary hemosiderosis. Saeed MM, Woo MS, MacLaughlin EF, Margetis MF, Keens TG. (USC) Chest 1999 Sep; 116(3): 721-725. These patients are living longer. (Previous mean survival was 2 ½ years.) These authors report ___ patients. Mean age at diagnosis was 4 ½ years (range of 1 year to 8 years). All presented with anemia and pulmonary infiltrates. 70% had fever, 65% had hemoptysis. The diagnosis is by lung biopsy or BAL. Five-year survival on immunosuppressants was 86%. Some died of massive hemoptysis. There is a female predominance.

Histological demonstration of hemosiderin deposits in lungs and liver from victims of chronic physical child abuse. Dorandeu A, Perie G, Jouan H, Leroy B, Gray F, Durigon M. International Journal of Legal Medicine 1999; 112: 280-286. Forbes & Acland (2004) report that this study quantitatively compared the amount of hemosiderin in the lungs of 15 chronic child abuse cases with 15 age-matched SIDS cases. The quantity of hemosiderin was significantly greater in the chronic abuse cases. BUT, four of the abuse cases had no hemosiderin and three of the controls had hemosiderin, leading Forbes & Acland to opine that "the stain is not specific."

- 15 chronic child abuse
 - 11 had hemosiderin (73%)
 - 4 did not have
- 15 SIDS cases
 - 3 had hemosiderin (20%)
 - 12 did not have.

Iron disequilibrium in the rat lung after instilled blood. Ghio AJ, Richards JH, Crissman KM, Carter JD. Chest 2000; 118(3): 814-823. Cited by Forbes & Acland (2004) for the proposition that these authors found hemosiderin in that rat after four days after instillation of blood.

Frequency of pulmonary hemosiderosis in eastern North Carolina. Jackson CM and Gilliland MGF. Am J Forens Med Path 2000 Jan; 21(1): 36-37. Retrospective study by doing iron stains on the lung tissue of all of the 206 young children (premature newborns out to age 49 months) autopsied over an 18-year period in an area subject to frequent flooding. Of these, 23 had pulmonary siderophages. (About 11%). By original dx these broke out as:

SIDS	7
Infection	7
Congenital anomaly	3
Prematurity	2
Undet	4

There were no fungi. The authors do not explain what the iron means. They refer to Dehner's study (1993) reporting a 7-18% rate of pulmonary hemosiderin finding in stillborns and liveborns in the past. Refers also to Stewart et al. consideration (1985) that hemosiderin could be a marker for previous "near-miss" SIDS episodes. Note that in this present study there is no particular association with SIDS. The main point of this article is to deconfirm the earlier Cleveland study, and to show that there is no evidence of an emerging fungal disease as a cause of this. Reference to the pede path literature by them shows that lung bleeding at autopsy is frequent in stillborns and liveborns; but the significance of pulmonary hemosiderin is left up in the air. They don't comment on the interstitial versus alveolar distinction raised by Becroft & Lockett (1997) and Byard (1997). The article ends with the somewhat cryptic statement that, "Our study confirms that an iron stain may identify sufficient pulmonary hemosiderosis to reconsider attributing death to SIDS." Does this mean that the *amount* of hemosiderosis is a significant diagnostic variable? Forbes & Acland (2004) conclude that this work goes against the suggestion that hemosiderin macrophages are a marker for previous asphyxic abuse, because there was no difference in hemosiderin amount or prevalence between the SIDS cases and the cases of asphyxia (22 cases) and physical abuse (15 cases.)

Pulmonary hemosiderin in deceased infants: baseline data for further study of infant mortality. Randy Hanzlick and Kevin Delaney. Am J Forens Med Path 2000 Dec; 21(4): 319-322. Did Prussian blue stains on the autopsy lungs of 59 infants without regard to cause of death. Examined four slides on each case (anterior and posterior upper lobes). Each of three pathologists scored the staining on each section 0 through 4, for a maximum possible score of 48 (16 per pathologist.) The overall average score per infant was 6. There were six cases with an iron score of 12 or higher; one of these was SIDS, the others were congenital heart disease, abruptio placentae, bronchopneumonia, overlaying, and drowning. "The findings of this study suggest that the presence of hemosiderin in infant lungs, especially if focally abundant and present in many or most microscopic fields, should prompt special consideration that the cause of death may not be SIDS." This article is cited along with Stewart, 1985 and Becroft, 1997 by Schluckebier et al (2002) for the proposition that "Some authors have suggested their presence in large numbers should preclude a diagnosis of SIDS." This article is discussed by Forbes & Acland in their 2004 review to the effect that in three of the the high-iron-score cases the high score is not explained by the acute-death history and in these three cases, episodes of previous asphyxia cannot be discounted, but neither can they be assumed. In the other three cases, the high score can be explained on the grounds of the history.

Alveolar hemorrhage syndromes: update on pulmonary hemosiderosis. Epstein CE and Fan LL. J Respir Dis Pediatr 2001 Feb; 3(1): 49-56. The generic term is PH -- pulmonary hemorrhage. This includes a list of DDX, which they break out into two broad groups: those that are not part of a vasculitis, and those that are part of one. (Or, as they put it, “without pulmonary capillaritis,” and “with pulmonary capillaritis,” -- “capillaritis” not being defined but apparently meaning any derangement of the vascular structure.

Without capillaritis

Cardiac

Noncardiac

Idiopathic pulmonary hemosiderosis (IPH)

Acute pulmonary hemorrhage of infancy

Bone marrow transplantation

Immunodeficiency disorders

Heiner's syndrome (cow's milk allergy)

Celiac sprue

Infanticide

With capillaritis (systemic vascular diseases that include pulmonary “capillaritis”)

Goodp, SLE, IgA nephr, PAN, JRA, ITP, WG, Behcet's, Henoch, drug rxn...

Now as to IPH, it mainly presents in infancy and early childhood. It usually has cough and anemia as its cardinal signs, and the initial ddx is that of anemia. It can have an acute presentation or an insidious presentation. The acute presentation is severe hemoptysis, with or without wheezing, dyspnea, cyanosis. The insidious presentation is recurrent episodes of pulmonary bleeding sometimes associated with fevers. Symptoms include pallor, lethargy, cough (which may be the only symptom), and FTT. The authors also wrote another paper, next below, on the time-course of hemosiderin production.

Time course of hemosiderin production by alveolar macrophages in a murine model. Epstein CE, Elidemir O, Colasurdo GN, Fan LL. Chest 2001; 120(6): 2013-2020. Discussed by Forbes & Acland in their review (2004) to the effect that these authors using a mouse model found that hemosiderin first became detectable in lavage three days after tracheal instillation of blood, peaked at days 4-14, and was still present in some at 60 days.

Nasal and intrapulmonary haemorrhage in SIDS. Becroft DMO, Thompson JMD, Mitchell EA. Arch Dis Child (Br.) 2001; 85: 116-120. According to Forbes & Acland (2004), these authors studied the epidemiology of intrapulmonary hemorrhage in 115 cases of SIDS, finding a significant association with younger infant age (<13 weeks), bedsharing, and the nonprone sleep position in univariate analysis, while younger age remained the only significant variable in multivariate analysis. 47% of the infants had at least some pulmonary hemorrhage. Therefore, the finding is nonspecific. As to hemosiderin this study found large numbers of hemosiderin macrophages in 5 cases and small numbers in an additional 17 cases, with no relationship between hemosiderin and the finding of acute hemorrhage.

Pulmonary hemorrhage in deceased infants: baseline data for further study of infant mortality. Hanzlick R. Am J Forens Med P 2001 Jun; 22(2): 188-192. Found that lung hemorrhage is common but usually “patchy, focal, and sporadically distributed.” Resuscitation may exacerbate it. Tends to be more prominent in long postmortem interval. Position when found may affect its distribution. “The constellation of significant pulmonary hemorrhage, elevated macrophage counts, and above-average pulmonary hemosiderin load is rare.” Idiopathic pulmonary hemosiderosis (IPH) is also rare and lacks diagnostic criteria.

Pulmonary siderophages and unexpected infant death. Schluckebier DA, Cool CD, Henry TE, Martin A, Wahe JW. Am J Forens Med P 2002 Dec; 23(4): 360-363. (Denver CME) Retrospectively reviewed two years of all infant deaths n=43. Used iron stains and blindly counted siderophages per 20 hpf, ranging from less than 5 to over 500. Sidero categories set up were:

Category 1	less than 5 sideros/20hpf
Category 2	5-200 “
Category 3A	100-500 “

	Category 3B		over 100 in any one lobe
	Category 4		over 500 “
Cases analyzed:			
	SIDS		16
	Asphyxia		5
	Undet		6
	Other		16
All the SIDS cases fell into category 1 (less than 5 sideros/20hpf).			
Found:	Category 1	32	16 SIDS, 1 SUDS, 3 undet, 1 drown. 1 positional, 1 interst pn, 1 CHD, 8 diseases and accidents
	Category 2	6	1 CHD, 3 NAT, 2 undets
	Category 3	4	1 NAT, 1 mechanical asphyxia, 1 UNDET probable asphyxia, 1 UNDET/UNDET
	Category 4	1	Probable asphyxia by suffoc

The authors specifically anticipated the counterargument that siderophages might be merely a by-product of mechanical ventilation (citing Pappin, 1994, Goretsky, 1996). So they collected all the ventilator cases in Table 2 (six cases), showing that while all but one of these had siderophages, the only one with a large number of siderophages was a homicide.

Concluded: “All of our cases that demonstrated large numbers of pulmonary siderophages were cases in which the circumstances surrounding the death were suspect.” “This study provides further evidence that unexplained pulmonary siderophages can be a marker for trauma or repeated hypoxia/ asphyxia. Siderophages may also be increased for other reasons, but not to the same degree. Siderophages are not increased in SIDS or in acute asphyxial deaths. Because iron-laden macrophages often are not recognized on routine hematoxylin and eosin examination, iron stains may be helpful in the eval of infant deaths. If siderophages are present in increased numbers without an obvious explanation, further investigation is warranted.” And “This study supports the belief that routine iron stains can be useful in further invest of unexplained infant deaths.” But Forbes & Acland (2004) reviewing the article, conclude that this study supports the view that unexplained alveolar siderophages can be a marker for trauma or repeated hypoxia, but this study alone is not large or detailed enough to provide substantial proof. Forbes & Acland point out that of the six deaths regarded as suspicious by the authors, two had no history of abuse and one had been on a ventilator.

States that the DDX of pulmonary hemorrhage in infants includes

- Complications of prematurity
- Complications of NICU
- Hypoxia/asphyxia
- Trauma
- IPH
- Fungal

(Compare Epstein & Fan, 2001, above.)

Interestingly, in this study of 43 coroners autopsies including eleven with pulmonary hemosiderosis, in only one case did the original pathologist pick up the fact that there were pigmented macrophages in the lungs, and none of the cases had iron stains done in the original processing. –JKR

MMWR: Acute Idiopathic Pulmonary Hemorrhage Among Infants. MMWR March 12, 2004, vol. 53, no. RR-2. “AIPH is the sudden onset of pulmonary hemorrhage in a previously healthy infant in whom differential diagnoses and neonatal medical problems that might cause pulmonary hemorrhage have been ruled out.” For a differential diagnosis see below. (Infant has to be previously healthy and of GA > 32 weeks.) The CDC uses three levels of diagnosis:

Definite cases
Probable cases
Suspected cases

For a definite case, all of three diagnostic criteria to be met:

1. Abrupt onset of overt bleeding or obvious evidence of blood in the airway, identified either by laryngoscopy / intubation, or by identification of hemosiderin macrophages in BAL

and

2. Severe-appearing illness leading to acute respiratory distress

and

3. Diffuse pulmonary infiltrates

Diagnosis requires also that the infant be previously healthy (never hospitalized or intubated) and physical abuse ruled out, and of course that any etiology for the bleeding, such as coagulopathy or a lung mass, be ruled out.

Probable and suspected cases include those who are found at autopsy to have evidence of airway bleeding not due to traumatic intubation. They propose using these criteria to do clinical and epidemiological research, as was previously done with SIDS.

Known causes of pulmonary hemorrhage to be ruled out include:

mechanical disruption of normal vascular tissue (mitral valve disease and intentional suffocation)

vascular inflammation

vascular malformations

coagulopathies

lung immaturity

cow's milk allergy

pulmonary capillary hemangiomatosis

pulmonary hemorrhage of the newborn

(This is part of IRDS and BPD, usually in SGA preemies with history of perinatal stress.)

Alveolar hemosiderin macrophages indicate degradation products of hemoglobin, according to Miller in Andrew Churg's 1995 textbook (see above). "Hemosiderin is thus a pathologic state indicative of bleeding of any type into the lungs secondary to the processing of hemoglobin in the red blood cells in the airway by alveolar macrophages. (Citing Miller, 1995 and Boat, 1998). One clinical text (Murray & Nadel, *Textbook of Respiratory Medicine*, 1988) defined IPH as pulmonary hemorrhage that is not due to

- trauma
- bleeding from the large airways
- tumors
- left ventricular failure

This present MMWR article gives a list of *neonatal* medical problems associated with pulmonary hemorrhage:

- asphyxia
- instrumentation/intubation/mechanical ventilation/NGT
- HMD
- BPD
- chronic lung disease
- any left-sided congenital heart disease or with a L-R shunt
- cor triatriatum
- hemolysis
- pulmonary hypertension of the newborn
- surfactant administration
- venoocclusive disease

It also gives a differential diagnosis of pulmonary hemorrhage:

pulmonary	<ul style="list-style-type: none"> congenital or acquired lung disorders lung disease of prematurity primary ciliary dyskinesia bronchiectasis cystic fibrosis chronic aspiration gastroesophageal reflux inhaled toxins radiation injury drug reaction insecticides
cardiac	<ul style="list-style-type: none"> pulmonary hypertension congenital heart disease myocarditis pulmonary vascular congestion mitral stenosis congestive heart failure venoocclusive disease
hematologic	<ul style="list-style-type: none"> thrombocytopenia coagulopathies (congenital or acquired) <ul style="list-style-type: none"> hemophilia vWBD vitamin K deficiency due to

use of anticonvulsants during pregnancy
 failure to give vitamin K at birth
 antibiotic administration in infancy
 exclusive breast feeding
 anticonvulsants or herbal teas while nursing

vascular

hemangiomas
 vasculitis (e.g. Henoch)

gastrointestinal

celiac sprue

renal

nephritis
 with immune complexes (e.g. Goodp)
 without immune complexes

SIDS*

physical abuse

unintentional injury

infection

lung
 systemic

collagen vascular diseases

WG
 tuberous
 lymphangiomyomatosis
 lymphangiomyomatosis
 pulmonary-renal syndrome (=Fanconi's syndrome?)
 SLE

* The authors comment that "Factors not covered in the inclusion and exclusion criteria for acute IPH that are known risk factors for SIDS should be identified -- race, low birthweight, prematurity, cosleeping, or type of bedding." I would caution that many forensic pathologists would not make a diagnosis of SIDS if there is evidence of previous bleeding into the lungs. –JKR

Suggest an environmental assessment of the home.

Other comment by JKR: This publication is intended as a surveillance definition of idiopathic acute IPH. These criteria are designed for research and are not necessarily applicable in the setting of forensic autopsy diagnosis.

What is the significance of haemosiderin in the lungs of deceased infants? Forbes A and Acland P. Med Sci Law 2004; 44(4): 348-352. This is a literature review. It basically asks two questions:

(1) Does the published literature support the proposition that pulmonary siderophages in a SIDS case indicate previous episodes of asphyxia?

(2) Does the published literature support the proposition that an increased amount of pulmonary hemorrhage in a SIDS case indicates acute asphyxia?

As to both questions, the authors conclude that the answer is “no.”

As to hemosiderin, the authors briefly analyze the articles by Stewart et al. (1985), Becroft & Lockett (1997), Dorandeu et al. (1999), Hanzlick & Delaney (2000), Jackson & Gilliland (2000), and Schluckebier et al. (2002).

Four articles point toward hemosiderin as a suspicious finding -- the original findings by Becroft & Lockett in their seven suffocated infants, the conclusion of Hanzlick & Delaney that a high hemosiderin score suggests a cause of death other than SIDS, the finding of Dorandeu et al. that abused infants had a significantly higher hemosiderin score than SIDS infants, and the finding of Schluckebier et al. that all six of their high-hemosiderin-score infants were from suspicious circumstances.

These findings, however, are counterbalanced by other findings. Becroft & Lockett’s larger study failed to bear out an association with asphyxia. Most of Hanzlick & Delaney’s positive cases had an innocent explanation. Jackson & Gilliland did not find any association with asphyxia. Dorandeu et al. found positivity in 20% of their controls and negativity in 27% of their known abuse cases, indicating no predictive value. Half of Schluckebier’s positive cases had innocent explanations. In other words, as Forbes & Acland conclude, “The stain is nonspecific.” Their conclusion: “The available literature has very little supporting evidence for using pulmonary haemosiderin as grounds for suspicion of previous asphyxic abuse.”

As to pulmonary hemorrhage, everyone knows that pulmonary hemorrhage is a common finding in SIDS cases (47% in one study). But does an increased amount of hemorrhage indicate asphyxia? Yukawa et al. indicated “yes” in an article that quantified hemorrhage and found that increased amounts did correlate with respiratory obstruction. Berry, however, pointed out that this could be accounted for by the fact that increased hemorrhage tends to correlate with younger age. Becroft also found this, showing an association with age under 13 weeks. Hanzlick found no correlation between amount of hemorrhage and amount of hemosiderin. Taking these articles together, Forbes & Acland conclude that “significant pulmonary hemorrhage has not been shown to be specific for asphyxic death in infants.”

[Comment by JKR: Hemosiderin is nonspecific, certainly. In the articles marshalled by Forbes & Acland, a significant amount of pulmonary alveolar hemosiderin was found in 66 cases out of a total of 527 infants. Of the 66 positive cases, 26 had a history of past ALTE, asphyxia or physical abuse. The other 40 had either no history or a history of NICU treatment, mechanical ventilation, or congenital heart disease. The innocent cases outnumber the guilty. But in a given autopsy case, what if there is no innocent explanation?]

Pulmonary intra-alveolar siderophages in SIDS and suffocation: A San diego SIDS/SUDC research project report. Krous HF, Wixom C, Chadwick AE et al. Pediatric Developmental Pathology 2006; 9: 103-114. Did iron stains on the lungs of 91 SIDS and 29 suffocs (27 acci and 2 homi). Performed PS counts. No significant difference was found. According to Dr Greenbaum’s review in the Winter 2007 *Quarterly*, four so-called SIDS cases had >200 siderophages, and one of these had healing posterior rib fractures, another had a history of thrombocytopenia, and one was overage (9 months old). In other words, were these appropriate SIDS cases? Dr Greenbaum comments that this study “provides strong ev that the number of PS is not a reliable method for differentiating SIDS deaths from those caused by suffoc.” [Comment by JKR: No one ever claimed that it was a reliable method for doing that. It’s just one more thing to be taken into consideration.]

A comparison of pulmonary intra-alveolar hemorrhage in cases of sudden infant death due to SIDS in a safe sleep environment or to suffocation. Krous HF, Haas EA, Masoumi H, Chadwick AE, Stanley C. FSI 2007. This is hemorrhage, not siderophages. The amount of hemorrhage doesn’t independently distinguish between SIDS and suffocation. See above under “SIDS vs. suffocation.”

ALTE

The definition of an ALTE: “An episode that is frightening to the observer and that is characterized by some combination of apnea (central or occasionally obstructive), color change (usually cyanotic or pallid, but occasionally erythematous or plethoric), marked change in muscle tone (usually marked limpness), choking, or gagging.” NIH Consensus Development Conference on Infantile Apnea and Home Monitoring. Pediatrics 1987; 79: 292-299

Prolonged apnea and the SIDS: clinical and laboratory observations. Pediatrics 1972; 50: 646-654. Well-known paper finding that recurrent ALTE was a prodrome to SIDS. But, as noted by Berman in 1997 (q.v.), some of Steinschneider’s cases were the Waneta Hoyt suffocations.

Child abuse simulating “near-miss” sudden infant death syndrome. Berger D. J Pediatr 1979; 95: 554-556. Cited by Pitetti et al., below, for the proposition that “An ALTE may be a secondary manifestation of child abuse, specifically from a smothering attempt, an intentional poisoning, or shaken infant-impact syndrome.”

Postnatal medical and epidemiological risk factors for the SIDS. Damus K, Pakter J, Krongard E., et al. In: Harper R, Hoffman H, eds., *Sudden Infant Death Syndrome: Risk Factors and Basic Mechanisms*. New York: PMA Publishing; 1984: 187-201. Cited by Pitetti et al, 2002, below, for the proposition that “Reported estimates of the incidence of ALTEs in children vary between 0.5% and 3%.”

Early neonatal sudden infant death syndrome and near death of full term infants in maternity wards. Polberger S, Svenningsen N. Acta Paediatrica Scandinavica 1985; 74: 861-886. Cited by Pitetti et al, 2002, below, for the proposition that “Reported estimates of the incidence of ALTEs in children vary between 0.5% and 3%.”

Infantile Apnea and Home Monitoring: Report of a Consensus Development Conference. Bethesda, MD: US Department of Health and Human Services; 1986. NIH Publ. No. 87-2905. Consensus panel on infant apnea defined ALTE as an event that is characterized by some combination of apnea, color change, marked change in muscle tone, choking, or gagging, and is frightening to the observer.

Apparent life-threatening prolonged infant apnea in Saskatchewan. Sunkaran K, McKenna A, O’Donnell M., et al. Western Medical Journal 1989; 150: 293-295. Cited by Pitetti et al, 2002, below, for the proposition that “Reported estimates of the incidence of ALTEs in children vary between 0.5% and 3%.”

Parental administration of chemical agents: a cause of apparent life-threatening events. Hickson G, Altemeier WA, Martin ED, Campbell PW. Pediatrics 1989; 83: 772-776.

Diagnosis and management after life-threatening events in infants and young children who received cardiopulmonary resuscitation. Samuels MP, Poets CF, Noyes JP, Hartmann H, Hewertson J, Southall DP. Br Med J 1993; 306: 489-492. Cited in Their subsequent letter (1995, below) for the proposition that “In our series of 157 patients who had apparent life-threatening events who had received CPR, we found that child abuse was the cause of events in 25 of 77 (33%) of diagnosed cases.”

Child abuse and apparent life-threatening events. Samuels MP and Southall DP. Pediatrics 1996 Jul; 96(1): 167-168. “In response to your editorial by Doctors Little and Brooks, we also consider that it is a disservice to children if child abuse is not considered as part of the differential diagnosis of infants with ALTE. In our series of 157 patients who had ALTE, we found that child abuse was the cause of events in 25 of 77 (33%) diagnosed cases. Some cases of child abuse involved the fabrication of events, or interference with monitor data, while more serious abuse resulted from the intentional suffocation by a parent.” Describes the case of a 14 month-old with recurrent ALTE whose mother’s psychosocial history gave cause for concern and covert video was done with diagnostic results. The reference to Doctors Little and Brooks refers to “Accepting the unthinkable,” 1994. See under “SIDS versus Suffocation.”

Apparent life-threatening events. Brooks JG. Pediatric Reviews 1996; 17: 257-259. Cited by Pitetti et al, below, for the proposition that “ALTE is not considered a diagnosis, but is rather a description of an event. In as many as half of all events, no discernible cause will be found.”

Wrong turns in SIDS research. (commentary). Abraham B. Berman. Pediatrics 1997 Jan; 99(1): 119-121. Criticizing Steinschneider, 1972, on ALTE being a prodrome of SIDS, because Steinschneider’s ALTE cases were in fact suffocations. But see Pitetti, 2002, below, finding incidentally that SIDS was strongly associated with sibling ALTE. And see the 1987 NIH Consensus Development Conference on apnea saying that a minority of infants who die of SIDS may have had previous significant apneic episodes (possibly relying in Steinschneider’s work.)

Unnatural sudden infant death. Meadow R. Arch Dis Chil 1999; 80: 7-14. See under “SIDS versus suffocation.”

Analysis of missed cases of abusive head trauma. Jenny C, Hymel KP, Ritzen A, Reinert SE, Hay TC. JAMA 1999; 281: 621-626. See below under “Shaken.”

Otolaryngological manifestations in children presenting with ALTE. McMurray NS and Holinger ED. 1999. 7-15% of infants with ALTE go on to die of SIDS. In our series of 30 patients seen in consultation, 53% were found to have GERD, 40% laryngeal abnormality, 13% tracheal abnormality, 10% pharyngeal abnormality.

Functional magnetic resonance imaging of the brain in response to hypercapnea in congenital central hypoventilation syndrome (CCHS). American Journal of Respiratory and Critical Care Medicine. 1999; 159(3):

Sudden Infant Death Syndrome: Problems, Progress & Possibilities. Byard RW, Krous H., eds. New York: Oxford University Press, 2001. Suggests researching ALTE as a promising approach to studying the role of known risk factors in causing SIDS. Referring to Steinschneider’s work on ALTE as a prodrome of SIDS and the criminal-justice sequel to his cases. The 1986 NIH Consensus Development Conference on Apnea (Little et al., 1987a) to the effect that “a minority of infants who die of SIDS may have had previous apneic episodes.” Discusses monitoring in infants who have had one ALTE; if there isn’t another ALTE for one month, monitoring can safely be stopped because the recurrence risk goes away (recurrent ALTE’s always occur within a month). Other discussion in other chapters re MSBP etc.

Considering suffocatory abuse and Munchausen by proxy in the evaluation of children experiencing apparent life-threatening events and SIDS. Truman TL, Ayoub CC. (Tallahassee) Child maltreatment 2002 May; 7(2): 138-148. (See also under “MSBP” and “SIDS versus Suffocaton”) Retrospectively studied 138 young chil admitted over a 23 year period for recurrent ALTE, unexplained death, or SIDS. There were 35 deaths: 25 of the deaths were diagnosed as SIDS. Not all the deaths were autopsied. Retrospectively re-evaluating their charts and backgrounds for risk factors for abuse showed that of the SIDS deaths,

54% were not SIDS

37% had confirmed abuse or suspicious for abuse

Recommend that all admissions for ALTE, SUDS, SIDS, have an autopsy , involvement of child protection team, and death scene investigation.

Prevalence of retinal hemorrhages and child abuse in children who present with an apparent life-threatening event. Pitetti RD,. Maffei F, Chang K, Hickey R, Berger R, Pierce MC. Pediatrics 2002 Sep; 110(3): 557-562. See under “RH -- In General.” Prospective study of 128 ER admissions under 24 months with ALTE, covering a two-year study period. ALTE defined according to the 1986 NIH Consensus Development conference on apnea as “some combination of apnea, color change, marked change in muscle tone, choking, or gagging, and that are frightening to the observer.” Found child abuse in 3 pts and RH in

1. Found that 15 had a family history of SIDS, but the authors do not comment on this striking association. The final diagnoses in their 128 pts, which could well serve as a DDX for ALTE, were:

GERD	51
ALTE/apnea	38
Choking	11
Infection	6
Bronchiolitis	5
URI	5
Sz	4
Abuse	3
Swallowing dso	3
Breathholding	2

The authors point out that ALTE or similar nonspecific ER visits were precursors to a dx of AHT in 31% of Jenny's cases of missed AHT. Child abuse "can be easily missed." Gives a discussion of RH in nontrauma settings such as CPR. Says that routine dilated fundoscopy is a good noninvasive screening test for AHT in an ALTE population. Pupil dilation wd be contraindicated in pts with altered mental status bc it covers up signs of brain herniation. But they encountered no complications of the procedure. It does require the presence of a pediatric ophthalmologist, which they admit probably restricts its routine use as a screening test to academic centers.

QT dispersion in infants with ALTE syndrome. Goldhammer EI et al. (Haifa). Studied 89 ALTE infants and 18 controls. Found that QTc was significantly greater in the ALTE group. These were infants avg 2 ½ months old who were found limp.

Apparent life-threatening events in infants: high risk in the out-of-hospital environment. Stratton S, Taves A, Lewis R et al. Ann Emerg Med 2004; 46: 711-717. This is from Los Angeles. According to Trokel's review in the *Quarterly* for Autumn 2004, EMS transported 60 ALTE infants. Mean age 3 mos. Most of them were found by the hospital to have nothing wrong with them, but specific diagnoses were:

pneumonia	12%
GERD	10%
Sz	8%
sepsis	7%
URI	7%
Intracranial hemorrhage	3%

The reviewer notes that the two abused infants appeared well to the EMS rescuers and only detailed ER evaluation revealed the dx. One of them had activated the EMS system twice for ALTE.

Yield of diagnostic testing in infants who have had an apparent life-threatening event. Brand DA, Altman RL, Purtill K, Edwards KS. Pediatrics 2005 May; 115(4): 885-893. According to Dr William T. Basco's summary on MedScape, this was a retrospective chart review from DG on 3 years worth of infants consisting of 243 infants. ALTE was defined as "the presence of one or more of the following symptoms: breathing irregularity, color change, altered muscle tone, or altered mental status." Identified causes of ALTE included 33% infections, 38% GI including reflux, 13% neurological, and less than 3% each for airway issues, congenital malformations, and other. 16% had an unknown cause. In 49%, the ultimately diagnosed cause was suggested by the initial H&P, and 21% required no further testing. If the initial H&P did not suggest the diagnosis, then the most useful tests were eval for GE reflux, urine culture, neuroimaging, pneumogram, RSV screening, pertussis antigen, and WBC. Full sepsis evaluations and testing for metabolic disorders were not useful.

Clinical Report: Distinguishing sudden infant death syndrome from child abuse fatalities. AAP, NAME. Pediatrics 2006 Jul; 118(1): 421-427. Discusses history of ALTE as one of the indicators of possible

imposed suffocation. See Dr Keens' article, below, referring to the two schools of thought as to whether ALTE predicts anything.

Apparent life-threatening event or child abuse? (case report) Waseem M, Pinkert H. *Pediatric Emergency Care* 2006; 22: 245-246. This eleven month-old brought in by stepfather because he had an apneic episode. In the emergency room, he was lethargic. History compatible with ALTE. Fortunately, they decided to do a CT scan. This showed an SDH. Cautions that SBS is in the differential of ALTE, and a head CT should always be done.

Apparently life-threatening events in infant car safety seats. Tonkin SL, Vogel SA, Bennet L, Gunn AJ. *BMJ* 2006; 333: 1205-1206. See below under "Car seats."

Do all infants with ALTE need to be admitted? Claudius I and Keens T. *Pediatrics* 2007 Apr; 119(4): 679-683. Can ALTE be stratified into low-risk and high-risk types? Yes, provided you do it conservatively so as to be sure ALL the high-risk cases are identified (100% sensitivity). Prospectively administered a questionnaire to ER physicians to study all ALTE infants seen in the ER over a three-year period. Had 59 pts. They followed up each pt for three months, looking for sequelae that would indicate the infant should have been admitted. Such sequelae (my word, not the authors') include recurrence, death, SIDS, sepsis, pertussis, physical abuse, arrhythmia, metabolic disease, seizure, pericardial tamponade. The risk of these has to be weighed against the burdens of a hospital admission. The authors studied the literature for possible risk factors for serious sequelae, and found (1) family history of SIDS, (2) moderate prematurity, (3) previous ALTEs, (4) age, (5) URI sx, (6) child's color and tone during the ALTE, (7) duration of the ALTE, (8) interventions required, (9) appearance of the child in the ED, (10) suspicion of child abuse, (11) multiple ALTEs within 24 hours. The authors subsequently assigned each pt based on outcome followup to the category "hospitalization required" (HR) or "hospitalization not required" (HNR).

Found that the following two criteria were 100% predictive of need for hospitalization: (A) age under 30 days, and (B) multiple ALTE's. Pts who had one or both of these were eight (8) pts, all needing hospitalization because they turned out to have GERD, oxygen requirement, need for intubation, encephalitis, seizure, suspected nonaccidental trauma, or UTI.

Of the other 51 pts, 2 suffered subsequent ALTE's and 1 developed possible cardiomyopathy. Interestingly, none got SIDS.

The authors say that there have been two schools of thought about sequelae of ALTE. One, more or less the Steinschneider school, argues that ALTE can be a predictor of SIDS, abuse, or other serious sequelae. The other, the Hodgman-Hoppenbrouwers school, argues that there are no predictors of SIDS. [In my experience with SIDS autopsies, a past history of ALTE is extremely rare in SIDS cases.]

[Comment by JKR: Another study the authors might like to do retrospectively on the same patients' records would be to see if any of their 59 patients had known risk factors for either SIDS or child abuse. This article gives quite a different slant from those above.]

CENTRAL HYPOVENTILATION (ONDINE'S CURSE)

Beal SM, Recurrence of Sudden Unexpected Infant Death in a Family. In: *Sudden Infant Death Syndrome: Problems, Progress & Possibilities.* Byard RW and Krous HF. New York: Oxford University Press, 2001, p. 286. Rare disorder which may sometimes be familial, but not necessarily. Listen for a family history. Death in the neonatal period apparently is typical. Citing Haddad et al., *Congenital failure of automatic ventilation, gastrointestinal motility, and heart rate.* *Medicine*, 1978.

The role of beta-amyloid precursor protein staining in the neuropathologic evaluation of sudden infant death and in the initiation of clinical investigations of subsequent siblings. Byard RW, Blumbergs P, Kennedy JD, Riches KJ, Martin J, Thompson GN. *Am J Forens Med P* 2006 Dec; 27(4): 340-344. In this case report, they happened to discover BAPP positivity in the brain of a SIDS infant. This suggested previous episodes of hypoxia. They hypothesized a central mechanism. When a younger sibling was born,

they studied him and found that he had central apnea -- a familial disorder. See above under "SIDS Biology."

BRUGADA'S SYNDROME

Double-wave reentry as a mechanism of acceleration of ventricular tachycardia. J Brugada, L Boersma, C Kirchhof, P Brugada, M Havenith, HJ Wellens and M Allessie (Maastricht) *Circulation*. 1990;81 :1633 – 1643. These guys induced something called double-wave reentry in rabbit epicardium. It causes sudden cardiac death with VF or VTach.

Prolongation of the QT interval and the sudden infant death syndrome. Schwartz PJ, Stramba-Badiale M, Segantini A, Austoni P, Bosi G, Giorgetti R, Grancini F, Marni ED, Perticone F, Rosti D, Alice P. *N E J Med* 1998; 338: 1709-1714. An 18-year prospective study involving over 34,000 infants with day3/day 4 of life ECG's. In this cohort, 24 infants were diagnosed as SIDS. Review of the ECG's revealed that 50% of the SIDS victims had a rate-corrected QT interval >440 ms, as compared with 2.5% of the remaining cohort. [From Weese-Mayer et al., 2007, below]

Molecular autopsy of sudden cardiac death (SCD): The challenge of forensic pathologist to the complexity of genomics. (letter) Oliva A, Pascali VL, Hong K, Brugada R. *Am J Forens Med P* 2005 Dec; 26(4): 369. See above under "Sudden Unexpected Death in Childhood (SUDC)." An important letter.

Brugada syndrome masquerading as febrile seizures. Skinner JR, Chung S-K, Shelling AN, Crawford JR, McKenzie N, Pinnock R, French JK, Rees MI. *Pediatrics* 2007 May; 119(5): e1206. From the abstract: "Fever can precipitate ventricular tachycardia in adults with Brugada syndrome, but such a link has not been reported in children. A 21-month-old white girl presented repeatedly with decreased conscious level and seizures during fever. During a typical episode, rapid ventricular tachycardia was documented. The resting 12-lead electrocardiogram revealed a Brugada electrocardiogram signature. Resting electrocardiograms of the asymptomatic brother and mother were normal, but fever in the mother and pharmacologic stress with ajmaline in the brother revealed Brugada electrocardiogram features. Genetic testing revealed an *SCN5A* mutation in the affected family members." Now, hear this: "Sudden death syndromes are attributed partly to cardiac arrhythmia syndromes, in particular long QT syndrome and cardiomyopathic disorders. In Brugada syndrome, sudden unexpected death can occur as a result of ventricular fibrillation or rapid ventricular tachycardia. The 12-lead electrocardiogram (ECG) typically reveals an elevated ST segment in the anterior precordial leads with a right bundle branch block-like appearance. Approximately 10% to 30% of affected Brugada syndrome cases have been linked to mutations within *SCN5A*, a gene that codes for the α subunit of the voltage-gated sodium channel $Na_v1.5$ and is also associated with long QT syndrome type 3." The ECG's show a pathologic ST segment, in both the child and the mother.

Research review: Sudden infant death syndrome: review of implicated genetic factors. Weese-Mayer DE, Ackerman MJ, Marazita ML, Berry-Kravis EM. *Am J Med Genet Part A* 143a: 771-788 (2007). (See under "SIDS Genetics.) The Schwartz-QT hypothesis: "Cardiac channelopathies comprise a compendium of genetic heart rhythm disorders that are pursuant to defective cardiac channels. These heritable arrhythmia syndromes include congenital long QT syndrome (LQTS), Brugada syndrome (BrS), and catecholaminergic polymorphic ventricular tachycardia (CPVT). The hypothesis that abnormal cardiac repolarization and QT prolongation might account for a subset of SIDS cases was first suggested in 1976 (Maron et al., 1976; Schwartz, 1976). More than 20 years later, results from an 18-year prospective study involving over 34,000 infants with day3/day 4 of life ECG's lent clear support to the Schwartz-QT hypothesis (Schwartz et al., 1998). In this cohort, 24 infants were diagnosed as SIDS. Review of the ECG's revealed that 50% of the SIDS victims had a rate-corrected QT interval >440 ms, as compared with 2.5% of the remaining cohort."

CAR SEATS

Premature infants in car seats. Bull MJ, Stroup KB. Pediatrics 1985; 75: 336-339. Cited by AAP report (1996) for the proposition that semiupright car seats may be unsafe for preemies bc of apne-bradys.

Risk of hypoventilation in premature infants in car seats. Willett LD, Leuschen MP, Nelson LS, Nelson RM. J Pediatr 1986; 109: 245-248. Same.

Automotive restraint systems for premature infants. Bull MJ, Weber K, Stroup IKB. J Pediatr 1988; 112: 385-388. Same.

Ventilatory changes in convalescent infants positioned in car seats. Willett LD, Leuschen MP, Nelson LS, Nelson RM. J Pediatr 1989; 115: 451-455. Same.

Monitoring premature infants in car seats: implementing the American Academy of Pediatrics policy in a community hospital. Pediatrics 1993; 91: 1137-1141. Same.

Safe transportation of premature and low birth weight infants. AAP Committee on Injury and Poison Prevention and Committee on Fetus and Newborn. Pediatrics 1996 May; 97(5): 758-760. This is all about asphyxia. Cites Bull et al., 1985, 1988; Willett et al., 1986, 1989; Bass et al., 1993 for the following propositions:

1. "Current information suggests that each preterm infant born at less than 37 weeks gestation should have a period of observation in a car safety seat before hospital discharge to monitor for possible apnea, bradycardia, or oxygen desaturation..."
2. "Families should minimize travel for infants at risk of respiratory compromise."
3. "Infants with documented desaturation, apnea, or bradycardia in a semiupright position should travel in a supine or prone position in an alternative safety device. The use of other upright equipment, including infant swings, infant seats, and infant carriers, should be avoided."

Fall and suffocation injuries associated with in-home use of car seats and baby carriers. Pollack-Nelson C. Pediatric Emergency Care 2000 Apr; 16(2): 77-79. This review of NEISS data for 1997, infants 6 months and under, plus death certificates in the CPSC's death certificate files. Found that in 1997, 8700 infants suffered fall injuries while in car seats or baby carriers, about 1/3 of which resulted from the seat being placed on an elevated surface. There were 15 suffocation deaths resulting from seat overturn on soft surfaces. Conclusion: "Manufacturers have an obligation to pursue design alternatives that will reduce the likelihood of seat overturn."

Respiratory instability of term and near-term healthy newborn infants in car safety seats. Merchant JR, Worwa C, Porter S, Coleman JM, de Regnier RA. Pediatrics 2001 Sep; 108(3): 647-652. Premature infants who are discharged from NICU's are known to be at increased risk for apne-brady's when in an upright position. Also, small infants do not fit securely in car seats. The AAP recommends a period of observation in placing any premature infant in a car seat (premature here = born at <37 weeks). It is not clear whether this recommendation should apply to the minimally preterm infants (born at 35-36 weeks) who are healthy at birth. Took fifty healthy preterm neonates and fifty term neonates from the newborn nursery and monitored them in car seats. Found that 24% of the preterms and 4% of the terms did not fit securely in the car seat. Mean oxygen saturation declined significantly in both preterm and term (down to 94%). Seven infants (3 preterm and 4 term) desaturated to below 90%. 12% of the preterms had apne-bradys, while none of the terms did. Conclusion: car seats definitely cause desaturation. During the first months of life they should be used only for travel. "Our data support the current AAP recommendations that all infants who are born at less than 37 weeks' gestation, including those who are admitted to level I community hospitals, be observed for respiratory instability and secure fit in their car seats before hospital discharge. Because lowering of oxygen saturation values was seen uniformly in all newborn infants, car seats should be used only for travel, and travel should be minimized during the first months of life."

Commented on in Respiratory instability of term and near-term healthy newborn infants in car safety seats. Ackley HA. Pediatrics 2002 Apr; 109(4): 713 and Oxygen desaturation in term infants in car safety seats. Bass JL, Bull M. Pediatrics 2002 Aug; 110(2 pt 1): 401-402.

Burns from hot car seats. Photo given. Reece & Ludwig, 2d ed. at 41, 188.

Head injuries in infants: the risks of bouncy chairs and car seats. Wickham T and Abrahamson E. (London) Arch Dis Chil 2002; 86: 168-169. This British prospective study of 131 infants mean age 6 mos presenting to ER with a hx of head injury. 114 pts had skull series, not reported if any had CT scans. The histories were:

Fell off a surface	52
Fell out of a bouncy chair or car seat	17
Fell off a push chair	16
Fell over	10
Dropped	15

Categorized as to whether landed on a hard surface or not, whether the source was on an elevated surface or not. Apparently sx were rare, a few skull fxx, no serious injuries. Commented on in Head injuries in infant bouncy chairs and car seats. Wyatt J, Bellis F (UK) Emerg Med J 2002 Sep; 19(5): 436. See also "Head Injury -- Fall vs. Inflicted," below.

Effects of child seats on the cardiorespiratory function of newborns. Nagase H, Yonatan M, Ustani Y, Nakamura H. (Kobe) Pediatrics International 2002 Feb; 44(1): 280-283. Performed sleep apnea monitoring experiments on 15 healthy term newborns in two kinds of Japanese car seats (chair type and bed type). Also measured heart rate, chest impedance, nasal air flow, and O2 saturation. Also with controls placed supine on a nursery cot. Monitored for episodes of oxygen desaturation below 95% for longer than 10s or below 90% and longer than 10s over a 30 minute observation period. Newborns placed in the chair-shaped car seat had significantly more episodes of desaturation. Moderate desaturation was observed in four of 15 infants in the chair-shaped seat and none in the bed-shaped or on the cot. Mild desaturations were observed an average of seven times in 30 minutes in 15 infants placed in the chair-shaped seat, compared to only one in each of two infants in the bed-shaped and none in the cot. Conclusion: "The results suggest that prior to discharge the degree of oxygen desaturation that occurs when an infant is placed in a chair-style car seat should be checked."

Effect of head support on oxygen saturation in preterm infants restrained in a car seat. Dollberg S, Yacov G, Mimouni F, Ashbel G. (Tel Aviv) Am J Perin 2002 Apr; 19(3): 115-118. We prospectively tested the hypothesis that prevention of lateral movement of the head, using a specially designed head support apparatus, would prevent oxygen desaturation in preterm infants restrained in car seats. 34-35 weekers. Positioned supine for 20 minutes, sitting for 20 minutes in regular car seat, and sitting for 20 minutes in car seat fitted with the special head support. Measured the time spent with oxygen saturation below 96%, 94%, 92%, and 90%. Found no significant differences.

AAP Medical Library, 2002. <http://www.medem.com/medlb/> Car seats for children with special needs -- premature babies. "... Some babies who were born prematurely have breathing problems when they sit semi-reclined in a car safety seat. Make sure that the hospital staff observes and monitors your baby in a car safety seat before going home. Your baby may need to use a car bed if he has any of the following while in a car safety seat:

- a decrease in oxygen levels
- slow heart rate
- apnea"

Product recalls. Infant car seats/ carriers. Child Health Alert (United States), June 2002

Early NICU discharge of very low birth weight infants: a critical review and analysis. Merritt TA, Pillers D, Prows SL. *Semin Neon* 2003 Apr; 8(2): 95-115. Cardiopulmonary stability while in a car seat affects the time of discharge.

Simple car seat insert to prevent upper airway narrowing in preterm infants: a pilot study. Tonkin SL, McIntosh CG, Hadden W, Dakin C, Rowley S, Gunn AJ (New Zealand). *Pediatrics* 2003 Oct; 112(4): 907-913. These researchers tested a foam rubber seat insert that provided a pocket for the newborn's head so that it could tilt backward and provide less anterior flexion-compression of the upper airway. They tested it on 17 premature newborns being discharged home. Found that it reduced the frequency of desaturations <85% from 3.5 episodes to 1.5 episodes and bradycardia <90 bpm from 1 to 0.1 episodes.

Big head, little body syndrome: what EMS providers need to know. De Boer SL, Seaver M. *Emerg Med Serv* 2004 Jul; 33(7): 47-48, 50-52. Effects of this syndrome on rescue considerations in auto accidents, drowning prevention, airway management, burns, cervical spine stabilization, and car seats.

Car-seat test. Stein N. *Pediatrics* 2004 May; 113(5): 1469.

Sudden Infant Death Syndrome risk factors with regards to sleep position, sleep surface, and co-sleeping. Alexander RT, Radisch D. *JFS* 2005 Jan; 50(1): 147-151. Does not refer to car seats, but found that out of 102 infants, only 2% were sleeping in "other" than bed, crib, sofa, etc. when they died. Indicating that death in car seat must be very uncommon.

Selecting and using the most appropriate car safety seats for growing children: guidelines for counselling parents. AAP Committee on Injury and Poisoning. *Pediatrics* 2002 Mar; 109(3): 550-553. Does not discuss flipping over risk. Does mention need for screening preemies for desaturation.

Clinical Report: Distinguishing sudden infant death syndrome from child abuse fatalities. AAP, NAME. *Pediatrics* 2006 Jul; 118(1): 421-427.

Apparently life-threatening events in infant car safety seats. Tonkin SL, Vogel Sa, Bennet L, Gunn AJ. *BMJ* 2006; 333: 1205-1206. According to Dr Rivara's review in the Spring 2007 *Quarterly*, these British pediatricians run a referral clinic for victims of ALTE. Out of 43 patients, 9 had suffered an ALTE while in a car seat. Of these nine, the median age was five weeks. Only one was premature. The clinicians noted that some of the infants had their necks flex forward while in the car seats. None of them suffered any further trouble, and none of them died. Dr Rivara questions the value of the study, because it's not clear whether their being in car seats had anything to do with their history of ALTE.

INFANT BOTULISM

Infant botulism acquired from household dust presenting as sudden infant death syndrome. Nevas M, Lindstrom M, Virtanen A et al. *J Clin Microb* 2004; 43: 511-513.

Clinical mimics of infant botulism. Francisco AMO, Arnon SS. *Pediatrics* 2007 Apr; 119(4): 826-828. Studied the 681 pts who received Human Botulism Immune Globulin. Found that about 5% of them did not have botulism. Instead, they had spinal muscle atrophy, metabolic disorder, infectious diseases, and miscellaneous conditions. See below.

Ingested spores of *C. botulinum* or *C. butyricum* or *C. baratii* colonize the colon and produce botulin in the lumen of the colon. This blocks the release of acetylcholine at the NM junction and other cholinergic circuits. Results in constip, lethargy, poor feeding, gen weakness, decreased head control, hypotonia, diminished reflexes, hypoventilation, and cranial nerve palsies. Cardinal features are symmetrical bulbar palsies, such as ptosis, sluggish pupillary light response, ophthalmoplegia, poor suck, decreased gag reflex, difficulty swallowing, expressionless face. [Comment by JKR: How could this frightening spectacle be mistaken for SIDS?]

As to the mimics, they were as follows:

Metabolic dsos

GA1, maple syrup urine dis, Leigh's syndrome, succinic semialdehyde dehydrogenase deficiency, mitochondrial dso

Spinal muscular atrophy

Infectious

encephalitis, RSV bronchiolitis, viremia

Miscellaneous

Guillain-Barre syndrome, neuroblastoma with Lambert-Eaton syndrome, cerebral atrophy, CVA, spinal epidural hematoma, diaphragmatic paralysis, demyelinating disease

RECURRENCE RISK

Population-based recurrence risk of sudden infant death syndrome compared with other infant and fetal deaths. Oyen N, Skjaerven R, Irgens LM. *Am J Epidemiol.* 1996; 144: 300–305. Cited along with Levene, Bacon, 2004 by the new 2005 AAP Policy Statement as finding that the SIDS recurrence risk in a family is 2 – 6%.

Sudden unexpected death and covert homicide in infancy. Levene S, Bacon CJ. *Arch Dis Child.* 2004; 89: 443–447. Cited along with Oyen et al., 1996 by the AAP 2005 Policy Statement as finding that the SIDS recurrence risk in a family is 2 – 6%.

AAP Task Force on SIDS. Policy Statement: The changing concept of SIDS: Diagnostic coding shifts, controversies regarding sleeping environment, and new variables to consider in reducing risk. *Pediatrics* 2005 Nov; 116(5): 1245-1255. Refers in the past tense to the work of Southall and Meadow (1997, 1999, 2002) saying that the suspicion of foul play should be increased if SIDS recurs in a family. However, cites Carpenter's conclusion that a SIDS recurrence has an 87% chance of being a natural death. (Carpenter RG, Waite A, Coombs RC, et al. Repeat sudden unexpected and unexplained infant deaths: natural or unnatural? *Lancet.* 2005;365 :29–35). The risk of covert homicide (6 – 10%) and the risk of recurrent SIDS (2 – 6%) are in the same range. "Therefore, the task force supports the position that the vast majority of either initial or second sudden unexpected infant deaths within a family seem to be natural rather than attributable to abuse, neglect, or homicide." However, see Gornall, 2006, above under "SIDS versus suffocation."

Clinical Report: Distinguishing sudden infant death syndrome from child abuse fatalities. AAP, NAME. *Pediatrics* 2006 Jul; 118(1): 421-427. Says that the risk of recurrence is controversial. Once the death has been thoroughly evaluated, the parents should be told that the risk of SIDS in subsequent children is not likely increased.

VACCINATION

Task force on SIDS. The changing concept of sudden infant death syndrome: diagnostic coding shifts, controversies regarding the sleeping environment, and new variables to consider in reducing risk. *Pediatrics* 2005; 116: 1245-1255. Cited by Fern Hauck and Carl Hunt in their 2006 review article in *Can Med Assoc J* (see above under "SIDS Biology) for the proposition that "among immunized infants, no

temporal relation between vaccine administration and death has been identified. Parents should be reassured that immunization does not present a risk for SIDS.”

DTP vaccination or shaken baby syndrome? The role of irresponsible medical expert testimony in creating a false connection. Chadwick DL, Parrish R. <http://www.dontshake.org/sbs-fal00dtp.html> 2000

Public opponents of vaccination: a case study. Vaccine 2003; 21: 4700-4703. Dr Chadwick reviews this article in the Winter 2007 *Quarterly*. It's about a certain Vera Scheibner, an Australian opponent of vaccination who thinks vaccination causes subdural hematomas, among other things.

Do immunisations reduce the risk for SIDS? A meta-analysis. Vennemann MMT, Hoffgen M, Bajanowski T, Hense H-W, Mitchell EA. (Munster) Vaccine 2007. Studied nine published case-control studies. Conclusions: “Immunisations are associated with a halving of the risk of SIDS. There are biological reasons why this association may be causal, but other factors, such as the healthy vaccinee effect, may be important. Immunisations should be part of the SIDS prevention campaigns.” What is the healthy vaccinee effect? It is the fact that immunization is avoided in ill or febrile infants; therefore, infants who get vaccinated are healthy. Conclusion: “Parents can be reassured that immunisation with vaccines on the current schedule, particularly DTP vaccine, does not cause SIDS.” The protective effect survived multivariate analysis to exclude the confounding variables of lower-class status, smoking mothers, and intention not to breastfeed.

SMOKING

(see above under “SIDS Biology”)

Surgeon general's speech of June 27th, 2006:

<http://www.surgeongeneral.gov/news/speeches/06272006a.html> at a press conference launching his new report entitled, “The Health Consequences of Involuntary Exposure to Tobacco Smoke.” The report is posted at <http://www.surgeongeneral.gov/library/secondhandsmoke/> His speech refers to the 1986 report on secondhand smoke. States that the scientific community has reached consensus on the point that secondhand smoke harms people's health. Draws our attention to several conclusions he has reached:

1. “Secondhand smoke exposure causes heart disease and lung cancer in adults and sudden infant death syndrome and respiratory problems in children.” ...

He expands on the first conclusion by saying that secondhand smoke contains 50 carcinogens and 250 toxic chemicals. Under “SIDS,” he has this to say:

“In an important new finding, we have determined that secondhand smoke is a cause of SIDS. Infants who die from SIDS tend to have higher concentration of nicotine in their lungs and higher levels of cotinine (a biological marker for secondhand smoke exposure) than infants who die from other causes. We have also found that infants who are exposed to secondhand smoke after birth are also at increased risk of dying of SIDS. In addition, babies of nonsmoking women who are exposed to secondhand smoke during pregnancy are at risk for a small reduction in birth weight. Chemicals in secondhand smoke appear to affect the brain in ways that interfere with its regulation of infants' breathing.”

NEOMEMBRANES

See also “Shaken” and “Dura mater & neomembranes,” below.

Subdural neomembranes and sudden infant death syndrome. Rogers CB, Itabashi HH, Tomiyasu U, Heuser ET. JFS 1998 Mar; 43(2): 375-376. Cranial dura maters of 36 consecutive infants with SIDS and 16 control infants were examined microscopically to determine if subdural neomembranes are associated with SIDS. Found neomembranes in 31% of SIDS and 13% of controls. The overall prevalence was 25%. “In all but two cases, birth trauma could be excluded as a cause by aging the neomembranes histologically.” No association was found with type of delivery.

Natural history of chronic SDH. Lee KS (Korea). Brain Injury 2004; 18(4): 351-358. Chronic SDH has two origins -- one from subdural hygromas and the other from acute SDH's. It occurs only in pts with a suitable premorbid condition, i.e. sufficient potential subdural space (PSS). In unresolved subdural hygromas, proliferation of dural border cells produces the neomembrane. Unresolved hygromas become chronic SDH's by repeated microhemorrhages from fragile new vessels, which were grown into the neomembrane. When the PSS is sufficient, acute SDH's may become chronic SDHs. Chronic SDH's enlarge when rebleeding exceeds absorption and they become symptomatic. When the neomembrane is matured, the neocapillary is no longer fragile. If absorption exceeds rebleeding, the hematoma will disappear. Maturation of the neomembrane and stabilization of the neovaasculature eventually result in spontaneous resolution. The fate of a chronic SDH depends on the premorbid status, the dynamics of absorption-expansion, and the maturation of the neomembrane.

Intracranial hemorrhage in asymptomatic neonates: prevalence on MR images and relationship to obstetric and neonatal risk factors. Looney CB, Smith JK, Merck LH, Wolfe HM, Chescheir NC, Hamer RN, Gilmore JH. Radiology 2006 Dec; They imaged 88 asymptomatic term neonates with a 3T MRI at mean age 20 days +/- 7. 65 vag deliv, 23 c/s. Found 16 SDH, 2 SAH, 6 ICH. Seven infants had two or more types of hemorrhages. All the ones with ICH were vag del, but this was not associated with prolonged labor or traumatic birth or assisted birth. Conclusion: Asymptomatic ICH fol vag birth in term neons appears to be common, with a prev of 26% in this study.

CERVICAL SPINE / SPINAL CORD

(Some courtesy of Mary Case MD, presentation at the second national SBS conference, 1998. See handout in file. Dr. Case after performing posterior neck dissections in 50 infants both shaken and nonshaken, found that "both shaken and control children frequently have spinal epidural hemorrhage, and no relationship could be established between shaking and the hemorrhages." Further that "The mechanism of **grasping a child by the head** by placing the hand on the child's head or neck and then violently shaking the child's body...may be suspected if petechial hemorrhages are found on the skin of the face on the cheeks or ears or on the neck along with a cervical cord and lower medulla contusion." Atlanto-occipital dislocation is more common in children than adults. Discusses the anatomy of the infant vertebral column. Discusses SCIWORA. Discusses other types of vertebral column injuries. "In conclusion, spinal cord injury is not common in child abuse and probably occurs in fewer than 3% of cases." Also points out that in brain death, the upper cervical spinal cord may have hemorrhagic infarction caused by occlusion of arachnoid vessels by floating fragments of dissolved cerebellum.

Sudden death related to spinal injury. Towbin A. Lancet 1967; 2: 940-942. He observed epidural hemorrhages in the cervical spines of five children who had died sudden unexpected death. Caffey (1974) cites this article as support for his (Caffey's) hypothesis that manual whiplash shaking of the head may be a major causal factor in sudden unexplained infant deaths ("cot" or "crib" death) associated with epidural hemorrhages of the cervical spine as found by Towbin.

Spinal injury related to the syndrome of sudden death ("crib death") in infants. Towbin A. Am J Clin P 1968; 49(4): 562-567.

Spine and spinal cord trauma in the battered child syndrome. Swischuk LE. Radiology 1969; 92: 733. Cited by Caffey (1974), 398, as follows: "Fractures of the spine with local injuries to the spinal cord of one infant were attributed by Swischuk to manual whiplash shaking."

"Spinal injury" and sudden death -- a second look. Harris LS and Adelson L. Am J Clin Path 1969; 52(3): 289-295. 18 out of 19 SUDS infants had epidural hemorrhage in the neck, greater in the lower neck. Concluded to be antemortem but nontraumatic. Refuting the above papers by Towbin.

Pediatric neck injuries. Hill SA, Miller CA, Kosnik EJ et al. J Neuros 1984; 60: 700-706. Cited by Wang et al. (J Pede Surg 2001, see under "Fall vs Inflicted") for the proposition that pediatric spinal cord injury is a rare entity, affecting 18/million/year. Radiographic evaluation of the cervical spine is mandatory in fall victims bc serious conseq.

The infant whiplash-shake injury syndrome. Hadley MN, Sonntag VHK, Rekate HL, Murphy A. Neurosurgery 1989; 24: 536-540. Reported autopsies on 6 shaken babies. 4/6 had cervical subdural blood, 4/6 had cervical cord contusions. Mary Case comments that cranial subdural blood would be expected to drain down into the spinal area, in other words is an artifact. Cited by Suzanne Starling in her chapter on AHT in the third edition of Giardino & Alexander, *Child Maltreatment* (2005) for the proposition that "Other researchers have refuted the theory that impact is necessary to produce the symptoms seen in shaking, citing cases with no evidence of impact and using more sensitive detection measures." (p. 41)

Isolated spinal cord injury as a presentation of child abuse. Piatt JH. Pediatrics 1995; 96: 780-782. Describes a 15 month old with acute quadriplegia after history of falling off a couch. Had multiple bruises and a healed fx of the clavicle. Cervical hematomyelia by MRI.

Isolated cord injury in child abuse. (letter). Parrish R. Pediatric Trauma and Forensic Newsletter 1996 Jan. Upper cervical cord and caudal medulla contusion with vitreous hemorrhage caused by holding the child's head and shaking body. 2 month old infant.

Pediatric cervical spine injury: a three year experience. Givens TG et al. J Trauma 1996; 41: 310-314.

Cervical spine MRI in abused infants Feldman KW, Weinberger E, Milstein JM, Fligner CL. Child Abuse & Neglect 1997; 21(2): 199-205.

12 children with inflicted head injury underwent cervical spine MRI at Seattle CHMC: 8 were done pre-mortem and 4 were done postmortem. **None of the MRI scans showed any evidence of cervical cord injury** or blood around the cord. Five infants died and were autopsied. These had: 1 cervical SDH continuous with cranial SDH, 3 had cervical SAH associated with diffuse cranial SAH. **All five cords were negative for cord injury at autopsy.** Found: it was not productive to use MRI to look for cord injury unless there was clinical evidence of cord injury. Comments that one would expect cord injuries to be more frequent in SBS **but this has not been observed**, possibly because cord injury requires a sharply localized subluxation.

[Hangman's fracture] Pediatr Radiol 1997; 27: 776

Cervical spine injury in child abuse: report of two cases. Rooks VJ, Sisler C, and Burton B. Pediatr Radiol 1998 Mar; 28(3): 193-195. Rarely been reported. We found these two lower c-spine fx-dislocs in two twin girls who were abused. Should do routine c-spine x rays in suspected child abuse. In Child Abuse Quarterly for October 1998, Wilbur Smith comments that (1) this shows the propensity for repeated abuse in the same family, (2) C-spine injuries in young infants are easily overlooked bc of lack of specific signs, (3) C-spine injuries occur in 2% of most large series of abusive head injury; this is surprisingly low.

Neck and cervical spine injuries in SBS and accidental head trauma. Research presentation at the Second National SBS Conference, 1998. Kathleen M. Dully, MD, CDR, MC, US Navy, Department of Emergency Medicine, Naval Hospital, Camp Pendleton 92055-5191 (abstract in file)

Autopsy results were presented of 46 children admitted with intracranial bleeding but no skull fx or EDH. 30 SBS and 16 MVA. Autopsy results were used because they already knew that even when there is known myelopathy, imaging only shows abn in 40%; in other words, 60% are SCIWORA. So this paper used autopsy results.

Autopsy results:

	<u>SBS</u>	<u>MVA</u>
Soft tissue hemorrhage	13%	63%
c-spine bone injury or distraction	0%	8%
epidural, subdural, or subarachnoid blood	20%	44%

parenchymal stem or cord injury	6%	50%
stem/cord lac or crush	0%	31%

Spinal cord injury in child abuse by shaking. A six-year prospective autopsy study of posterior neck dissections in children under 3 including a control group, presented at the Second National SBS Conference, 1998. Mary E. S. Case, MD, Michael Graham MD, and Joan E Wood MD. Note CC# 96-8501. See Geddes, 2001, below.

Pediatric cervical spine injuries: report of 102 cases and review of the literature. Eleraky MA, Nicholas T, Adams M, Rekate HL, Sonntag V. J Neuros 2000 Jan; 92 (1 Suppl.): 12-17. (have abstract) Reviewed by Ken Feldman in Child Abuse Quarterly for July 2000. A retrospective review of ten years' pediatric spinal cord injury material not stratified by age except as to 0-9 and 10-16. 82% had vertebral fractures or subluxations, 18% had SCIWORA. In the younger age group, falls and MVA's accounted for 72% of the cases. The younger group had more upper-spine injuries, such as C1 or C2 subluxations. 39% in this group had subluxations and 29% fracture-subluxations. In the older group 80% had fracture-subluxations. There were no cases of delayed onset of symptoms or delayed recognition. Apparently there were no documented abuse cases. Ken Feldman comments that cervical cord injury is rare in child abuse.

Characteristics of pediatric cervical spine injuries. Kokoska ER, Keller MS, Rallo MC, Weber TR. J Ped Surg 2001; 36: 100-105. Data from the National Pediatric Trauma Registry 1994-1999 on all blunt trauma pts with c-fx, disloc, or SCIWORA. N=408. 44% MVA, 14% auto vs, ped, 16% sports. Younger children more often sustained high cervica (C1-C4) and had more dislocations and cord injuries, while older chil had more fractures. No children with dislocations had neurologic sequelae(!) and 83% of those with fractures had no seq.

Pediatric cervical spine injuries: defining the cause. Patel JC, Tepas JJ, Mollitt DL, Pieper P. J Ped Surg 2001; 36: 373-376. Data from the National Pediatric Trauma Registry, 1988-1998. (have) n=1098. 42% caused by automobile collisions, some from falls, some penetrating trauma, No cases of child abuse. 49% had SCIWORA. Often in infants their vertebrae do not break but merely sublux because of large head, soft ligaments. See Ken Feldman's review in Child Abuse Quarterly, July 2001.

Neuropathology of inflicted head injury in children. I. Patterns of brain damage. Geddes JF, Hackshaw AK, Vowles GH, Nichols CD, Whitwell HL. Brain 2001 Jul; 124 (Pt. 7): 1290-1298. From the abstract: autopsy brains on 37 infants and 16 other children aged 13 mos to 8 years, all with fatal AHT. 36% had skull fx, 72% had SDH, 71% had RH, 82% had death due to increased ICP/ cerebral edema. By histology 77% had severe HIE, and 40% had axonal injury caused by ischemia, but only 3 cases (5%) had DAI in the sense of diffuse traumatic axonal injury. But 11 **infants** (and **only** infants) had **localized TAI** localized to the **caudal medulla or the cervical spinal cord**. See under "Shaken." Note that localized TAI is different from cord contusion; therefore this study does not contradict the results of Ken Feldman et al., above. -- JKR

Pediatric traumatic atlanto-occipital dislocation: five cases and a review. Kenter K, Worley G, Griffin T, Fitch RD. J Ped Orthop 2001; 21: 585-589. From Daniel R. Cooperman's review in *Child Abuse Quarterly*: a rare type of injury. These were all automobile-related injuries to older children. Unrestrained passenger or auto vs ped.

Unusual upper cervical fracture in a 1-year-old girl. Mousny M, Saint-Martin C, Danse E, Roumbouts J-J. J Ped Orthop 2001; 21: 590-593. Front-end auto vs. fixed object, child in car seat facing forward, head snapped forward, resulting in fx of the body of C-2. The usual C-2 fx is between the odontoid and the body.

Cervical spine injuries in children: A review of 103 patients treated consecutively at a level 1 pediatric trauma center. Brown RL, Brunn MA, Garcia VF. J Ped Surg 2001; 36: 1107-1114.

52%	MVA
27%	sports

15% falls
3% child abuse

All the child abuse were under one year. Abuse constituted 75% of the cases under one year of age. All these infant victims had SCIWORA -- no radiographic abnormality seen.

Pediatric spine and spinal cord injury after inflicted trauma. Ghatan S, Ellenbogen RG. (Seattle) *Neuros Clin N Amer* 2002 Apr; 13(2): 227-233. Rare sequelae of intentional trauma. Easily overlooked. Probably underreported. But the upper cervical spine and brain stem “may significantly contribute to the major morbidity, mortality, and neuropathology in shaken infants.” These structures are highly vulnerable to shaking injury on theoretical grounds, and outcomes with cognitive delay due to hypoxic damage (global brain injury) could have occurred secondary to brainstem and high cervical cord injury.

Hangman’s fracture caused by suspected child abuse: a case report. Ranjith RK, Mullett JH, Burke TE. (Ireland) *J Pediatr Orthop B* 2002 Oct; 11(4): 329-332. Normal variation in radiographic appearance is confusing. This is only the second case reported. 23 mos f 5 d hx irrit reluctant to move her neck. Neck stiffness and supported her neck with her hands. Managed conservatively with a cervical collar. The father denied abuse, no confession, not clear how they did abuse. Says both plain films, CT, and MRI are required for the diagnosis.

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/> Zimmerman gives a brief review of the state of the art of radiography of the cervical spine in child abuse as of 2002 (p. 87). He first refers to Geddes finding (2001) of cervical spine injury by pathology in 11 cases, “Thus, this may be a site of injury that produces central apnea and subsequent hypoxic/ischemic brain injury.” (p. 87) [The “unified hypothesis” --JKR] Hadley et al (1989) found injuries of the cervicomedullary junction in 5/6 SBS cases by clinicopathologic study, consisting of SDHs, EDHs, or cord contusions. But Ken Feldman doing MRI (1997) found NO evidence of cervical cord injury or SDH in 12 SBS infants, but did find diffuse SAH of the cord in 1/5 fatal cases by autopsy.

CARDIAC (including STRESS CARDIOMYOPATHY and COMMOTIO CORDIS)

See also under “Unclassifiable.”

See also the web site of the Sudden Arrhythmia Death Syndrome (SADS): <http://www.sads.org>

See also under “Drown,” below.

Human stress cardiomyopathy: myocardial lesions in victims of homicidal assaults without internal injuries. Cebelin MS, Hirsch CS. *Human Pathology* 1980 Mar; 11(2): 123-132. Reviewing autopsies over a 30-year period, they found 15 cases of individuals who died as a direct result of physical assault without sustaining any internal injuries. In 12/15 they found myofibrillar degeneration of the heart, “comparable to lesions described in stressed animal experiments.” Consider that this represents “stress cardiomyopathy” due to stress catecholamines. See Zumwalt & Hirsch, below.

[Traumatic rhabdomyolysis] Schwengel & Ludwig, *Pediatric Emergency Care* 1985; 1: 4. Mukerji & Siegel, *AJR* 1987; 148: 1203. (References provided by Dr. Reece.)

Pathology of fatal child abuse and neglect. Zumwalt RE and Hirsch CS. In: Helfer RE and Kempe RS, *The battered child*, 4th ed., Chicago: University of Chicago Press, 1987, pp. 270-271 (Citing “myofibrillar degeneration” reported by Cebelin and Hirsch, 1980, which can be reproduced in animal models by the infusion of catecholamines, and elevated levels of CK reportedly found in the pericardial fluid of adults dying of violence. “Potentially difficult problems of autopsy interpretation arise when injuries are

restricted to the soft tissues and there is no cranial or visceral trauma that helps to explain the fatal mechanism. Such victims are virtually 'beaten to death,' but we ordinarily cannot provide an unequivocal, structurally demonstrable translation of the lethal pathophysiology. Slowly accumulating hemorrhage into traumatized soft tissues is usually insufficient in amount to produce exsanguination, and the slight-to-moderate pulmonary fat embolism that inevitably results from crushing adipose tissue has no important physiological effect. We believe that the cumulative burden of painful injuries and psychological stress kills because of a responsive overproduction of catecholamines with resulting hormonally mediated myocardial injury."

Age-related changes in normal human hearts during the first 10 decades of life. Part I: Growth: A quantitative study of 200 specimens from subjects from birth to 19 years old. Scholz DG, Kitzman DW, Hagen PT, Ilstrup DM, Edwards WD. *May Clin Proc* 1988; 63(2): 126-136. Gives normal heart weights.

Case report: nonfracture-associated fatal fat embolism in a case of child abuse. Nichols GR, Corey TS, Davis GJ. *JFSCA* 1990 Mar; 35(2): 493-499. 2 year old.

Hypovolemic shock in a child as a consequence of corporal punishment. Eichelberger SP, Beal DW, May RB. *Pediatrics* 1991 Apr; 87(4): 493-499.

Pathology of fatal abuse. Kirschner RH and Wilson H. In: Reece RM and Ludwig S., eds. *Child abuse...*, 2d ed. (2001), pp. 495-497. "Obvious signs of physical abuse or neglect but no identifiable 'fatal' injuries." "Perhaps even more difficult is the child abuse case that involves significant soft-tissue injuries, usually inflicted almost immediately before death, where autopsy reveals no anatomic cause of death... Some children may show extensive subcutaneous hemorrhage from a severe beating or beatings, with injuries primarily to the back, buttocks, and legs, and none of significant head injury... The common feature in all such cases is severe physical and psychological stress related to pain... Several proposed mechanisms may lead to death in these cases. ...stress cardiomyopathy as the mechanism of death... catecholamines..." (citing Cebelin & Hirsch and giving a photomicrograph which shows focal myocyte dropout with lymphoplasmacytic infiltrate in a chronically abused 4 year old.) "The absence of such foci does not negate the diagnosis. Release of myoglobin, tissue lipases, or other enzymes into the blood might similarly provoke sudden cardiovascular collapse. Extensive hemorrhage into areas of soft-tissue injury has been reported to produce significant anemia or exsanguination, and may play a role in some deaths (citing Zumwalt & Hirsch, *Subtle child abuse*, 1980), but we did not consider it to be a factor in our cases." Adding that they have never found fat emboli.

Intimal tears of the right atrium of the heart due to blunt force injuries to the abdomen: its mechanism and implications. Cumberland GD, Ricci L, McConnell CF. *Am J Forens Med Path* 1991; 12(2): 102-104. Waterhammer in blunt abd trauma child abuse.

Homicidal cardiac lacerations in children. Cohle SD, Hawley DA, Berg KK, Kiesel EL, Pless JE. *JFS* 1995 Mar; 40(2): 212-218. 6 cases; 5 of them are right atrial lacerations.

Child homicide caused by commotio cordis. Boglioli LR, Taff ML, Harleman G. *Pediatric Cardiology* 1998; 19: 436-438. Recited by Baker et al. 2002 (see below) as reporting the case of a 23 month old boy who was witnessed to sustain a blow to the chest from a babysitter. Instant VF; autopsy negative. See also Denton & Kandelkar, 2000, below.

Homicidal commotio cordis in two children. Denton JS, Kandelkar MB. *JFS* 2000; 45: 734-735. Cited by Baker et al., 2002 as reporting two children, a 14 month old and a 3 year old. See Baker et al., below.

Severe blunt cardiac injury in an infant: a case of child abuse. Karpas A, Yen K, Sell LL, Frommelt PC. *J Trauma* 2002 Apr; 52(4): 759-764. From the summary by Dr Reece in *Quarterly*: Five month old with a congenital VSD presented with bradycardia 2d AV block, and cardiogenic shock, hx that he had fallen on the floor. External exam normal. Radiographs normal. No rib fx. Echo found a large PW LV aneurysm. Father admitted that he had punched the infant. Survived with surgical repair.

Cardiac pathology in sudden unexpected infant death. Dancea A, Cote A, Rohlicek C, Bernard C, Oligny LL. (Quebec) J Peds 2002 Sep; 141(3): 336-342. Retrospective study of all autopsies of sudden infant deaths between 7 days and 2 years of age occurring in the province over a 13 year period. Had about 820 cases. 32% died awake, 68% died while asleep. Found that 82 cases had cardiac pathology, of which 54% were a malformation and 46% nonstructural pathology.

Malf	44
VSD	10
ASD	4
AS	4
Coronary anomaly	4
Other	22
Nonstructural pathology	38

The cardiac pathology was considered to be the COD in 84% of the cases. In the other cases it was considered to be of unknown significance. "Cardiac pathologic features are frequent when the child is witnessed to be awake at the time of sudden death." Commented on by Byard, below.

Unexpected infant death: occult cardiac disease and sudden infant death syndrome: how much of an overlap is there? Byard R. J Peds 2002 Sep; 141(3): 303-305. Commenting on Dancea et al., above.

Homicidal commotio cordis: the final blow in a battered infant. Baker AM, Craig BR, Lonergan GJ. Child Abuse & Neglect 2003 Jan; 27(1): 125-130. (have) Case report of the exhumed body of a previously abused 7 week old who was previously autopsied and signed out as a natural due to "pulmonary hemorrhage due to undetermined etiology." Four rib fractures noted at the original autopsy were ascribed to CPR. One year later a 3 month old sibling later presented with a broken femur which the father said he tripped on something and accidentally stepped on the baby. This history was accepted by the clinicians. One month later the sibling underwent a chest x-ray for unexplained vomiting and was found to have multiple healing rib fractures. On this information, they went back and reviewed the preautopsy x-rays of the deceased 7 week old, which were never reviewed at the time of the original autopsy. Review showed 17 healing rib fractures. On this basis the body was exhumed. Reautopsy showed 52 rib fractures in various stages of healing, etc. The father was interviewed before the exhumation, and said that on several occasions he had gotten so frustrated with the boy's crying that he "squeezed his ribs ...and...heard his ribs break." Further, that on the day of death he "went over to him and hit him with my fist on the center of his chest...He stopped breathing..." The reautopsy did not show any contusion of the precordium or chest wall. The Dx of commotio cordis was based on the father's confession. He was convicted of murder.

The authors review the literature on commotio cordis. Usually a sports injury causing VF. (Abrunzo, 1991; Maron et al., 1995, 1999). Altercations in adults, MVA's. (Frazer & Mirchandani, 1984; Tsatsakis, 1997). Autopsies show no ev of myocardial contusion.

Homicidal commotio in children: three cases in the literature:

Boglioli, Taff & Harleman, 1998: 23 month old boy was witnessed to sustain a blow to the chest from a babysitter. Collapsed immediately and pron at the scene. Autopsy was negative for ev of chest trauma or cardiac injury. Dx homicide.

Denton & Kandelkar, 2000: 14 month old boy struck hard in the chest by his father. Immediately unr. VF. Autopsy negative. Dx homicide.

Denton & Kandelkar, 2000: 3 year old struck once on the lateral chest and once on the sternum by boyfriend. Collapsed. Autopsy showed chest contusions as described by the bf. No internal injuries. Homicide.

"Commotio cordis should be included in the differential dx of any child fatality where otherwise non-lethal chest injuries are present..." But because the autopsy is usually negative, the dx rests on the timeline of the event and witness and caregiver statements.

Criminal consequences of commotio cordis (letter). Denton JS, Filkins JA, Stephenson D. Am J Cardio 2002 Nov; 90(10): 1181-1182

Sudden Death in Infancy, Childhood, and Adolescence. Roger W. Byard. West Nyack, NY: Cambridge University Press, 2004. <http://www.cambridge.org>

Long QT syndrome and other channelopathies -- testing for. FAMILION test. Genaissance Pharmaceuticals, Five Science Park, New Haven, CT 06511. 203-773-1450. <http://www.genaissance.com>
See also the Death Genomics Laboratory at the Mayo Clinic, Michael J. Ackerman, MD, 507-284-0101, ackerman.michael@mayo.edu

Pathology of the Heart and Sudden Death in Forensic Medicine. Fineschi V, Baroldi G, Silver M. CRC Press, 2006. 536 pp. <http://www.crcpress.com>

AUTOPSY TECHNIQUE

The assessment of the duration of illness in children unexpectedly dead. John L. Emery. Med Sci Law 1964; 4: 39-42 Thymus, liver, and costochondral junction histologic changes over hours and days and weeks post onset of illness or injury.

- Thymus: Pets at 0-12h, macrophages at 12-48h, many macrophages over 48h, loss of corticom demarc at 3-7d, early invol 7-14d, increased invol 14d
- Liver: Cloudy swelling at 0-12h, central pallor and some hydropic changes 12-48h, Fatty droplets 48h, becoming more...
- CCJ: "Failure of capillary loop permeation" 3-7d, "early bridging and matrix banding" 7-14d, "bridging and banding 14d to weeks.

With discussion. "Hydropic droplets can easily be mistaken for fatty droplets." As to CCJ, "In general, the bones growing most rapidly are those which show the most change in response to any general growth upset of the body. We use the CCJ of the fifth rib bc it is a site of rapid growth..." "When anything interferes with body metabolism diminishing the rate of growth, two types of changes occur. First, the reabsorption of the trabeculi continue[s] with at the same time a diminishing rate of production of trabeculi. This results in some trabeculi disappearing and the capillary loops cover two or more cartilage cell columns. This we call 'bridging.' The other change is a building up of a zone of matrix between the end of the cartilage cell column and the capillary, forming a transverse band of matrix. This we call 'banding.'" (He is talking about really severe growth arrest as in cancer, severe metabolic disease, most often seen in hospitalized children, even though he refers to those "found dead." This "banding" is a chronic effect seldom seen in forensic material. -JKR) Thanks to Dr. Andrews for this article.

The battered child syndrome: responsibilities of the pathologist. Curphey TJ, Kade H, Noguchi TT and Moore SM. California Medicine 1965 Feb; 102-104. Says that the pathologist must get pre-autopsy x-rays and photograph and diagram all external injuries and must section and date all osseous injuries. While aging soft tissue injuries is not within the state of the art, nonetheless they must be sectioned so that the resulting information can be used to test the witnesses' accounts and possibly obtain a confession. "...[I]t is this added dimension of time or age of injury that distinguishes the medico-legal autopsy from (t)he hospital autopsy." Thanks to Dr. Chadwick for finding this article.

Clinical significance of postmortem cultures. Wilson WR, Dolan CT, Washington JA, Brown AL Jr, Ritts RE JR. Arch Path Lab Med 1972 Sep; 94(3): 244-249

The examination of the sudden infant death syndrome infant: investigative and autopsy protocols. Jones AM and Weston JT. JFS 1976; 21: 833-841. See under SIDS.

SIDS Research: Part I: An overview of major trends. JFS 1976; 21(4): 833-841. Includes autopsy protocol.

The postmortem examination on the abused child: pathological, radiography, and legal aspects. Perspec Ped Path 1984; 8: 313-343

Time course of hemosiderin production and clearance by human pulmonary macrophages. Sherman JM, Winnie G, Thomassen MJ et al. Chest 1984; 6: 409-411. Cited by Schluckebier et al, 2002 for the proposition that "[I]t takes macrophages 33 to 48 hours to break down hemoglobin. Siderophages can remain in the lungs for as long as two weeks, if not longer."

Pathology of fatal child abuse and neglect. Zumwalt RE and Hirsch CS. In: Helfer RE and Kempe RS, eds., *The Battered Child*, 4th ed. Chicago: University of Chicago Press (1987)

Histological assessment of gestational age in human embryos and fetuses. Barbet JP, Houette A, Barres D, Durigon M. Am J Forens Med P 1988; 9: 40-44. Referenced by Piercecchi-Marti et al. (2004) as a standard source for histology of organ development, along with *Potter's*.

Age-related changes in normal human hearts during the first 10 decades of life. Part I: Growth: A quantitative study of 200 specimes from subjects from birth to 19 years old. Scholz DG, Kitzman DW, Hagen PT, Ilstrup DM, Edwards WD. Mayo Clin Proc 1988; 63(2): 126-136. Gives normal heart weights.

Organ weights in SIDS. Siebert JR, Haas JE. Pediatr Pathol 1994; 14: 973-986. This work supports the fact that the organ weights published from hospital sources are abnormally low. See Scholz et al., above.

Instruction and reference manual for the international standardized autopsy protocol for sudden unexpected infant death. Krous H. J SIDS Infant Mortal 1996; 1: 203-246

Instruction and Reference Manual for the State of California Standardized Autopsy Protocol for Sudden Unexpected Infant Death. Approved January 1997. Appears to be Dr Krous's International Manual. Also adopting the CDC "Guidelines for Death Scene Investigation of Sudden Unexplained Infant Deaths," implemented by California in April 1997.

Coroner training needs: a numeric and geographic analysis. Hanzlick R JAMA 1996; 276: 1775-1778.

On the need for more expertise in death investigation (editorial). Hanzlick R. Arch Path Lab Med 1996; 120: 329-332. Cited by the NAME Board (2001) along with the other Hanzlick articles here as support for the proposition that "room remains for improvement in death investigation nationwide."

Documentation and preservation of visual evidence of child abuse. Martin A. Finkel and Lawrence R. Ricci. Child Maltreatment 1997; 2: 322-330. Reece: very important.

Post-mortem radiological examination in infants: evidence of child abuse? Thomsen TK, Elle B, Thomsen JL. FSI 1997; 90(3): 223-230

Practice guidelines for examination of the placenta. Arch Path Lab Med 1997 May; 121(5): 449-476. Consensus statements from the CAP XIX Conference on the Examination of the Placenta: Patient Care and Risk Mangement, 1991. I have the brief summary that is published online at <http://www.guideline.gov>

Common errors in forensic pediatric pathology. William Q. Sturner. Am J Forens Med Path 1998 Dec; 19(4): 317-320. One of the country's most experienced forensic pathologists with many years of scholarly involvement in the study of infant deaths presents this plea for thoroughness and detail in the documentation of postmortem findings in child deaths, beginning with the death scene and ending with the final medical evaluation. He emphasizes detailed photography and tissue collection, because while errors

of interpretation can always be corrected by review of the morphologic findings, morphologic findings left undocumented are irretrievably lost.

Dr. Sturner names specific things to be looked for at the death scene and on the outside of the body, things to be photographed, preautopsy things to be done, anatomic details to be recorded internally, and things to be examined histologically. Among the many important “tricks of the trade” he mentions, I find the following five worthy of special note:

1. Examination of the “bed of death,” including all its contents and accoutrements, and an attempt to reconstruct the accident if an accident is part of the differential.
2. Thoroughly photographing the external aspects of the body and its coverings, including the orifices, as well as patterns of livor and external drainages and markings, before it is altered or washed.
3. Conservative evaluation of possible abnormalities of the anus and genitalia in view of the known difficulties thereof. This is an active area of forensic research at present.
4. Extensive histologic study of the upper respiratory tract and the lungs. [In this connection, I would like to independently note that dramatically superior histology has been obtained by formalin-inflating infant lungs at the autopsy table and fixing the intact lungs in a separate container. –JKR]
5. Obtaining consultation and detailed review of the significance of the overall findings before rendering a diagnosis.

(cf. “SIDS redux: is it or isn’t it?,” *supra* under “SIDS vs. Suffocation.”)

Medical examiner and coroner systems: history and trends. JAMA 1998; 279: 870-874.

Postmortem extravasation of blood potentially simulating antemortem bruising. Burke MP et al. Am J Forens Med Path 1998 Mar; 19(1): 46-49. Postmortem mutilation of a body caused considerable blood loss into the tissues of the head and face.

Autopsies in children: are they still useful? Kumar P, Taxy JB, Angst DB, Mangurten HH. Arch Pediatr Adol Med 1998; 152: 558-563. Reviewed 297 hospital deaths. 107 were autopsied, 190 were not. In the autopsied group, major disagreement with the clinical dx occurred in 6.5%, complete concordance in 66%. The authors argue that this supports the value of the hospital autopsy, but I’m not sure these results say that.

AAP position statement on investigation methods, 1999.

Investigation and review of unexpected infant and child deaths. Pediatrics 1999; 104: 1158-1160

Parietal fissures. This developmental anomaly can be mistaken for fractures. “Pseudofracture.” See Fenton LZ, Sirotak AP, Handler MH, Parietal pseudofracture and spontaneous intracranial hemorrhage suggesting nonaccidental trauma: report of two cases. Pediatr Neurol 2000; 33: 318-322, reviewed in Child Abuse Quarterly, July 2001.

An approach to dissecting the congenitally malformed heart in the forensic autopsy: the value of sequential segmental analysis. Horn KD and Devine WA. Am J Forens Med Path 2001 Dec; 22(4): 405-411. Gives the chamber identification method used for a case of cardiac and pulmonary isomerism. Actually a better review than Becker & Anderson.

Sudden Infant Death Syndrome. Byard RW and Krous HF, eds. New York: Oxford University Press, 2001. This book is in the Carpenter Library. Includes an International Standardized Autopsy Protocol as Appendix I.

The important need for autopsy standards. <http://sids-network.org/experts/> To help improve uniformity of information, the Division of Maternal and Child Health of the Department of Health and Human Services held a meeting in 1975 which resulted in investigative and autopsy protocols for examining the SIDS infant. These protocols were included in an article.

Rationale and technique for examination of nervous system in suspected infant victims of abuse. Judkins AR, Hood IG, Mirchandani HG, Rorke LB. Am J Forens Med P 2004 Mar; 25(1): 29-32. Advocates removing the brain and spinal cord all in one piece at autopsy. by means of first opening the vertebral canal by a posterior approach and freeing the entire cord, then turning the body over and dissecting the head in standard fashion, pulling the spinal cord out with the brain, through the foramen magnum. Suggests doing it under water for softened brains. Because the cervicomedullary junction is a critical site of injury in SBS. Says that “[T]here are 4 specific injuries that result from violent shaking: (1) subdural hematoma (typically between the 2 cerebral hemispheres); (2) retinal and optic nerve sheath hemorrhages; (3) tears of the cerebral white matter (especially corpus callosum); and (4) tears and hemorrhages of cervical (or more caudal) spinal cord and/or nerve roots.” (p. 29) Offers further advice on gross dissection of the brain and eye. As far as sections of the brain, advises 20 areas to be sectioned:

1. Superior frontal gyrus 2cm posterior to the frontal pole
2. Gyrus rectus at the same level
3. Centrum semiovale
4. Corpus callosum:
 - a. Genu
 - b. Anterior midbody
 - c. Splenium
 - d. Any abnormal areas
5. Motor cortex at the level of the genu of the internal capsule
6. Globus pallidus and anterior thalamus
7. Putamen and insula
8. Thalamus at the level of the posterior limb of the internal capsule
9. Parietal cortex at the same level
10. Hippo
11. Calcarine cortex
12. Midbrain
13. Vermis
14. Dentate nucleus
15. Rostral pons
16. Midpons
17. Medulla -- mid and caudal
18. Cervicomedullary junction
19. All of the spinal cord, including the nerve roots and the spinal dura
20. Dura and any subdural clots

Says that microscopic study of the brain should focus on:

1. Acute neuronal necrosis
2. Acute swelling, pyknosis, karyorrhexis or apoptosis of oligos
3. Retraction balls
4. Tissue necrosis
5. Unsuspected inflammatory disease

See also Dr Rorke’s essay “Neuropathology of inflicted childhood neurotrauma,” in Reece & Nicholson, eds, *Inflicted Childhood Neurotrauma* (consensus development conference), 2003.

Study of the brainstem, particularly the arcuate nucleus, in SIDS and SIUD. Maturri L, Ottaviani G, Alfonsi G, Crippa M, Rossi L, Lavezzi AM. Am J Forens Med P 2004 Mar; 25(1): 44-48. Examined brainstems from 106 SIDS, 30 control infants, and 51 stillborns. Found “a remarkable variability” of the arcuate nucleus in both size and neuronal density. Does not describe any gliosis or anything remotely approaching gliosis (*q.v.*). The description of this nucleus will vary depending on what level it is sectioned at. Advises uniform use of their “simplified technique,” which involves taking three transverse sections of the medulla, at the following levels (gives diagrams front & back):

1. From the pontomedullary junction down to the superior border of the olivary eminence
2. The midportion of the olivary eminence (obex)
3. The lower pole of the olivary eminence

You will take serial sections of each of these blocks (72 slides from each block). This will make possible standardized interlaboratory comparison of the brain stem.

Fetal visceral maturation: a useful contribution to gestational age estimation in human fetuses. Peircecchi-Marti M-D, Adalian P, Liprandi A, Figarella-Brnager D, Dutour O, Leonetti G. JFS 2004 Sep; 49(5): 912-917. Sampled organs from 448 normal fetuses from 20 weeks onward to develop a quantitative way of dating fetuses, based on the organ histology development given by Barbet et al, 1988 and *Potter's Pathology of the Fetus and Infant*. Kidneys: $1.51 \times \text{glom count} + 14.37$. Lungs: $6.06 \times \text{radial airspace count} + 8.86$, where "radial airspace count" equals the number of airspaces from the outermost bronchiole to the pleura along the shortest radial, as described by Emery and Mithal, 1960. Also for lungs: maturational stage: following Wigglesworth, gives four photos: pseudoglandular, canalicular, saccular, alveolar: $\text{age} = 1.24 \times \text{maturational stage} - 5.56$. Also the number of Hassall's corpuscles per hpf. Also the stratum corneum. Also the cutaneous adnexa. Also the thyroid gland. Also the adrenal gland. Total of six elements to be coded 0 (for putref) 1 2 3 4. They admit that just using the femur length was actually more accurate than all these soft-tissue items put together, but still, they advise, "In practice, it appears that the pathologist must correlate histological with anthropometric data in order to estimate gestational age as accurately as possible." See their femur length paper at

Sudden Death in Infancy, Childhood, and Adolescence. Roger W. Byard. West Nyack, NY: Cambridge University Press, 2004. <http://www.cambridge.org>

Autopsy Pathology. Finkbeiner, Davis & Ursell. Philadelphia: Churchill Livingstone, 2004. ISBN 0-443-07676-6. <http://www.us.elsevierhealth.com>

Pediatric forensic pathology in crisis. (editorial) Byard RW, Krous HF. Pediatric Developmental Pathology 2004; 7: 212-213. The need for pathologists who examine infants and children in a forensic setting to have specialized training. Recommend:

- a. Recognize pediatric forensic pathology as a separate subspecialty
- b. Uniform standards of investigation
- c. Create guidelines for courts accepting a pathologist as an expert witness in this area

[See also Dr Byard's "Lessons from the Sally Clark case, 2004, next below. -JKR]

Unexpected infant death: lessons from the Sally Clark case. Byard RW. Med J Aust 2004; 181: 52-54. All of Dr Williams' autopsy findings evaporated under later expert scrutiny. Dr Byard attributes this to lack of specialized training. See "in crisis," above.

Guidelines in pathologic and forensic-medical diagnostic of sudden unexpected infant (SIDS) and fetal death. Maturri L, Ottaviani G, Alfonsi G, Rossi L, Lavezzi AM. University of Milan, Institute of Pathology, 1st School, Lombardy Region. Privately published at http://users.unimi.it/~pathol/sids/sids_e.html

Autopsy Pathology: A Manual and Atlas. Finkbeiner et al. Philadelphia: Elsevier. ISBN 0-443-07676-6.

Role of the Medical Examiner in Fatal Cases. Michael Graham, MD and Tracey Corey, MD. In: Giardino AP and Alexander R, *Child Maltreatment: A Clinical Guide and Reference, 3d ed.* St. Louis: G.W. Medical Publishing, Inc., 2005.

RH

The following is my list of DDX. A discussion can be found in Levin, 2000. Another list can be found at http://www.chmcc.org/programs_services

1. Vaginal delivery (33%)
2. Caesarean section (7%)
3. Hemorrhagic disease of the newborn -- early-onset or late-onset form
4. CO intoxication
5. Spontaneous SAH
6. HTN
7. Papilledema
8. Chest trauma
9. Traumatic asphyxia
10. Abdominal trauma
11. Seat belt injury
12. Air bags
13. Head trauma
14. SBS
15. ECMO
16. Sepsis
17. Meningitis
18. Coagulopathy
19. Hypofibrinogenemia
20. Galactosemia
21. Extended resuscitation
22. Difficult intubation
23. Leukemia
24. Sickle-cell anemia
25. Rickettsia (see Tsutsugamushi disease)
26. CMV
27. Falciparum
28. Vivax
29. Rocky Mountain
30. General anesthesia
31. Juvenile X-linked retinoschisis
32. Valsalva's hemorrhagic retinopathy
28. Terson's syndrome
29. Purtscher's syndrome
30. Pertussis
31. AVM
32. Vasculitis
33. Fibromuscular dysplasia
34. Caval syndrome
35. High-altitude retinopathy
36. Bungee jumping
37. Rift valley fever
38. Hemophagocytic syndrome
39. Hemophagocytic lymphohistiocytosis
40. Toxo
41. MMM
42. Leptospirosis

43. Behcet's disease
44. Vitamin B deficiency (including Wernicke's encephalopathy)
45. Folate deficiency
46. Pernicious anemia
47. Liver disease (causing vitamin K deficiency)
48. Gastroenteritis (causing vitamin K malabsorption)
49. Strangulation
50. Coats' disease (tiny retinal AVM's)
51. Respirator retina
52. Menkes' disease
53. Retinal artery microaneurysm
54. Ligation of the retinal artery
55. Tsutsugamushi disease (insect-borne Rickettsiosis)
56. Cat scratch disease
57. Hemorrhagic macular dystrophy
58. AIDS
59. Hermansky-Pudlak syndrome
60. Glanzmann's thrombasthenia
61. Soccer ball impact
62. Cavernous sinus thrombosis
63. PNH
64. Supersonic bailout
65. Experimental rocket-sled deceleration (Stapp)
66. In-utero cocaine exposure
67. Hyponatremic seizures
68. Borelliosis
69. Glutaric aciduria
70. ALL
71. Meningococcal septicemia (group C)
72. SLE
73. CPR
74. Protein C deficiency (or Protein S deficiency, for that matter). See "Coagulopathy."
75. Osteogenesis imperfecta
76. Rendu-Osler-Weber syndrome
77. Cataract extraction
78. Ocular decompression syndrome in glaucoma treatment
79. RetCam
80. Hyperemesis gravidarum (RH in the gravida)
81. Scurvy
82. Vogt-Koyanagi-Harada disease
83. Interferon
84. Prematurity?
85. Steroids
86. Dengue
87. Paintball
88. Airbags
89. Incontinentia pigmenti

Unusual causes

High-altitude retinopathy in mountain climbers. Shults WT, Swan KC. Arch Opth 1975; 93: 404-408

Retinal hemorrhage following anesthesia. Bolder P and Norton M. *Anesthesiology* 1984; 61: 595-597. Case report of a lap chole pt who had a single unilateral venous hemorrhage attributed to valsalva/increased retinal vein pressure with Trendelenberg if patient gets light.

Respirator retina. Foos RY and Rhodes RH. *Arch Ophth* 1984; 102: 296-303. These are flame hemorrhages seen in association with respirator brain. Typically they consist of laked erythrocytes, similar to the white-matter petechiae seen in respirator brain and subacute anoxic encephalopathy.

Ocular complications associated with bungee jumping. David DB, Mears T, and Quinlan MP. *Br J Ophth* 1994; 78: 234-235

Bungee jumping and intraocular hemorrhage. Jain BK and Talbot EM. *Br J Ophth* 1994; 78: 236-237

Ophthalmic complications of bungee jumping. Chan J. *Br J Ophth* 1994; 78: 239

Retinal hemorrhage secondary to airbag-related ocular trauma. Sastry SM et al. *J Trauma* 1995 Apr; 38(4): 582. RH in one eye in an adult driver due to impact of the bag on the eye.

RH associated with spinal cord AVM. Clark RSB, Orr RA, Atkinson CS et al. *Clinical Pediatrics* 1995; 34: 281-283 See under "Terson's."

Retinal hemorrhage in meningitis. (letter). Fraser SG, Horgan SE, Bardavio. *Eye* 1995; 9(Pt 5): 659-660. A 12 year old with acute meningococcal meningitis was found to have a large subretinal and vitreous hemorrhage in the left eye after she complained of blurring of vision in the left eye. No coagulopathy was present. 5% of cases of meningitis have fundus involvement such as papilledema, optic atrophy etc. In his review, "The differential diagnosis of inflicted childhood neurotrauma," in *Inflicted Childhood Neurotrauma* (2003), Dr Reece quotes this article as stating that "[A]s far as we are aware, intra-ocular haemorrhage has not been described in any form of meningitis."

Retinal hemorrhages associated with in utero exposure to cocaine: experimental and clinical findings. Silva-Araujo AL, Tavares MA, Patacao MH, Carolino RM. *Retina* 1996; 16(5): 411-418. Produced blot and dome-shaped intraretinal hemorrhages and areas of ischemia located in the temporal part and often extending to the periphery of the retina by injecting pregnant Wistar rats with cocaine. Observed similar lesions in human newborns with prenatal cocaine exposure. "Morphologically similar to neonatal retinal hemorrhages, had a higher incidence than in controls; they also took longer to resolve."

Bilateral papilledema with retinal hemorrhages in association with cerebral venous sinus thrombosis and PNH. Hauser D et al. *Am J Ophth* 1996 Oct; 122(4): 592-593.

Two cases of incontinentia pigmenti simulating child abuse. Ciarello L and Paller AS. *Pediatrics* 1997 Oct; 100(4):e6. Lethal X-linked dominant causes RH and cotton-wool spots plus burn-like skin eruptions. See above under "DDX -- Specific Disease Entities."

Retinal haemorrhages and convulsions. Sandramouli S, Robinson R, Tsaloumas M, Willshaw HE. *Arch Dis Child* 1997; 76: 449-451. 33 children examined ophth within 48 h of adm for sz. No RH. Some had CPR: still no RH. Prospective study by ophths of 33 chil w motor sz. Aged 4 m to 14 yr. None were child abuse. None had RH. They added on one supplemental infant, a LBW 33 wk nb who devel sz on the 10th dy of life and had one RH in the posterior retina (reviewer Alex Levin says this was probably a birth hemorrhage.) Levin comments, "To my knowledge, this is the first study which attempts to document what has empirically been recognized for many years, that sz do not cause RH." According to "Child abuse and the eye" (q.v.), the same authors later reported a premature infant who had repeated sz beginning at 10 days of life and was found to have unilateral RH at 23 days. According to *Eye*, "Convulsions in young children rarely, if ever, cause RH." 7 See Tyagi et al., 1998.

Late-form hemorrhagic disease of the newborn: a fatal case report with illustration of investigations that may assist in avoiding the mistaken diagnosis of child abuse. Ruttly GN, Smith CN and Malia RG. Am J Forens Med Path 1999 Mar; 20(1): 48-51. See below under "In general."

White-centered retinal hemorrhages in ocular toxoplasmosis. Hayashi S et al. Retina 1997; 17(4): 351-352

Ophthalmic findings in a case of hemophagocytic syndrome. Kawamura H et al. Am J Ophth 1997 Aug; 124 (2): 261-263

Retinal hemorrhages in cerebral malaria. Olumese PE et al. East Afr Med J 1997 May; 74(5): 285-287

Fundus changes in Behcet's disease. Dominguez LN and Irvine AR. Trans Am Ophth Soc 1997; 95: 367-382

Retinal manifestations of idiopathic myelofibrosis, a hematologic disorder. Haskes C and Gagnon K. J AM Optom Assoc 1998 May; 69(5): 319-328. Retinopathy secondary to anemia from myeloid metaplasia in polycythemia vera. Blood AM et al. J Am Optom Assoc 1997 Nov; 68(11): 734-738

Ocular manifestations in the acute phase of leptospirosis. Martins MG et al. Ocul Immunol Inflamm 1998 Jun; 6(2): 75-79

[Warfarin for impending central retinal vein occlusion]. Furuta M, Sekiryu T, Sato H, Fujiwara T. Nippon Ganka Gakkai Sasshi 1999 Feb; 103(2): 124-128. Abstract: We reviewed the records of 10 patients who had impending central retinal vein occlusion in order to judge whether anticoagulant treatment with Warfarin was indicated. 6 men and 4 women. Of 6 eyes, retinal hemorrhage disappeared completely within 6 months. Late venous circulation time at fluorescein angiography was a useful index for this treatment. See the same group's 2002 work also.

Retinal venous macroaneurysm associated with premacular hemorrhage. Khairallah M, Ladjimi A, Messaoud R, Ben Yahia S, Hmidi K. (Tunisia). Ophthalmic Surgery with Lasers 1999 Mar; 30(3): 226-228. 50 y o man treated with a laser for this preretinal hematoma from this rare condition. "Hemorrhagic detachment of the internal limiting membrane or subyaloid hemorrhage in the macula may occur after retinal vessel rupture with physical exertion (Valsalva's retinopathy), or in retinal vaascular diseases, such as prol;iferative diabetic retinopathy, and retinal *arterial* macroaneurysm. Venous is rare.

Bilateral vitreous hemorrhages in an infant with low fibrinogen levels. J AAPOS 1999 Aug; 3(4): 255-256. RH progressed to vitreous hemorrhage in this child with hypofibrinogenemia. Etiology not given in the abstract.

Air bags and ocular injuries. Stein JD, Jaeger EA, Jeffers JB. Transactions of the American Ophthalmological Society 1999; 97: 59-82, with discussion. Review of the literature disclosed 44 articles describing 97 patients with the following injuries:

corneal abrasion	49%
hyphema	43%
vitreous or retinal hemorrhages	25%
retinal tears or detachments	15%
ruptured globe	10 pts

Can include impact at 30 mph or less.

Ocular findings in glutaric aciduria type 1. Kafil-Hussain NA, Monavari A, Bowell R et al. J Pediatr Ophth 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.

Hermansky-Pudlak syndrome presenting with subdural haematoma and retinal haemorrhages in infancy. Russell-Eggitt IM, Thompson DA, Khair K, Liesner R, Hann IM. Journal of the Royal Society of Medicine 2000 Nov; 93(11): 591-592.

Hyponatremic seizures in infancy: association with retinal hemorrhages. Krugman SD, Zorc CC, Walker AR. Pediatric Emergency Care 2000 Dec; 16(6): 432-434. Comment 2001 Aug; 17(4): 313-314. Presented two cases with the unexpected finding of RH. Association never previously reported. But both cases were SBS.

High altitude retinal hemorrhages in a Colorado skier. Honigman B, Noordewier E, Kleinman D, Yaron M. High Altitude Med Biol 2001 Winter; 2(4): 539-544. RH are commonly seen at altitudes above 4270 m. Case report of a 29 year old skier who started at 2930 m and went up to 3470 m and developed acute mountain sickness with bilateral RH and macular involvement.

RH and fatal stroke in an infant with fibromuscular dysplasia. Currie ADM, Bentley CR, Bloom PA. Arch Dis Child (UK) 2001 Mar; 84(3): 263-264. Autopsy case report of intracranial hemorrhage and RH due to fibromuscular dysplasia.

Bilateral retinal haemorrhages: an unusual presentation of pernicious anaemia. Gupta V, Bremner FD, Telfer P. (London). Br J Haematol 2001 Mar; 112(4): 831.

Carbon monoxide poisoning and the eye. Denniston A. J Royal Soc Med 2001 Aug; 94(8): 425-426

Optic nerve lesion following neuroborreliosis: a case report. Had NFL near disc due to optic neuritis. See under ONSH.

Ocular morbidity associated with airbag deployment: a report of seven cases and a review of the literature. Ball DC, Bouchard CS. Cornea 2001 Mar; 20(2): 159-163. Corneal abrasion, corneal decompensation, corneal alkali injury, hyphema, iris sphincter tears, vitreous hemorrhage, commotio retinae. Found 74 published cases.

[Purtscher-like retinopathy in systemic lupus erythematosus: two cases.] Sallami D, Ben Zina Z, Jelliti B, Abid D, Feki J, Chaabouni M. J Fr Opht 2002 Jan; 25(1): 52-55.

Bilateral massive choroidal hemorrhage secondary to Glanzmann's syndrome. Saatci AO et al. (Turkey) Ophth Surg Lasers 2002 Mar-Apr; 33(2): 148-151.

Macular hemorrhage secondary to increased intrathoracic pressure and difficult intubation. Abbas A, Hyman G. (Pak) J Pak Med Assoc 2002 Jun; 52(6): 265-266.

A case of hereditary hemorrhagic telangiectasia (Rendu-Osler-Weber's disease) with dystrophic calcinosis cutis and retinal lesions. Takahama H et al (Japan). J Dermatol 2002 Aug; 29(8): 512-515.

Retinal hemorrhages in meningococcal septicemia. Dinakaran S, Chan TK, Rogers NK, Brosnahan DM. JAPOS 2002 Aug; 6(4): 221-223. (Sheffield) Prospective study of all chil adm w meningoc. They all had direct and indirect opth. Twelve chil, mean age 4.5 yrs. All had a coagulopathy. RH + in five chil (42%), and all of these had group C Meningococcus, as did all the fatal cases. The abstract does not detail the RH.

[Purtscher-like retinopathy in systemic lupus erythematosus: two cases.] Sellami D, Ben Zina Z, Jelliti B, Abid D, Feki J, Chaabouni M. J Fr Opht 2002 Jan; 25(1): 52-55. (Tunisia). Multiple whitish patches over the macular and peripapillary areas in the absence of trauma, apparently in adults.

[Ocular complications in systemic lupus erythematosus -- choroidal and retinal pigments epithelial changes.] Sawa M, Saito Y, Kameda C, Tano Y. Nippon Ganka Gakkai Zasshi 2002; 106(8): 474-480. Cotton-wool patches and RH are common findings in SLE. Probably caused by long-term steroid therapy.

Hemophagocytic lymphohistiocytosis masquerading as child abuse: presentation of three cases and review of central nervous system findings in hemophagocytic lymphohistiocytosis. Rooms L, Fitzgerald N, McClain KL. Pediatrics 2003 May; 111(5 Pt 1): e636-640. Causes intracranial hemorrhage and RH.

Retinal hemorrhages in type I osteogenesis imperfecta after minor trauma. Ganesh A, Jenny C, Geyer J, Shouldice M, Levin AV. Ophth 2004 Jul; 111(7): 1428-1431. Report of 3 patients who developed RH and SDH after minor trauma. Cases found through SIGCA-MD-L.

Patient 1	unilateral flame hemorrhages in the posterior pole OS + SDH
Patient 2	RH and vitreous hemorrhages bilateral + SDH
Patient 3	scattered intraretinal hemorrhages bilateral + SDH

“This is the first report of RH and SDH after trivial trauma in pts with type I OI. The collagen defects underlying this disorder of bone and connective tissue may predispose pts with type I OI to RH and SDH after minor trauma.”

Retinal hemorrhages in an infant following RetCam screening for retinopathy of prematurity. Adams CGWm Ckarj BJ, Fang S, Hill M. Eye 2004; 18: 652-653. 25 week preemie.

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. This scholar of vitamin C argues that infantile scurvy can mimic SBS. See above under “Differential diagnosis -- specific disease entities.” In this article, he refers to the work of Lund and Kimble in 1943 as follows: “In 1943 Lund and Kimble reported: ‘Hyperemesis gravidarum may lead to dangerously low levels of vitamin C. Clinical scurvy may appear. The retinal hemorrhages of severe hyperemesis gravidarum are a manifestation of vitamin C deficiency and are similar to petechial hemorrhages seen elsewhere. The hemorrhages cease after adequate therapy with vitamin C...’” Clemetson goes on to say, “Whenever a woman complains of excessive vomiting in pregnancy and is found to have acetone or acetoacetic acid in the urine due to starvation, even for a day or two, she should be admitted to hospital and given intravenous fluids and supplementary vitamins. For some reason, vitamin C deficiency develops very rapidly in hyperemesis gravidarum. Retinal hemorrhages and jaundice used to be indications for therapeutic abortion to prevent the development of Wernicke’s encephalopathy...” Citing Lund CJ, Kimble MS, Some determinants of maternal and plasma vitamin C levels. Am J Ob Gyn 1943; 46: 635-647.

Retinal hemorrhages in type I osteogenesis imperfecta after minor trauma. Ganesh A, Jenny C, Geyer J, Shouldice M, Levin AV. Ophth 2004; 111: 1428-1431. Three patients culled from SIGCA-MD-L. All had short-fall histories and two presented with seizures. Two had blue sclerae. Pt 1 had flame hemorrhages in the posterior pole. Pt 2 had intraretinal hemorrhages and vitreous hemorrhages. Pt 3 had small scattered intraretinal hemorrhages in the posterior pole. The authors postulate that the RH were caused by softness of the globe and capillary fragility. Not clear what if any the intracranial injuries were.

Vogt-Koyanagi-Harada disease occurring during interferon alpha therapy for chronic hepatitis C. Kasahara A, Hiraide A, Tomita N et al. (Osaka) J Gastro 2004; 39(11): 1106-1109. Had RH + cotton wool spots.

Paintball injuries in children: more than meets the eye. Listman DA. Pediatrics 2004 Jan; 113 (1 Pt 1): e15-e18. Incidence of paintball eye injuries seen in ED’s is 1200/year. Include hyphema, retinal detachment, cataract, corneal abrasion, vitreous hemorrhage, commotio retinae. Many pts sustained permanent visual impairment.

Retinal hemorrhages in premature infants: a pathogenetic alternative diagnosis to child abuse. Fledelius HC. Acta Ophth Scand 2005; 83(4): 424-427. Eleven infants who had RH observed in the first months of life. Four had ROP. EGA 27-34 weeks. One infant had Zellweger’s syndrome. One had factor II

deficiency. Some bleeding appeared to be truly postnatal. The RH took much longer to resolve than is normally expected in newborns.

Retinal hemorrhages in four patients with dengue fever. Chlebicki MP, Ang B, Barkham T, Laude A. (Singapore) *Emerging Infectious Diseases* 2005; 11(5): 770-772. RH coincided with the platelet nadir. Note that dengue is a viral hemorrhagic fever, which can cause the dengue hemorrhagic syndrome (DHS).

Dietary folate deficiency and bilateral retinal hemorrhages. Hughes M, Leach M. *Lancet* 2006 Dec 16; 368(9553): 2155. Case report by these ophthalmologists of a 33 year old vegetarian who had lived on nothing but fruits and vegetables for years, presented with lethargy, aphthous ulcers, and BOV. Hgb 4.8, MCV 119, WBC 6.1, plts 86,000, red cell folate 103 mcg/L (n=200-800), B-12 178 ng/L (n=210-1000). VA was reduced. Schilling test was normal. Fundoscopy showed multiple bilateral RH (fundus photographs given showing flame hemorrhages extending 3DD OD and out to the periphery OS). VA, hemorrhages, and the other symptoms resolved over a few months with oral folate and cyanocobalamin replacement therapy. "Retinal haemorrhage is a well recognised complication of the hyperdynamic retinal circulation and tissue hypoxia induced by severe anaemia."

Retinopathy in a multiple sclerosis patient undergoing interferon therapy. Saito H, Suzuki M, Asakawa T, Kato S. *Multiple Sclerosis*, 2007

In general

Per Dr. Levin, six things that should be documented in every eye exam for RH:

1. What type of hemorrhages? (subretinal, preretinal, intraretinal; flame, dot-blot)
(see diagram below under "Histology")
2. Where located?
3. Number? (qualitative characterization is ok)
4. Is traumatic retinoschisis present? What findings support it?
5. Any evidence of an underlying ocular disease?
6. Papilledema?

Source: SIGCA-MD-L, June 27, 2000

Jane D. Kivlin, Ophthalmic Manifestations of Shaken Baby Syndrome. Chapter 8 in: Lazoritz S and Palusci VJ, eds., *The Shaken Baby syndrome: A Multidisciplinary Approach.* New York: The Haworth Maltreatment and Trauma Press, 2001.

The American Academy of Ophthalmology web site has an essay on SBS/RH which appears to be essentially a rehash of Dr Levin's published work.

http://www.aao.org/aao/education/library/shaken_baby.cfm

Multiple fractures in the long bones of infants suffering from subdural hematoma. Caffey J. *Am J Roentg* 1946; 56: 163. Reporting six infants and suggesting that they are victims of intentional "battering." Two had RH, and these two also had SDH.

Pre-retinal and optic nerve sheath hemorrhage: pathologic and experimental aspects in subarachnoid hemorrhage. Smith DC, Kearns TP, Sayre GP. *Trans Am Acad Ophth Otol* 1957; 61: 201-211. Plunkett (AJFMP 2001 Mar) cites this art for the proposition that RH can be produced experimentally by ligating the central retinal vein or by abruptly increasing ICP.

Ophthalmologic hydrostatic pressure syndrome. Lyle DJ, Stapp JP, Button RR. Am J Ophth 1957; 44: 652. Cited by Ober (1980) as “further evidence that hemorrhagic retinopathy can result from indirect force was found in human volunteers subjected to high decelerative forces (negative acceleration.) Experiments using rocket-propelled sleds that were decelerated by a braking system reproduced the forces encountered in supersonic escape from aircraft. Retinal hemorrhages were produced in subjects exposed to forces as great as 46G, and the fundus lesions were thought to be caused by combined hydrostatic and decelerative forces. Similar fundus changes have been observed in pilots following bailout from supersonic aircraft.” The context is that Ober is developing an argument that RH can be caused by shaking, since in his autopsy case, there was an occipital bruise but no skull fx and minimal intracranial injury, yet there was complete retinal detachment. Also citing Stapp, 1971, in Aerospace Medicine.

The eye of the “battered child.” Kiffney GT. Arch Ophth 1964; 72: 231-233. Cited by Caffey in his 1972 Jacobi Lecture on the theory and practice of shaking infants as a case report of bilateral retinal detachments in a battered girl of 7 months. Caffey adduces this report with others by Maroteaux (1967) and Friendly (1971) as support for the statement that “The retinal lesions caused by shaking will undoubtedly become valuable signs in the diagnosis of subclinical inapparent chronic subdural hematoma, and also become valuable signs in the diagnosis of subclinical inapparent chronic subdural hematoma, and also become a productive screening test for the prevalence of whiplash-dependent mental retardation and other types of so-called idiopathic brain damage.” In 1974 Caffey summarizes this article by saying, “In 1964 Kiffney found bilateral retinal detachments behind incomplete cataracts in one so-called battered infant 7 months of age, which were originally diagnosed as retinoblastomas.” Ober (1980) cites this as a case report of a 7 month old with a history of repeated head injuries and traumatic detachment of the retina.

The fundi of battered babies. Maroteaux P, et al. Presse Medicale 1967; 75: 711. Cited by Caffey in the Jacobi Lecture (1972) as having found “permanent, stable retinal lesions in the peripheries of the ocular fundi of previously battered children.” Caffey finds three articles on RH in battered children up to 1971 -- Kiffney, Maroteaux, and Friendly. In 1974 Caffey wrote, “Maroteaux and associates found plaques in the ocular fundi which tended to be located in the periphery of the temporal segments of the retinas. The authors state that these lesions cannot be satisfactorily explained on the basis of battering and they question the validity of the term ‘battered child’ for all abused infants, and its worldwide use in English. They propose that some of the affected infants are victims of overvigorous *manipulations*, not battering.” (emphasis in original).

Fundi of battered babies. Gilkes MJ and Mann TP. Lancet 1967; 2: 468-469. According to Ober, Gilkes & Mann, along with Caffey, were the first to implicate an indirect mechanism, rather than direct eye trauma, in causing intraocular injury in battered infants. These authors suggested “a sudden, extreme rise in both intracranial and intraocular venous pressure” instead of direct ocular trauma. One case was a child who had been swung by his feet and had head impact. The authors also suggested a similarity to Purtscher’s retinopathy in the case of an infant who had been gripped by the chest and shaken, citing Marr & Marr, 1962. Caffey discusses this article as follows: “In 1967 Gilkes and Mann stated that they had found only one reference, that of Kiffney, on the status of the eyes of abused infants. In their cases, they were impressed with the extensive spread and the persistence of the signs of ocular hemorrhages, both preretinal and intraretinal, and by the presence of gross papilledema in some cases. These authors cite the patients of Wallis who suffered from subdural hematomas induced by the parents who ‘gripping the infants by the ankles, swung him in a circle around their head’ (so-called cracking the whip); and the infant of Breinin who ‘had a traumatic retinopathy’ after having been gripped by the chest and shaken violently.” Caffey, 1974. This article is cited by Luthert in “Why do histology on RH?” (2003) along with Kaur, Taylor, 1992 as the sources considering the possibility of increased ICP as the cause of RH in battered babies.

Ocular signs observed in the syndrome of Silverman. Aron JJ et al. Ann Oculist 1970; 208: 533. Caffey cites this article after that of Maroteaux, as follows: “Aron and associates found similar retinal spots located in the peripheries of the temporal segments of the fundi, some with retinal detachments in all 18 abused infants. More than half of these lesions had persisted for more than ten years and one as long as 19 years.” (Caffey, 1974)

Ocular manifestations of physical child abuse. Friendly DS. Trans Am Acad Ophth Otol 1971; 75: 318-332. Followup study to Gilkes & Mann, establishing that RH is common in child abuse. According to Ober (1980), he found ev of ocular trauma in 46% of 28 children with unexplained fractures, and 35% of 26 pts with unexplained soft-tissue injuries. mostly RH associated with intracranial bleeding, but also finding orbital ecchymosis, anisocoria, sixth nerve palsy, dysconjugate eye movements, subconj hem, hyphema, cataract, dislocate lens, papilledema, optic nerve atrophy, vitreous hemorrhage, and retinal detachment. Three cases had retinal detachment thought to be due to direct ocular trauma. This article is one of the three cited by Caffey in the Jacobi Lecture of 1972 on shaking. Caffey cites it as having found "retinal hemorrhages associated with intracranial bleedings" in five battered children. In his later literature review (1974), Caffey states, "In five abused infants Friendly found vitreous hemorrhages, bilateral cataracts, dislocated lenses and retinal detachments. The first case is possibly an example of bilateral cataracts from manual whiplash shaking in view of the lack of a history of trauma and lack of external signs of trauma to the head."

Infantile subdural hematoma and its relationship to whiplash injuries. Guthkelch AN, Br Med J 1971 May 22; 2(759): 430-431 (have) Stating that SDH is one of the commonest features of the battered child syndrome and suggesting that SDH can be due to minimal or indirect or whiplash trauma to the neck in both adults and infants. The author presents two cases: Case 1 a 6 month boy with SDH, no notation concerning the eyes, Case 2 a 6 month boy adm comatose with a tense fontanelle and bilateral retinal haemorrhages, SDH confirmed by tap, readmitted with recurrent SDH and grip marks on the arms, confession of "might have" shaken. Guthkelch merely mentions this clinical finding of RH, does not comment on it. Caffey (1974) cites this art as reporting two instances of intraocular hemorrhages in battered infants, although Caffey states that he himself was the first to report this association (1946).

Pathogenesis of occlusion of the central retinal vessels. Hayreh S. Am J Ophth 1971?; 72: 998-1011. Very high ICP causes occlusion of the central retinal vein.

Stapp DP. Biodynamics of deceleration impact and blast. In: Randel HW (ed.), *Aerospace Medicine, 2d edition.* Baltimore: Williams & Wilkins, 1971, p. 133. Cited by Ober (1980) along with Lyle, Stapp, Button, 1957, for the proposition that pure deceleration can cause RH and retinal detachment without direct eye trauma.

Ophthalmic manifestations of the battered-baby syndrome. Harcourt B and Hopkins D. Br Med J 1971; 3: 398-401. According to Ober (1980) these authors reported a case of organized vitreous hemorrhage with tractional retinal detachment associated with severe head trauma. According to Caffey (1974), "Eleven abused infants studied by Harcourt and Hopkins had ocular complications, eight had permanent impairments of visual acuity and ten had intraocular bleedings."

Ocular damage in the battered-baby syndrome. Mushin AS, Morgan G. Br Med J 1971; 3: 402-404. According to Caffey's summary (1974), "Mushin found ocular changes in 12 of 19 *battered infants*, all 12 of whom had permanent impairment of vision in one or both eyes."

Ocular injury in the battered-baby syndrome: report of two cases. Mushin A, Morgan G. Br J Ophth 1971; 55: 343-347. According to Ober (1980), Mushin presents two cases of retinal detachment in children with head trauma and comments on the considerable force required to cause direct damage to the retina. Caffey cites this article in "The whiplash shaken infant syndrome" (1974) as a report of "intraocular hemorrhages" reported in shaken infants (as opposed to "so-called battered infants"). Leestma (2005) states that "In this case, the father admitted to strangling the baby with a blanket and then spent the remainder of the night shaking the baby in an attempt to revive him. Pharyngeal bruising and hemorrhages were noted at autopsy and 'extensive' bruising, sites not specified, was noted as well."

Ocular clues to child abuse. Jensen AD, Smith RE, Olson MI. J Pediatr Ophth Strab 1971; 8: 270-272. According to Ober (1980), reports ev of ocular trauma in 40% of 48 battered children -- mostly RH associated with intracranial bleeding, but also finding orbital ecchymosis, anisocoria, sixth nerve palsy, dysconjugate eye movements, subconj hem, hyphema, cataract, dislocate lens, papilledema, optic nerve atrophy, vitreous hemorrhage, and retinal detachment.

Experimental effect of intracranial hypertension upon intraocular pressure. Lehman RAW, Krupin T, Podos SM. J Neuros 1972; 36: 60-66. Plunkett (AJFMP 2001 Mar) cites this art for ICP as cause of RH. "Whenever ICP exceeds retinal venous pressure."

"Fallen fontanelle" (caida de mollera): a variant of the battered child syndrome. Guarnaschelli J, Lee J, Pitts FW. JAMA 1972; 222: 1545. According to Caffey's summary (1974), the Mexican grandmother...holding the infant topsy-turvy by its ankles, with its head over a pan of water, and then shaking the infant up and down while an assistant slapped and pounded on the soles of its feet. The sunken fontanel did rise and had become bulgy when admitted to the hospital. Subhyaloid hemorrhages were found in the ocular fundi... Notice that this entity has never been reported since. --JKR

On the theory and practice of shaking infants. Its potential residual effects of permanent brain damage and mental retardation (the Jacobi lecture) Caffey J. Am J Dis Child 1972 Aug; 124(2): 161-169 (have) See below under "Shaken."

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. (have) See below under "Shaken." In this article, at pages 399-400, Caffey reviews the literature on ocular lesions in child abuse. (See also his 1972 article, *supra*, where he reviews the RH literature including Friendly's work.) I have incorporated his references into the above citations, particularly since some of these older articles are no longer available. JKR

Retinal detachment in a battered infant. Weidenthal DT, Levin DB. Am J Ophth 1976; 81: 725. According to Ober (1980), case report of an 8 week old battered infant with bilateral giant retinal tears and retinal detachments thought to be due to direct eye trauma.

Retinal hemorrhages in posterior vitreous detachment. Cibis GW, Watzke RC, Chua J. Am J Ophth 1975; 80: 1043-1046. Cited by P Luthert in "Why do histology on RH" (2003) to the effect that "Possibly the most direct evidence for vitreoretinal tractional forces causing retinal haemorrhage comes from cases of haemorrhage associated with posterior vitreous detachment."

Extensive retinal haemorrhages in infancy -- an innocent cause [letter.] Bacon CJ, Sayer GC, Howe JW. BMJ 1978 Feb 4; 1(6108): 281 (have) This brief and mystifying case report is of a two month old boy who was left sleeping in the garden in his pram. After half an hour his father found him not breathing. There was some slight blood on the pillow. The wife put the baby over her shoulder and slapped him repeatedly on the back to revive him. Some bloody mucus was expelled from the nostrils. In the ER he was shocked and cyanotic with some blood caked in his nostrils. No trauma seen. In the eyes they could see "extensive fresh haemorrhages in the nerve-fibre layer of both fundi. There was bilateral macular oedema but no swelling of the optic discs." Hgb 8.2. Labs normal, LP and skeletal survey as well as subdural tap were normal. He recovered completely in three days, with resolution of the retinal haemorrhages. They questioned the parents closely bc of a suspicion of child abuse, and did a social-service investigation, consulted the family's private doctor, found no indication of abuse. Their final conclusion is that the mother's thoracic compressions caused the RH. "RH can be produced by thoracic compression that is insufficient to cause detectable damage to the chest itself. (citing Morgan, 1945). The retinal vessels of infants may be particularly vulnerable, for many babies have extensive fundal haemorrhages after a na apparently normal birth. We think that this baby became apnoeic after aspirating blood from an epistaxis, and that his mother's life-saving measures transmitted pressure from the thorax to retinal veins that were already compromised by hypoxia."

Retinal hemorrhage in the battered child. Eisenbrey AB. Child's Brain 1979; 5: 40-44. Found RH in 1 of 32 nonbattered cases (3%), and in 16 of 26 battered children (62%). The one instance in nonbattered was a birth hemorrhage after traumatic delivery. Cited by Buys, Levin et al. (1992) for the proposition that "The presence of retinal or vitreous hemorrhages in a child younger than 3 years of age who has no other medical risk factors has been considered by some to be pathognomonic of child abuse (nonaccidental) injury." According to these authors, Eisenbrey found RH in 16/26 battered children and only 1/32 nonbattered

children, and that one was a neonate with a traumatic delivery. Buys, Levin find several faults with this study, which prompted them to undertake their own.

Retinal hemorrhages in infancy. Stanton AN. Br Med J 1979; 1: 616

Ocular manifestations of child abuse. Harley RD. J Pediatr Ophth Strab 1980; 17: 5-13.

Hemorrhagic retinopathy in infancy: a clinicopathologic report. Ober RR. J Pediatr Ophth Strab 1980; 17: 17-20. Discusses hemorrhage in all layers plus the subretinal space, and apparently (according to Lambert, Johnson & Hoyt, 1986) first proposes the vitreoretinal traction mechanism of RH in SBS.

Retinopathy in the sexually abused battered child. San Martin R, Steinkuller PG, Nisbet RM. Ann Ophth 1981; 13: 89-91

Ophthalmoscopic findings in occult child abuse. Giangiacomo J and Barket KJ. J Ped Ophth Strab 1985; 22(6): 234-236 The eyes of child abuse victims: autopsy findings

Serious head injury in infants: accident or abuse? Billmire ME and Myers PA. Pediatrics 1985; 75: 340-342. Studied 84 infants 0-1 yr with head trauma requiring a CT scan. 54 accidental and 28 abused. RH in 89% of the abused and 0 of the accidentals.

When uncomplicated skull fractures are excluded, 95% of all serious head injury to infants is child abuse. 19 cases of intracranial bleeding: 18 of these were abusive. No RH seen in non-abuse.

Optic nerve sheath and retinal hemorrhages associated with the shaken baby syndrome. Lambert SR, Johnson TE, Hoyt CS. Arch Ophth 1986; 104: 1509-1512. Collected by Leestma (2005) as one of the articles presenting cases with a history of admitted shaking (total 54 cases in the literature). As to this article, Leestma describes the case as, "The baby had apparently been vomiting for 4 days prior to admission. The child's babysitter admitted shaking the child vigorously 4 days before admission. Examination showed ecchymoses over the sternum and other injuries... At autopsy papilledema was noted in one eye. The sternal injury was said to be consistent with 3-5 days' duration, as was the age of the subdural hematoma the child had."

Hemorrhagic retinopathy following uncomplicated pediatric cataract extraction. Mets MB, Del Monte M. Arch Ophth 1986; 104: 979. Cited by P Luthert in "Why do histology on RH" (2003) to the effect that "The presence of retinal haemorrhage in two infants undergoing cataract extraction might also be consistent with the hypothesis that vitreous tractional forces may be important in the pathogenesis of infant retinal haemorrhage. Cataract extraction, however, is associated with changes in intraocular pressure and this in turn may have effects on retinal circulation."

Spontaneous subhyaloid and retinal haemorrhages in an infant. McLellan NJ, Prasad R, Punt J. Arch Dis Child 1986; 61: 1130-1132. MCA aneurysm causing intracerebral hemorrhage and RH in a 6 week old term female admitted for possible abuse. Developed seizures at home, sz in ER, full fontanelle, "extensive bilateral RH and a large R subhyaloid hem" on adm. (no fundus photo given) No history or signs of direct injury or neglect. Coags & plts normal, skel surv nl, LP bloody w xanthochromic supernatant. Initially dx'd as probable SBS. CT showed a large localized intracerebral hematoma (fig. 1). Cerebral angiography disclosed an MCA **aneurysm**. Comments in the abstract that "These findings were not the result of shaking." Cited by Luthert in "Why do histology on RH?" (2003) to the effect that "[A]lthough Terson's syndrome was originally defined in terms of vitreous haemorrhage associated with intracranial subarachnoid bleeding, it is well recognized that massive intracranial haemorrhage can lead to appearances remarkably like those seen in cases of alleged shaking." See also Hupp et al., 1984. Medele et al., 1998 for reports of RH caused by raised ICP and by optic nerve sheath trauma causing compression of the retinal vessels.

Protein C deficiency associated with vitreous hemorrhage in a neonate. Pulido JS, Lingua RW, Cristol S, Byrne SF. Am J Ophth 1987; 104: 546-547. Hardly surprising.

Autopsy findings in the eyes of fourteen fatally abused children. Rao N, Smith RE, Choi JH, Xiaohu X, Kornblum RN. FSI 1988; 39: 293-298. Cited by Gilliland, 2003 as having autopsied 14 fatally abused children in Los Angeles and characterized the ocular changes as 'subdural haemorrhage of the optic nerve and retinal haemorrhage which involved all the layers of the retina, but most commonly the nerve fibre layer, ganglion cell layer and inner nuclear layer. The presence of blood cavities within the retina partially supported the hypothesis of traumatic retinoschisis. The control cases of non-abused children rarely showed intraocular haemorrhage.'

Sequential cranial CT in infants with RH. Giangiacomo J, Khan JA, Levine C and Thompson VM. Ophth 1988; 95(3): 295-299

Mistakes in diagnosing non-accidental injury: 10 years' experience. Wheeler DM, Hobbs CJ. BMJ 1988; 296: 1233. Includes a case of valsalva's hemorrhagic retinopathy due to pertussis coughing.

Retinal hemorrhage predicts neurologic injury in the shaken baby syndrome. Wilkinson WS, Han DP, Rappey MD, Owings CL. Arch Ophth 1989; 107: 1472-1474

Do retinal hemorrhages occur with accidental head trauma in young children? Alario A and Duhaime 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 had intracranial injuries, 14 had uncomplicated skull fxx, 2 EDH. None had RH. See 1992 article, below, and 1999 household accident article, below.

Ocular and associated systemic findings in suspected child abuse. Elner SG, Elner VM, Arnall M, Albert DM. Arch Ophth 1990 Aug; 108(8): 1094-1101. See under "Traumatic retinoschisis." 10 child abuse cases. 7/10 had ocular injuries, which all included RH, vitreous hemorrhage, and ONSH. 4/10 had traumatic retinoschisis or tractional retinal folds.

Posterior segment manifestations of ocular trauma. Williams DF, Swengel RM, Scharre DW. Retina 1990; 10(suppl.): 535-544 Posterior segment manifestations of ocular trauma. Williams DF, Meiler WF, Williams GA. Retina 1990; 10(1)S: S35-S44

Ocular manifestations of child abuse. Levin AV Ophthalmology Clinics of North America 1990; 3(2): 249-264

Retinal hemorrhages, seizures, and intracranial hemorrhages: relationships and outcomes in children suffering traumatic head injury. Luerssen TG, Huang JC, McLone DG et al. Concepts in Pediatric Neurosurgery 1991; 11: 87-94. Studied 811 children with traumatic brain injury. Found RH in 27. Only 5 accidental cases had RH, which would be 0.6% of the accidental cases. 22 nonaccidentals had RH, which was 81% of the nonaccidentals.

Retinal hemorrhage in accidental head trauma in childhood. Elder JE, Taylor RG, Klug GL. J Paediatr Child Health 1991; 27: 286-289. 25 accident patients, no RH.

The eyes of child abuse victims: autopsy findings. Riffenburgh RS and Sathyavagiswaran L. JFS 1991; 36(3): 741-747

Ocular findings at autopsy of child abuse victims. Riffenburgh RS and Sathyavagiswaran L. Ophthalmology 1991; 98: 1519-1524. Histology of 190 consecutive cases of autopsy eyes received from 1985 to 1989.

55 abuse

43 possible abuse

92 SIDS, drownings, auto accidents

Found frequent hemorrhages in the abuse and possible abuse victims, few in the nonabuse. Characterized hems as most extensive in the inner layers [most frequently in the bipolar and nerve fibre layers] but always

present in the outer layers as well. Said that he hems ranged from small scattered foci to severe or widespread.

Vitreoretinal traction and perimacular folds in the eyes of deliberately traumatized children. Massicotte SJ, Folberg R, Torczynski E, Gilliland MGF, Luckenbach MW. Opth 1991; 1124-1127. The authors reviewed the clinical and autopsy findings in three SBS victims who had perimacular folds. Two had impact trauma, one was shaken only. In each case, the vitreous had partially separated from the retina but remained attached to the internal limiting membrane at the apices of the folds and the vitreous base, implicating traction in the pathogenesis of these folds. Gives specimen photographs including two histologic photos, one showing the vitreous and internal limiting membrane attached to the crest of the fold and separated from the retina within the valley by a large subhyaloid hemorrhage. Also showing the vitreous remaining attached to the internal limiting membrane while the internal limiting membrane is pulled away from the retina. See also under "histology." A case from this article is abstracted by Leestma (2005) in his collection of all the published cases with an admitted history of shaking, as follows: "In case 2, the babysitter of the child said she had rolled off the couch and landed on her milk bottle or may have slipped while in the bathtub, hitting her head. [9 month old] The 12 year-old sitter later admitted to shaking the infant and throwing her to the floor. There were skull fractures and multiple injuries... There were optic nerve sheath hemorrhages; both eyes had perimacular folds and hemorrhages to the ora serrata in the eyes." A case from this article is abstracted by Leestma (2005) in his collection of all the published cases with an admitted history of shaking, as follows: "In case 2, the babysitter of the child said she had rolled off the couch and landed on her milk bottle or may have slipped while in the bathtub, hitting her head. [9 month old] The 12 year-old sitter later admitted to shaking the infant and throwing her to the floor. There were skull fractures and multiple injuries... There were optic nerve sheath hemorrhages; both eyes had perimacular folds and hemorrhages to the ora serrata in the eyes."

The ophthalmologist's role in diagnosing child abuse. (editorial). Andrea C. Tongue, MD. Opth 1991 Jul; 98(7): 1009-1010. Argues for a conservative approach.

Retinal findings after head trauma in infants and young children. Buys YM, Levin AV, Enzenauer RW, Elder JE et al. Ophthalmology 1992; 99: 1718-1723. (have) Up to this time there was no prospective ophthalmological study comparing accidental and nonaccidental injury. The authors undertook this study to test the hypothesis of Eisenbrey (1979) and others that RH are diagnostic of child abuse. The authors prospectively performed initial and 48-hour followup indirect ophthalmoscopy on 79 children aged 1 to 35 months admitted with head trauma (both accidental and nonaccidental). The mechanisms of head injury were

Fall from a height	51
Fall down stairs	16
Missile injury	7
MVA	2
Nonaccidental	3

The findings were correlated with history, coma score, radiographic findings.

Results of the ophth exam were normal in all 75 accidental cases. All three children who had +RH were shaken babies. They had:

- Case 1. 6 mo boy with extensive chronic SDHs and old fx femur had a single dot hemorrhage at the posterior pole of the right eye (unilateral).
- Case 2. 6 mo boy died of SDH/SAH, fx clavicle. Fundoscopy showed extensive bilateral NFL, subhyaloid, and vitreous hemorrhages, and a left retinal detachment.
- Case 3. 1 month boy with cerebral hemorrhage and infarction, coma, multiple rib fxx. Bilateral NFL and subhyaloid hemorrhages.

"A review of the literature supports our conclusion that retinal hemorrhages only occur very rarely after accidental head trauma in children." 1721 Their review considers that the following articles published up to 1992 containing stratification by accidental and nonaccidental mechanisms:

Eisenbrey, 1979

Billmire and Myers, 1985
 Luerssen et al., 1991
 Elder, Taylor & Klug, 1991
 Duhaime, Alario et al, 1992

Fundus photographs are provided showing (a) extensive retinal and vitreous hems, (b) extensive confluent RH of SBS.

“Accidental head trauma in adults also may be associated with large areas of preretinal hemorrhage that may appear clinically to be very similar to the traumatic retinoschisis of shaken baby syndrome.” (citing Toosi, 1987)

Head injury in very young children: mechanisms, injury types, and ophthalmologic findings in 100 hospitalized patients younger than 2 years of age. Duhaime A-C, Alario AJ, Lewander WJ, Schut L, Sutton LN, Seidl TS, Nudelman S, Budenz D, Hertle R, Tsiaras W, Loporchio S. Pediatrics 1992 Aug; 90(2): 179-185. 100 consecutively admitted head injury patients under 24 months. 24 inflicted, 32 possibly inflicted, 44 accidents. All had ophth. RH were found in 10: 9 abuse and one fatal high-speed MVA. All 10 RH pts had SDH. Discusses translational versus rotational mechanisms.

“...[I]t is clear that RH can occur under a variety of circumstances, including vaginal delivery, spontaneous SAH, systemic HTN, intracranial HTN, thoracic or abdominal trauma, and in-hospital resuscitation.

Whether superimposed hypoxia or ischemia with reflow exacerbates the finding remains unknown.

Traumatic retinoschisis resulting from acceleration/deceleration forces applied to the eye has also been postulated as a mechanism for RH. The latter may be particularly relevant in very young children because of the more solid consistency of the vitreous body in the infant and the stronger adhesions at the vitreoretinal interface. Threshold values for the degree of deceleration required to result in RH have not been established. Since this study was completed we have seen three additional pts with well-witnessed accidental head injuries who had acute RH...” Also note the diagnostic chart. Cited by Scott Denton & Darinka Mileusnic, 2003 (see under “Time of injury -- head”) for the proposition that simple falls from low heights rarely result in significant primary brain injury.

Fundus hemorrhages in infancy. Kaur B, Taylor D. Surv Ophth 1992; 37: 1-17. Cited by Luthert in “Why do histology on RH” (2003), along with Gilkes & Mann, 1967, as a consideration of increased ICP as the cause of RH in child abuse.

Ocular and associated neuropathologic observations in suspected whiplash shaken infant syndrome: a retrospective study of 12 cases. Munger CE, Peiffer RL, Bouldin TW, Kylstra JA, and Thompson RL. Am J Forens Med Path 1993 Sep; 14(3): 193-200. Eye histopathology on 12 infants with fatal SBS, age range 1-21 months (avg 7 mos). Four had a history of shaking. Eight were presumptive shakings. 11 had intracranial hemorrhage, of which 10 had SDH and one had only SAH. All had either ICH or cerebral edema or both. **Nine had ONSH (75%),** and all nine of these had SDH. Average survival time was 91 hours (0 to 480h). 10 had cerebral edema. Two had hemosiderin, of which one had optic nerve atrophy. **All 12 had RH.** Five had perimacular folds (42%). All 12 had multilayered RH. “The intraretinal and preretinal hemorrhages were most prevalent at the posterior pole.” Describe the topographical location in the retina.

These authors comment on numerous issues in RH pathology. These issues include:

- (1) the mechanism of ONSH (they seem to adopt the increased ICP view which they attribute to Lambert et al.(1986), but do not comment on the fact that all of their ONSH cases had ICH.
- (2) the mechanism and frequency of perimacular folds,
- (3) the source of deep RH. (They think subretinal hem is probably dissection from above, but could be coming from the choroid.)
- (4) the mechanism of RH in SBS: (see below) “Our observations fail to define further the pathogenesis of RH in the SBS...” 199 But they reject the Purtscher’s (chest compression) mechanism because it is thought to cause only superficial hems and most of theirs were deep. As far as vitreoretinal traction, they say, “While retinal folds and separation of the internal limiting membrane are likely to be caused by vitreous traction, ... it is difficult to attribute the choroidal and intrascleral hemorrhages present in our cases to vitreous traction.” 199 (42% of their babies had choroidal hems and a couple had scleral

hems near the papilla.) They seem to prefer the ICP hypothesis, saying, “The parallel incidence of ICH and cerebral edema with RH [in their cases] implicates an increase in ICP as a possible pathologic mechanism of the RH.” 199.

- (5) The impact/pure shaking issue. Only five of their cases had autopsy evidence of head trauma. They cite Elner et al. (1990) and Rao et al. (1988) for the proposition that “shaking alone may not be adequate to induce the observed ocular lesions” 199.

These authors provide a list of all the mechanisms that have been proposed for RH in SBS:

1. chest compression (Tomasi, 1975)
2. central retinal vein obstruction from disc edema (Vanderlinden, 1974)
3. vitreoretinal traction (Ober, 1980; Greenwald, 1986)
4. ICP/ICH (“Terson’s”) (Toosi, 1987; Weingeist, 1986)
5. direct head trauma (Elner, 1990; Harcourt, 1971)

Accidental head trauma and retinal hemorrhage. Johnson DL, Braun D, Friendly D. Neurosurgery 1993; 33: 231-235. 140 accidentals with severe enough injury to have either skull fracture or ICH were examined prospectively. 70 falls (no RH). The purpose was to extend previous studies of accidental injury to include accidents with forces sufficient to cause skull fracture and/or intracranial hemorrhage. Two children, both involved in side-impact car accidents, had RH associated with severe head injury. “We reasoned that if falls or casual shaking (Caffey) could cause the syndrome, then RH should be very common in moderately severe accidental head injuries. We found instead that RH was rare (2 out of 166 or 1.2%) and occurred only with severe head injuries sustained in side or lateral impact crashes.” The authors explicitly liken this mechanism to the tin ear syndrome. Conclude that “RH occurs rarely in accidental head injury and is associated with extraordinary force.” They go on to discuss why this is the case.

“The vitreous of the young eye is firmly attached by a dense web of collagen fibrils along major retinal vessels. The retinal capillary networks (one in the nerve fiber layer and the other between the inner nuclear and outer plexiform layers) are suspended between the retinal precapillary arterioles and postcapillary venules. A firm attachment also exists between the vitreous and the lens. Shaking and abrupt deceleration impels the vitreous complex back and forth, exerts traction forces on the retina, and tears the vascular attachments. The magnitude of the forces determines the extent of the hemorrhage, but the threshold for hemorrhage is not known.” But why would “abrupt deceleration” not occur in accidental impacts? These authors do not explain this. See, e.g., the 2001 NAME “Position Statement” (under “Shaken”) for an argument that falls and other accidental impacts are primarily linear deceleration, and RH is associated with angular deceleration, for unclear reasons.

Retinal hemorrhage. Rosenberg NM, Singer J, Bolte R, et al. Pediatr Emerg Care 1994; 10: 303-305

Systemic and ocular findings in 169 prospectively studied child deaths: retinal hemorrhages usually mean child abuse. Gilliland MGF, Luckenbach MW and Chenier TC. FSI 1994; 68: 117-132.

Retrospectively reviewed the autopsies on 169 children ranging from premature neonates to 9 year olds were classified as follows:

<u>Cause</u>	<u>n</u>	<u>n</u>	<u>any RH</u>	<u>peripheral RH</u>	<u>ONSH</u>
Trauma	112				
Asphyxia mechanism		19	0	0	0
BFT trunk mechanism		13	0	0	1
Cranio-cerebral trauma mechanism		80	62	50	57
Natural disease	47				
CNS disease		13	4	3	2
SIDS		13	0	0	0
Other disease		21	0	0	0
Undetermined cause (suspected abuse)	10		4	3	2

Their “cranio-cerebral trauma” group included numerous abuse cases. In fact, it included 62 that were probable abuse bc the history did not account for the injuries; in 18 the history did account for the injuries.

Under “cranioerebral trauma,” the subgroup of 62 that had any RH, 9 were accidental head trauma and 53 were inflicted head trauma.

The 50 cases of peripheral (ora serrata) RH included 2 car passengers (unrestrained back seat passengers) and one fall case.

They state that shaking and impact injury were the mechanisms for the hemorrhages in 62 cases, compared to very few for other mechanisms such as ICP (no cases), retinal vein cong (2 cases), Terson’s SAH (2 cases), Purtscher’s (no cases). They strongly support vitreoretinal traction as the underlying mechanism of this.

They concluded that, “In the absence of a verifiable history of a severe head injury or life-threatening CNS disease, retinal and ocular hemorrhages were diagnostic of child abuse.”

Pathophysiology and treatment of carbon monoxide poisoning. Hardy KR and Thom SR. J Toxicol Clin Toxicol 1994; 32: 613-629 This article is cited by Armin Ernst and Joseph D Zibrak in their review article, Carbon Monoxide Poisoning, NE J Med 1998 Nov 26; 339(22): 1603-1608 for the proposition that, “The classic triad of cherry-red lips, cyanosis, and retinal hemorrhages occurs rarely.” We have a case of +RH in CO poisoning: CC# 2002-1470, diagnosed by Dr Whiteman.

Ocular and optic nerve hemorrhages in abused infants with intracranial injuries. Budenz DL, Farber MG, Mirchandani HG, Park H, Rorke L. Ophth 1994 Mar; 101(3): 559-565. Purpose: to ascertain whether the pathology of the eye and optic nerve might be useful in determining the manner of death. Autopsies of **13 infants dying of AHT**. Divided into two groups: 9 with autopsy evidence of blunt trauma to the head, and 4 without. Controls were six SIDS cases. Findings: ONSH was present in all 13. **Multilayered RH was present in at least one eye in 11.** The location and quantity of RH did not differ as between the blunt-trauma group and the no-blunt-trauma group. One SIDS baby had a few erythrocytes in the NFL of the optic nerve in one eye. Conclusion: this pathology “may be helpful” in determining the manner of death.

“Valsalva’s hemorrhagic retinopathy, resulting from sustained closure of the glottis and a subsequent rise in intrathoracic pressure, is associated only with superficial retinal hemorrhages.” Lambert, Johnson and Hoyt, *infra*, 1986, p. 1512, citing Duane TD, Valsalva hemorrhagic retinopathy. Am J Ophth 1973; 75: 637-642. *Seemle*, Annable in Reece’s Child Abuse, p.145: “...the Valsalva mechanism is unlikely to be the only explanation [for RH in SBS] because the RH in the SBS are in all layers and not just in the superficial layers, as is true with Valsalva’s retinopathy.” (emphasis in original).

Retinal hemorrhage in the young child: a review of etiology, predisposed conditions, and clinical implications. Gayle MO, Kissoon N, Hered RW, Harwood-Nuss A. J Emerg Med 1995; 13(2): 233-239. A literature review by ophthalmologists from an ophthalmologic point of view. Gives photos showing the different types of hems. Flame hems are NFL. Dot-blot and white-centered hems are deeper RH; the white center is a central white sparkle caused by probably a bend in the internal limiting membrane, which is highly reflective in children. Thumbprint hems are preretinal (subhyaloid) hems; these may settle out as boat-shaped hems.

The rate of disappearance depends on the size of the hemorrhage. Large ones take months. (See

Covers the CPR issue as of mid-1994, considering it a serious DDX issue of RH, although he discusses in detail only the studies of Gilliland (1993) and Fackler’s piglet model (1992) which found no ev for CPR RH. He concludes that “The evidence ... for the role of CPR in the genesis of RH is sparse and would suggest that even if CPR is administered, RH should be considered secondary to cranioerebral trauma.”

Covers the accident vs. inflicted issue, concluding that, “Based on these studies, RH in young children are thought to be nearly pathognomonic for child abuse.”

Extracranial diseases: blood dyscrasias such as leukemia and sickle-cell anemia, bleeding disorders and coagulopathies, Ricketsia, CMV, falciparum, Rocky Mountain.
RH have been reported after general anesthesia and ECMO. Also a rare ocular disorder called juvenile X-linked retinoschisis.

Birth hems are often seen in the newborn but resolve in a few days.

Intracranial causes: AVM, SAH, SDH,

Covers the familiar direct ophthalmoscope, which sees only the posterior pole of the retina, and the binocular indirect ophthalmoscope, which gives a wide-angle view of the entire retinal surface if dilatation is used and is less dependent on cooperation.

Fatal intramuscular bleeding misdiagnosed as suspected nonaccidental injury. Wetzel RC, Slater AJ, Dover GJ. Pediatrics 1995; 95(5): 771-773. (have) A case of late-onset HDN in a 10 week old female presenting comatose and hypotensive shock, temp 31, breast fed, no VK given (midwife delivery at home), had presented earlier with leg bruising. Now with large numbers of fairly large RH (photo given), severe cerebral edema, small amount of subdural blood along the falx, diffuse hypodensity (black brain), hgb 3.0, PT 19.2, PTT 47, fbg n, D-dimer neg, plasma factor VII activity less than 1% of normal before FFP. Died. Autopsy no ev of GI probs.

The association of vision-threatening ocular injury with infant walker use. [letter] Mark Koser MD and Patrick A DeRespinis MD, UMDNJ, Arch Pediatr Adol Med 1995 Nov; 149(11): 1275-1276. Retinal hemorrhages seen in walker-stairs. Cited by Cindy Christian, AC Duhaime et al in 1999 article on posterior-pole RH in household accidents, one of which was a walker-stairs. See also Andrew Sirotinak's letter: [letter] The association of vision-threatening ocular injury with infant walker use. A Sirotinak. Arch Pediatr Adolesc Med 1996 Jun; 150(6): 652.

Morphometrical analysis of retinal hemorrhages in the shaken baby syndrome. Betz P, Puschel K, Miltner E, Lignitz E, and Eisenmenger W. FSI 1996; 78: 71-80 (have) A morphometrical analysis in cases of physical abuse and in controls (severe head injury, spontaneous ICH, overdose, CPR). Cases as follows:

Controls

9	falls
7	traffics
6	spontaneous ICH
2	overdose
10	SIDS CPR
2	homicidal BFT

Physical abuse

7	infants aged 2 to 9 months with inflicted head trauma +/- shaking
---	---

Results:

- 24 cases of accidental head injury had 2 cases of RH. These were
 - a 35 y o fell down stairs had monolayer RH cov 3% of retinal area
 - a 56 y o traffic had localized monolayer RH cov 1% of retinal area
- 6 cases of spontaneous ICH had 0 RH
- 2 cases of OD brain death had 0 RH
- 10 cases of SIDS CPR had 0 RH
- 7 cases of child abuse had 6 of them had massive hems involving all layers of the retina in both eyes. In the remaining case, extensive RH were found in one eye. The extent of the RH was measured in four cases and was from 19% to 73% of the retinal area.

"Since [RH] can also occur in other conditions ... the[ir] value for the diagnosis of child abuse seems to be limited. ..." 74-5.

“Studies dealing with RH in the eyes of child abuse victims describe massive bleedings partly involving all layers of the retina. (citing refs).” 75

“[O]ur results showed striking differences not only in frequency but also in the extent of RH. In cases of severe head trauma, the maximal area fraction of retinal bleeding was 3.33%, whereas the maximum values in cases of physical child abuse ranged between 19.2% and 73.2%.” 77. “The number of child abuse cases investigated in our series is rather limited but they seem to be sufficient to confirm the hypothesis that massive RH exceeding (arbitrarily defined) 20-30% of the entire retinal area cannot be explained by a single traumatic event and must be regarded as a very strong indicator for violent shaking.” 77

Ocular and cerebral trauma in non-accidental injury in infancy: underlying mechanisms and implications for paediatric practice. Green MA, Lieberman G, Milroy CM, Parsons MA. Br J Ophth 1996; 80: 282-287. A forensic pathology study of the eyes and brains of 23 fatal AHT children in UK. 68% had ONSH. 63% had retinal detachment. 75% had subhyaloid hemorrhage. 75% had intraretinal hemorrhage. They tried to rank these by degree of trauma, suggesting the following rank-ordering from lowest to highest amount of force:

SDH
Subhyaloid, intraretinal, perineural hems
Retinal detachment
Choroidal and vitreous hems with cerebral lacerations

This art is cited by the NAME Ad Hoc Committee on SBS (2001) for the proposition that increasing severity of RH correlates with increasing severity of head trauma. Cited by Piatt, 1999 for the same proposition, and for the proposition that the physical mechanisms [note plural --JKR] “remain to be clarified.”

Bilateral subinternal limiting membrane hemorrhage with Terson syndrome. Friedman SM, Margo CE. Am J Ophth 1997 Dec; 124(6): 850-851. Reports bilateral dome-shaped posterior-pole subhyaloid hems in a pt with TS, **examined histologically.**

Unilateral retinal haemorrhages in non-accidental injury. Tyagi AK, Willshaw HE, Ainsworth JR. Lancet 1997 Apr 26; 349: 1224. Reporting three very young infants (5 weeks). Case 1 a 5 week male presenting with sz, SDH's of different ages, clefts in the frontal lobes, extensive RH and dense perimacular hems in the R eye; the L eye was normal. He does not say which side the SDH was on. Two more infants with unilateral RH briefly mentioned; these two infants developed permanent blindness due to optic nerve and occipital-lobe injury. “The mechanism of [RH in NAI] is uncertain but is probably related to raised intraocular venous pressure due to a sudden rise in intracranial pressure or raised central venous pressure. (citing Kaur and Taylor, 1992). [His proposed mechanisms fail to account for the unilaterality. Suggest these RH were probably ipsilateral to a large ICH. See also Paviglianiti et al., 1999; Drack et al., 1999. --JKR]

Significance of white-centered retinal hemorrhages in the shaken baby syndrome. Kapoor S, Shiffman J, Kiang E, Li H, Woodward J. Pediatr Emerg Care 1997; 13: 183-185 Six cases of SBS that had white-centered hemorrhages, a finding traditionally thought to be characteristic of infective endocarditis (Roth spots). Alex Levin comments in Child Abuse Quarterly for October 1998: “Despite the classic teaching that Roth spots are associated with SBE, these authors correctly add yet another diagnosis to the long list of possible causes of the white-centered RH. Virtually any condition that causes RH is associated with white-centered hemorrhages. This may be due to the presence of a central septic embolic focus, central fibrin, central resolution, collection of neoplastic cells, or even the light reflection off the center of the hemorrhage, particularly when it is pre-retinal...”

Subdural hematomas in children under 2 years: accidental or inflicted? A 10-year experience. Tzioumi D and Oates RK. Child Abuse & Neglect 1998 Nov; 22(11): 1105-1112. In this 10-year retrospective review of 38 children with SDH, had 55% NAI, 39% ACC, 6% SPONT. The four things that predicted NAI were:

1. Young age

2. RH
3. Rib & long bone fractures
4. Delayed rescue

84% of the non-accidental had RH, none of the accidental or spontaneous. See under Fall versus Inflicted

Bruising in non-accidental head injured children: a retrospective study of the prevalence, distribution and pathological associations in 24 cases. Atwal GS, Ruttly GN, Carter N Green MA. FSI 1998 Sep; 96(2-3): 215-230. 96% had RH. Almost all the bruising was on the face & forehead, very little on the chest & limbs. Gripping doesn't cause bruises.

RH in SBS. Oral research presentation by Dr. Alex Levin, MD, FAAP, FAAO, FRSC, The Hospital for Sick Children, University of Toronto. The Second National Conference on Shaken Baby Syndrome, Salt Lake City, September 14th, 1998 (have abstract).

1. The different types and locations of RH were discussed, including flame, dot-blot, and white-centered hemorrhages. Flame hemorrhages are limited to the nerve fiber layer and are nonspecific. White-centered hemorrhages are perivascular and are due to vasculopathy of any type, are nonspecific and non-traumatic. Dot-blot hemorrhages are traumatic and are the type seen in SBS. They are in the deeper layers of the retina. Dots are the smaller ones, blots the larger ones.

Location is important in differential diagnosis. The posterior pole (the area surrounding the macula and optic nerve) is the site of most nonspecific hemorrhages. Hemorrhages limited to the posterior pole are of little diagnostic value. Hemorrhages located farther out toward the periphery of the retina are much more significant from the point of view of diagnosing trauma.

The diagnostic picture of inflicted head trauma in an infant is that of numerous dot-blot hemorrhages located in the periphery of the retina, with or without traumatic retinoschisis. Clinical recognition of this pattern requires that the pupils be pharmacologically dilated and an indirect ophthalmoscope used. Any other pattern is nondiagnostic.

Traumatic retinoschisis is splitting of the retina between its layers. Ophthalmoscopically it is recognized acutely as a dome of blood pushing the vitreous humor forward. Later, after this hematoma resolves, it leaves a circular ridge in the retina where the edges of the hematoma were. This is often called a perimacular or retinal fold. In a postmortem fixed eyeball specimen it has to be distinguished from folds due to fixation artifact, which are not circular and usually more or less radiate from the optic nerve. Traumatic retinoschisis is caused by traumatic vitreoretinal traction, and in the proper clinical setting is highly specific for inflicted head trauma such as SBS. It tends to occur at the posterior pole because in infants this is an area of tight attachment of the vitreous humor to the internal limiting membrane of the retina; when the vitreous is accelerated forward, it pulls the superficial layers of the retina in this area forward with it, resulting in splitting of the retina.

Dr. Levin discussed birth hemorrhages, hemorrhages seen in accidental head trauma, and the incidence of hemorrhages after CPR in infants.

- a. Birth hemorrhages are very common (about 40% of newborns) and occur in both vaginal and abdominal deliveries. They are flame hemorrhages and always resolve completely in less than six weeks (usually about half that time). They are nonpathologic. He did not comment on whether they leave a deposit of hemosiderin.
- b. Hemorrhages seen in accidental HT are variable in type and location, but are generally few in number, limited or mostly limited to the posterior pole, and consist of flame hemorrhages or a mixture of flame, dot-blot, and preretinal or subhyaloid hemorrhages. The above-described pattern of inflicted head trauma RH is essentially never seen in accidental head trauma of infants.
- c. RH very rarely occur in CPR. There are several published articles, all of which support this, and another body of similar research was presented at this Conference.

In the rare case where RH occur in CPR (and are not due to some other disease or condition), they are few in number, limited to the posterior pole, and small in size. In other words, a pattern that cannot possibly be mistaken for SBS hemorrhages.

2. It is impossible to "date" retinal hemorrhages. They resolve at varying rates, depending on the size of the hemorrhages and the clinical condition of the patient.
3. Dr. Levin has performed a prospective study of autopsy eye specimens in which orbital exenteration, rather than enucleation, was performed. Orbital exenteration consisted of autopsy extraction en bloc of the entire orbital contents and sectioning thereof after fixation. In SBS cases this demonstrated hemorrhage of the orbital soft tissue away from the optic nerve. This hemorrhage was often adjacent to other cranial nerves or their branches, but also occurred in orbital fat. The meaning of this is that ONSH is not due, or certainly not solely due, to transmitted hemorrhage from within the cranial cavity. Rather, it results from direct mechanical forces impinging upon the orbital contents. Dr. Levin advises that in such autopsies, orbital exenteration be adopted as standard procedure, with routine histologic sectioning in the coronal plane of the posterior orbital tissue block.

Neuroimaging, physical, and developmental findings after inflicted and non-inflicted traumatic brain injury in young children. Ewing-Cobbs L, Kramer L, Prasad M, Canales DN, Louis PT, Fletcher JM, Vollero H, Landry SH, Cheung K. Pediatrics 1998 Aug; 102(2): 300-307.

In this prospective clinical comparison of 20 accidentally injured children (mostly automobile accidents) to 20 inflicted children, RH were present in 14 of the inflicted group (13 bilateral and 1 unilateral) and 0 of the noninflicted group. But only some of the inflicted group and none of the noninflicted group had funduscopic examinations. 14 inflicted had RH NOS -- 13 bilateral and 1 unilateral.

They showed other dimensions of comparison between the two groups. See below under "Shaken." This paper is important because it's one of the only direct prospective comparison studies with controls.

Can convulsions alone cause retinal hemorrhages in infants? Tyagi AK, Scotcher S, Kozeis N, Wilshaw HE. Ophthalmology 1998; 82: 659-660. Prospective study of 32 pediatric admissions under the age of 2 (average age 12 mos) to Birmingham UK Hospital with history of seizures, either (10) epileptic or (22) febrile. None had RH. This study complements their earlier study of 32 sz pts age 4 mos to 14 years. This study limits the age to under 2 years, same results.

Can convulsions cause retinal haemorrhages in infants? Tyagi AK, Scotcher S, Kozeis N, Willshaw H. Can J Ophth 1998; 82: 659-660. Likelihood less than 5%.

From Duhaime et al. Review article on shaken impact syndrome, NEJM 1998, p 1823:

"RH ... are found in 65 to 95 percent of (SBS) patients. The hems may be unilateral or bilateral, and retinal folds or detachments may be seen. The exact biomechanical forces necessary to cause retinal hemorrhages are unknown, but several mechanisms have been postulated, including increased retinal venous pressure, extravasation of subarachnoid blood, and traction of retinal vessels at the vitreoretinal interface due to angular deceleration. Although strongly associated with inflicted head injury, RH are not specific for the diagnosis, nor can they be dated with precision. Such hems have been reported in some cases of accidental trauma (especially SDH) and, in rare cases, after resuscitation; they can also occur with papilledema. RH are seen in up to 40 % of vaginally delivered newborns but resolve by one month of age. Nontraumatic causes include SAH, sepsis, coagulopathy, galactosemia, severe HTN, and other rare conditions. The dx of inflicted head injury cannot rest on the finding of RH alone, but the finding of severe bilateral RH with retinal folds or detachments is particularly suggestive of the diagnosis."

Peripheral RH: a literature review and report on thirty three patients. Tolls DB. J Am Optom Assoc 1998 Sep; 69(9): 563-574. These are mostly adults with nontraumatic RH picked up on office exam. DDx incl senility, vascular disease, hematologic dsos, infections, hypoxia, and "mechanical and iatrogenic causes."

Child abuse and the eye. The Ophthalmology Child Abuse Working Party of the Royal College of Ophthalmologists. Eye 1999; 13: 3-10. A review article summarizing the literature up to the end of 1997.

Precis: Severe retinal hemorrhages in infancy are rarely caused by anything other than abusive head trauma. The severity of RH varies in proportion to the severity of intracranial injury; the two are linked by a common etiology, which is violent tangential acceleration of the head (estimated 6000 R/s/s) with or without impact to the head. Vitreoretinal traction is believed to be the mechanism of severe RH in the trauma setting. Severe forms of RH such as subretinal hemorrhage, subhyaloid hemorrhage, retinoschisis, retinal folds, and retinal cysts are strongly associated with abuse and with severe head trauma caused by abuse. There is no form of RH, however, that is pathognomonic of abuse. Birth hemorrhages are discussed also.

1. What forces are needed to produce RH?

Conclusions: (1) RH are not reported in quite severe household falls, even when associated with skull fractures, but occur after very severe motor accidents. (2) RH is associated with types of cerebral injury that have been shown experimentally to require severe concussive forces. (3) Comparing the severity of the cerebral lesions with that of the RH tells us that “the finding of RH with additional features, such as perimacular folds, subretinal hems, choroidal hems, and vitreous hem, indicates a greater severity of applied force.

Details: Severe angular acceleration, or angular acceleration severe enough to produce severe brain injury. “There is reliable evidence to show that retinal damage can be produced by severe angular acceleration forces alone, without impact and without direct ocular injury. It has been suggested that this may result from direct vitreous traction on the retina.” (citing Green, 1996). No absolute value available, but the simian model of Ommaya predicts 6,000 R/s/s for severe intracranial injury. There is a good correlation of the severity of RH with the severity of intracranial injury. This answer is based on (a) RH seen in severe aircraft ejection, road accidents, bungee jumpers, and confessed infant shakings; (b) subdurals produced by nonimpact whiplash in monkeys and infants; (c) lack of RH in accidents unless very severe impact. “Much of the indirect evidence linking brain and eye injury is based on the good correlation between the severity of ocular injury and the severity of the intracerebral injury. This enables information gathered on the forces required for cerebral injury to be extrapolated to eye injury.” They then go into a very brief capsule discussion of head-trauma biomechanics: (a) monkeys got SDH and cerebral tears from whiplash alone when and only when they suffered a clinical concussion. (citing Ommaya, 1968). (b) the failure of the doll model to achieve SDH thresholds “may have reflected deficiencies in the model construction.” (c) not all infants with RH have impact injury. Citing Green, 1996; Gilliland & Folberg, 1996; Guthkelch, 1971; Hadley, 1989. (d) Duhaime, Alario, 1992 finding that in human infants, tangential acceleration caused much more severe brain deformation and shear than linear acceleration; “This may explain the propensity for RH to occur with shaking injury.”

2. Is impact necessary?

No, based on Guthkelch, 1971; Frank, 1985; Hadley, 1989; Green, 1996; Gilliland & Folberg, 1996, finding RH in shaken babies without known impact. But “Blunt trauma, in addition to angular acceleration, is probably necessary to produce *the most serious problems* in child abuse [because] the deceleration produced in impact injuries is much greater than [shaking].” Is this a statement that pure shaking is nonfatal, or that it doesn’t cause severe RH or severe eye damage? Not likely, because Gilliland & Folberg dealt with fatal cases.

3. What is the usual site and extent of RH in child abuse?

Haemorrhages are seen mostly at the posterior pole (Munger et al, 1993); however, the retinal periphery is often difficult to examine clinically. An autopsy study (Green et al., 1996) demonstrated that subretinal and subhyaloid haemorrhages were more common in the peripheral retina than at the macula or around the optic disc, and least common at the equator, but this pattern has not been assessed in survivors... Typically all layers of the retina are involved (Jensen et al, 1971; Mushin et al, 1971; Rao et al, 1988; Riffenburgh & Sathyavagiswaran, 1991; Riffenburgh & Sathyavagiswaran, 1991; Budenz et al, 1994;

Green, 1996). Betz, Puschel et al. Finding that RH greater than 20-30% of the retinal area require abusive shaking, not accidental impact. "Although it is much more likely that massive RH are caused by NAI, it cannot be said that *less* extensive haemorrhages are *less* likely to be associated with NAI."

4. Are any ocular fundus findings pathognomonic of child abuse?
 "Severe RH and perimacular folds are frequently seen in NAI, but they cannot be said to be pathognomonic (citing Tongue, 1991) as they occur in severe accidental injury and Terson's syndrome. However, they are much more likely to be seen in child abuse." The presence of perimacular folds may suggest that there have been cycles of acceleration and deceleration... Haemorrhagic retinal cysts and retinoschisis are probably much more frequent in child abuse than in accidental trauma." As noted above under Paragraph 1, comparing the severity of the cerebral lesions with that of the RH tells us that "the finding of RH with additional features, such as perimacular folds, subretinal hems, choroidal hems, and vitreous hem, indicates a greater severity of applied force."
5. Can intraocular haemorrhages increase after the injury?
 Yes. Analogy drawn to enlarging intracranial hematomas with a suggestion that the two form parts of a unitary pathologic process.
6. Can accidental injury cause RH?
 Rarely. "RH were not associated with witnessed accidental trauma unless it had been very severe." Citing Duhaime, Alario et al, 1992, Head injury in very young children: mechanisms, injury types, and ophthalmologic findings in 100 hospitalized patients younger than two years of age, for the proposition that of 100 consecutive adm under 2 yrs with 73 accidents and 24 abuse. Only one RH in accident, and that was a high-speed road accident. 9 RH in abuse cases. Citing Buys, Levin, 1992 for the proposition that in 79 head-injured children, all of 75 accidents had normal fundi while all 3 abuse had RH. Mentioning per Duhaime Alario that tangential acceleration gives much more brain shear and probably more RH.
7. For how long can birth-related haemorrhages persist?
 The finest NFL hemorrhages can disappear in 24h, whilst even extensive NFL hemorrhages are usually gone within a few days. Half of all hemorrhages disappear within 48h and Sezen found that 2.6% persisted at 72-120h after birth. In one case the haemorrhage persisted for 6 weeks. Larger subhyaloid and larger intraretinal haemorrhages seem to persist for the longest time. Conclusion: "The majority disappear by 8 days but some dome-shaped ... haemorrhages may persist for up to 3 months."
8. Is unilateral RH compatible with child abuse?
 Yes.
9. Is intracranial damage always accompanied by RH?
 No, but there is a close correlation.
10. Can a bleeding diathesis...cause RH similar to those seen in child abuse?
 Yes.
11. What other conditions of childhood may have RH?
 Leukemia, HDN, ROP, SSA, ECMO, Galactosemia, Henoch, Maternal cocaineism, Meningitis, AVM, X-linked, ICP, Intraocular surgery, HTN. 14 entities.
12. Can seizures cause RH?
 No.
13. Can CPR cause RH?

Very unlikely.

14. Can prolonged vomiting, gagging, etc. cause RH?

No.

15. Is it possible to determine, from an examination of the retina, the time at which an injury occurred or whether there have been haemorrhages at more than one time?

Long discussion dwelling mostly on hemosiderin. "If hemosiderin is found in the relatively vascular eye tissues in infants of more than 2-3 months old it is likely that this is from a haemorrhage occurring after birth." Query what is the value of this discussion: retinal hemosiderin cannot be seen through the ophthalmoscope. Its diagnostic significance in histologic specimens of retina is unknown.

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 sh R subretinal and preretinal hems 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

Letters by Coats and Lynch (J Pediatr 2000 Apr; 136(4): 564-565) Coats comments that (a) the contrast between the minor posterior RH seen by the authors and the major RH typical of AHT "need[s] to be made crystal clear," in order not to confuse workers and the courts. (b) the forces involved in the authors' three household accidents could have been extreme; two were falls from a height and the third sounds like an accelerated impact; they were not typical of household falls, and certainly were not trivial trauma. RH from accidental trauma involving extreme forces has been reported before (citing Johnson, Braun, Friendly, 1993.) One must be prepared by drawing these distinctions to counter the use of this article by defense counsel. Lynch wonders whether these really were accidents.

Late-form hemorrhagic disease of the newborn: a fatal case report with illustration of investigations that may assist in avoiding the mistaken diagnosis of child abuse. Ruddy GN, Smith CN and Malia RG. Am J Forens Med Path 1999 Mar; 20(1): 48-51. Hospital blood studies showed greatly prolonged PT PTT with a normal thrombin time and normal fibrinogen in this 9 week old Asian male who presented jaundiced and unconscious with failure to thrive and a 10-day history of vomiting and diarrhea. CT showed an acute SDH with midline shift and infarction of the underlying parietal lobe. He underwent craniotomy and evacuation but died in 24 hours. Autopsy showed a R sided SDH and no head bruises. Neuropath showed widespread bilateral perivascular and white-matter hemorrhages not typical of NAI. Eyes showed extensive bilateral RH. Optic nerves not reported. Baby had gastritis and hepatitis. The pathologists performed assays of Factors II, VII and X on frozen hospital blood -- these are vitamin K-dependent factors. All three were diminished. Factor V was normal. Further assays confirmed that this was due to VK deficiency or antagonism. Warfarin intake was ruled out. This baby had received prophylactic VK at birth. There is a late-onset form of HDN; it always presents with intracranial hemorrhage (84-100% subdural). It can be caused by gastroenteritis and liver disease. RH can occur in vitamin B deficiency or vitamin K deficiency.

Intra-ocular pressure changes during maximal isometric contraction: does this reflect intra-cranial pressure or retinal venous pressure? Dickerman RD, Smith GH, Langham-Roof L, McConathy WJ, East JW, Smith Ab. Neurological Research 1999 Apr; 21(3): 243-246. From the abstract: "Recent publications have suggested that intra-ocular pressure (IOP) may be an indirect assessment of intracranial pressure. ... Power athletes routinely utilize the Valsalva maneuver during weightlifting. In fact there are reports of stroke, cerebral hemorrhage, subarachnoid hemorrhage, conjunctival, foveal, and retinal hemorrhage,

retinal detachment, hiatal hernia, and pneumothorax associated with weightlifting. ... To date no human studies have examined the IOP changes with heavy resistance exercise. We (took) 11 power athletes ... Resting IOP 13 +/-2.8 mmHg. Raised to 28 +/- 9.3 (P=.0001).

Bilateral massive retinal hemorrhages in a 6-month-old infant: a diagnostic dilemma. Fenton S, Murray D, Thornton P, Kennedy S, O'Keefe M. Arch Ophthalmol 1999 Oct; 117(10): 1432-1434.

Bilateral periopticointrascleral hemorrhages associated with traumatic child abuse. Lin KC and Glasgow BJ. (Jules Stein Eye Institute UCLA). Am J Ophthalmol 1999 Apr; 127(4): 473-475. A 6 month old who died of SDH. Step sections of the eyes revealed scleral hemorrhages around both optic nerves.. Also diffuse multilayered retinal, vitreous, and subhyaloid hemorrhages. Conclusion: periopticoscleral hemorrhages are characteristic of blunt head trauma and may constitute important forensic evidence of child abuse.

Late-form hemorrhagic disease of the newborn: a fatal case report with illustration of investigations that may assist in avoiding the mistaken diagnosis of child abuse. Ruttly GN, Smith CN and Malia RG. Am J Forensic Med Pathol 1999 Mar; 20(1): 48-51. Hospital blood studies showed greatly prolonged PT PTT with a normal thrombin time and normal fibrinogen in this 9 week old Asian male who presented jaundiced and unconscious with failure to thrive and a 10-day history of vomiting and diarrhea. CT showed an acute SDH with midline shift and infarction of the underlying parietal lobe. He underwent craniotomy and evacuation but died in 24 hours. Autopsy showed a R sided SDH and no head bruises. Neuropath showed widespread bilateral perivascular and white-matter hemorrhages not typical of NAI. Eyes showed extensive bilateral RH. Optic nerves not reported. Baby had gastritis and hepatitis. The pathologists performed assays of Factors II, VII and X on frozen hospital blood -- these are vitamin K-dependent factors. All three were diminished. Factor V was normal. Further assays confirmed that this was due to VK deficiency or antagonism. Warfarin intake was ruled out. This baby had received prophylactic VK at birth. There is a late-onset form of HDN; it always presents with intracranial hemorrhage (84-100% subdural). It can be caused by gastroenteritis and liver disease. RH can occur in vitamin B deficiency or vitamin K deficiency.

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. (Available on Medscape). Dr. Piatt, a university neurosurgeon from Portland, presents a 4 mo infant who presented to an outlying hospital with status epilepticus and a history of macrocephaly. At six weeks his head circumference had been 41.5 cm (>95%). The history was that his mother was propping him up in a standing position against a piece of furniture. The father and grandmother were present. The mother got distracted and let the infant fall from his own height and hit the back of his head on the carpeted floor. Infant cried and then began a generalized seizure which progressed to status epilepticus. Emergency room evaluation revealed no injuries outside the head; skeletal survey was negative. Head CT revealed a small acute left frontal subdural hematoma that was associated with bilateral low-density extracerebral fluid collections. On Day 3, MRI showed that these fluid collections reflected diffuse enlargement of the subarachnoid space. The MRI is shown as figure 2. The caption to figure 2 reads as follows:

“Magnetic resonance imaging studies. Upper: A transaxial T1-weighted image of the head demonstrating a small left frontal subdural hematoma of mixed intensities (white arrow heads). There is a prominent, symmetrical, extracerebral fluid collection of the same intensity as ventricular cerebrospinal fluid (CSF) (white asterisk). Lower: coronal, T2-weighted image demonstrating that the extracerebral fluid remains at the same intensity as ventricular CSF, and anastomotic veins can be seen passing from the surface of the cortex to the dura through it (black arrows). The extracerebral fluid therefore represents enlargement of the subarachnoid space. These images are diagnostic of external hydrocephalus.”

Funduscopy examination revealed what are described as retinal, preretinal, and subhyaloid hemorrhages. A handwritten diagram of these is shown as figure 1; the caption of figure 1 reads as follows:

“Fundus drawings demonstrating extensive, deep blot hemorrhages in all four quadrants of both eyes. In this case report there were preretinal and subhyaloid hemorrhages in each eye as well.

Both optic nerve heads were well perfused and had sharp margins. There were no abnormalities of the nerve fiber layer. (Drawings courtesy of William Rodden, M.D., Ashland, Oregon.)” *

The patient recovered without specific treatment. An extensive police and social service investigation including polygraph testing failed to uncover any risk factors or circumstances suspicious for child abuse. The retinal hemorrhages cleared in 3 months and the subhyaloid hemorrhages cleared in 6 months. Neurosurgical followup showed persistent asymptomatic macrocephaly and external hydrocephalus. Development out to 44 months was normal.

The author hypothesizes that the longer course of the bridging veins through an enlarged subarachnoid space rendered them more vulnerable to shearing injury in a minor fall. This is his explanation for SDH in such a minor injury. [It harks back to the theories of Aoki. –JKR]

As to the RH, Dr Piatt gives the following discussion: “The association of retinal hemorrhage with catastrophic intracranial hemorrhage is familiar to all neurosurgeons. Aneurysmal subarachnoid hemorrhage is the paradigm, and the accepted mechanism is transmission of the sudden elevation of intracranial pressure to the central retinal vein and its choroidal anastomoses via the subarachnoid space of the optic nerve sheaths. (citing Khan & Frenkel, 1975 and Muller & Deck, 1974). In infancy, retinal hemorrhages have long been recognized as a distinctive feature of head injury caused by physical abuse, but the physical mechanisms remain to be clarified. (citing Green, Lieberman, Milroy et al., 1996). One possibility is that rupture of retinal veins is caused by a pressure surge transmitted either from the cranial cavity through the subarachnoid space of the optic nerve sheaths or from the chest through the cervicocephalic veins (the so-called Purtscher retinopathy). (citing Tomasi & Rosman on Purtscher retinopathy in the battered child syndrome, 1975). Another possibility, by analogy with subdural hemorrhage, is that a direct acceleration-deceleration insult to the globe causes shear stresses between the ocular tissues with differing mechanical properties; this mechanism of injury is especially prominent in autopsy cases. (citing Green, Lieberman, Milroy, 1975 and Massicotte, Folberg, Vitreoretinal traction, 1991). Because a gradation of inflicted ocular injury corresponds to the gradation of severity of the associated intracranial injury (citing Green, Lieberman and Wilkinson, Han, Rapley, Retinal hemorrhage predicts neurologic injury in the shaken baby syndrome, 1989), different mechanisms likely come into play in response to varying degrees of violence.”

As to the mechanism of the RH in the instant case, Dr Piatt proposes the hypothesis that the enlarged cerebral subarachnoid space might have been accompanied by an enlarged subarachnoid space of the optic nerves. Thus “a minor skull impact could generate an exaggerated intracranial pressure impulse that might, in turn, be transmitted undampened out along the optic nerves and into the orbits as far as the globes.”

[* Note by JKR: Dr Piatt is a faculty neurosurgeon at the University Hospital in Portland. It would appear that Dr Piatt or his staff saw the child in consultation at age 6 months (4 months after the injury). It would appear that Dr Rodden is the ophthalmologist who examined the patient at the outlying hospital in Ashland, which is about five hours drive south of Portland. It is not specified whether the drawing published was made at the bedside, redone from one made at the bedside, or drawn later from memory. To my eye, the drawing shows numbers of dot-blot hemorrhages appearing to be posterior to the equator, a boat-shaped preretinal hemorrhage about 1 DD below the disk OD, another probable preretinal hemorrhage 1 DD above the disk OD, and two probable subhyaloid or larger preretinal hemorrhages close to the disk, one OD and one OS.]

This paper is criticized by Dr Levin and others on the following grounds: first, that the retinal hemorrhages were never documented and never seen by an ophthalmologist [which appears not to be the case --JKR]; Secondly, that Dr. Piatt himself did not see the patient at the time of initial presentation, but only by referral later. [Not clear what would be the scientific relevance of this second objection --JKR]. Dr. Reece in his review comments that he also has seen infants in whom apparently minor trauma produced SDH and RH. Dr. Reece says more study of such cases is needed.

Chapter by Alex Levin in: *Recent Advances in Paediatrics – 18*, London: Churchill Livingstone, 1999. ISBN 044306430X. Comprehensive review.

Unilateral RH and ipsilateral intracranial bleeds in nonaccidental trauma. J AAPOS 1999 Dec; 3(6): 383-384. Presents three cases.

Unilateral RH in documented cases of child abuse. Drack AV, Petronio J, Capone A. Am J Ophth 1999; 128: 340-344. Presents four cases: ages 5mos to 17 mos.

- 1 6mo w L hemispheric infarct, L neck & eyelid ecchy. Unil L dot-blots
- 2 5mo apneic w L SDH old rib fx, R perim fold (yes R) Confessed shake
- 3 17m sz bilat SDH diffuse posterior-pole intraretinal RH R, ecchy ar R orbit
- 4 6mo preemie w pmh ROP fd at 6m to hv L postr pole intrar + macular preret bilat old and recent SDH, mult bone fxx, recent change of caregiver

A 12-year ophthalmologic experience with the shaken baby syndrome at a regional children's hospital. Kivlin JD. Trans Am Ophth Soc 1999; 97: 545-581. Prospective series of 116 ch admitted 1987-1998. 84% had RH. **"No fundus finding is pathognomonic for SBS."** "When RH are found in young children, the likelihood that abuse occurred is very high."

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 trauma admissions to Tufts Univ Hosp aged 1 week to 6 ½ years. See <http://www.archpediatrics.com/>

Type of injury	Abused	Acc
SDH	46% had	10% had
SAH	31% "	8% "
RH	33% "	2% " * **
Skin injuries	50% "	16% "

* No RH in accidental falls of under 4 feet. "RH are, if not diagnostic, compelling findings; most are seen in abusive head trauma."

** 18/54 AHT had RH, while 5/233 Acc HT had RH. Of these 5, 4 had obvious mechanism such as GSW face, fall from a great height, or MVA.

Child abuse and unintentional injuries: a 10-year retrospective. DeScala C, Sege R, Li G, Reece R. Arch Pediatr Adol Med 2000 Jan; 154: 16-22. See under Epidemiol. RH were 27.8% of abused vs .06% of accidentals. (The accidentals were overwhelmingly falls and MVA's.) RH were in 18 out of 54 abused chil and 5 out of 233 accidental chil: of these 5, 4 were GSW, fall from a height, or MVA. See Dr. Sege's discussion on Medscape at <http://www.medscape.com/medscape/pediatrics/>

Hyponatremic seizures in infancy: association with RH and physical child abuse? Krugman SD, Zorc JJ, Walker AR. Pediatr Emerg Care 2000 Dec; 16(6): 432-434. Two infants with inflicted hypon and the unexpected finding of RH. One was clearcut SBS with long bone fxx and SDH. The other had only cerebral edema and neglect – no intracranial blood. Thought to have been shaken.

Shaken baby syndrome: retrospective eye findings in 110 shaken babies. Kivlin JD, Simons KB, Lazoritz S, Ruttum MS. Ophth 2000 Jul; 107(7): 1246-1254. 123 shaken babies under three restrospectively reviewed, including 110 with clinical ophthalmology performed and 23 deaths with autopsies. The postmortem findings were:

Bilat RH	89%
RH at ora	77%
Traumatic retinoschisis	15%

The presence and extent of RH was significantly underdiagnosed clinically compared with postmortem. Dr Reece included this paper in his list of the top ten SBS papers at the 2006 National Shaken Baby Conference. His review includes the following data from combined survival and autopsy material:

clinical (n=96)	autopsy (n=27)
-----------------	----------------

bilat	68%	89%
unilat	11%	8%
preretinal	38%	42%
subretinal	10%	38%
VH	14%	63%

23 babies were autopsied. Postmortem eye findings included 42% with preretinal hemorrhages, 38% with subretinal hemorrhages, 25% with papilledema, and 63% with vitreous hemorrhage.

Non-ophthalmologists had a 29% false negative rate in detecting RH.

A lot of discussion of neurological outcome and visual outcome.

Dr Reece gives this quotation from the article's conclusions:

"The mere presence of any retinal hemorrhage is adequate to raise the concern of shaken baby syndrome."

See below under "Shaken" for some other more detailed discussion of this article.

RH and accidental household trauma. (letter) Lynch R. J Pediatr 2000 Apr; 136(4): 565.

Bilateral pre-retinal macular haemorrhage following blunt head injury. Eye 2000 Oct; 14 Pt 5: 807-808.

Fatal pediatric head injuries caused by short-distance falls. Plunkett J. Am J Forens Med Path 2001 Mar; 22(1): 1-12. Reports 18 cases of fatal pediatric short-fall head injuries culled from US government databases. He presents them in ascending order of age, from 12 months to 13 years. These were all witnessed falls, many on playground equipment. Fall distances ranged from 2 feet to 10 feet with most around 2 – 3 feet. Thirteen had SDH; of these, 7 had interhemispheric. None had DAI. Four (4) had RH; of these, 3 had bilateral multil, one had NOS. All the RH occurred in cases with SDH. He states that, "RH may be caused experimentally either by ligating the central retinal vein or its tributaries or by suddenly increasing intracranial pressure." (citing Smith, Kearns & Sayre, 1957 and Lehman, Krupin, Podos, 1972.) "Any sudden increase in ICP may cause RH." (citing Kirshner, "mistaken dx," 1985; Weedn, RH after CPR, 1990; David and Jain on bungee jumping, 1994.) "Vasospasm secondary to traumatic brain injury selectively increases venous pressure."

Manifestations of the shaken baby syndrome. Kivlin JD. Curr Opin Ophth 2001 Jun; 12(3): 158-163.

Neuropathology of inflicted head injury in children I. Patterns of brain damage. Geddes JF, Hackshaw AK, Vowles GH, Nichols CD, Whitwell HL. Brain 2001 Jul; 124 (7): 1290-1298. Found RH in 71% of 53 AHT victims studied neuropathologically. (See under "Shaken," below). Found no case of RH without SDH.

The spectrum of postmortem ocular findings in victims of shaken baby syndrome. Can J Ophth 2001 Dec; 36(7): 377-383. See letter, Can J Ophth 2002 Feb; 37(1): 4. From Sam Gulino's review in the *Quarterly* for July 03: Six sets of autopsy eyes from SBS css, or at least what were deemed to be SBS css. Ages 1 mo to 34 mo. All had ONSH. The intracranial findings seem vaguely described. The RH are not described as to layers involved.

posterior, midportion, and anterior	4
posterior and mid	1
posterior	1
macular folds	3
posterior vitreous detachment	3
traumatic retinoschisis	1

Does soccer ball heading cause retinal bleeding? Reed WF, Feldman KW, Weiss AH, Tencer AF. Arch Ped Adol Med 2002 Apr; 156(4): 337-340. No, it doesn't. These Seattle peds prospectively evaluated 21 youth soccer players who performed 79 headers, plus 30 controls, by indirect ophthalmoscopy. Found no RH in either group. Accelerometers on heads showed that the translational accelerations were 3.7 g and the rotational accelerations were negligible.

Retinal hemorrhages in infant head injury. Clark BJ, Adams GG, Luthert PJ. Brain 2002 Mar; 125 (Pt 3): 677-678 (letter) (have). Arguing for the abrupt-increase of ICP theory of RH, and against the vitreoretinal traction theory. Commenting on Geddes I and Geddes II (2001a). Clark et al do not give a detailed argument against the vitreoretinal traction theory, but only say generally that the VRT theory is “hypothesis and not established fact,” and that “The Geddes’ investigations have challenged the importance of DAI and hence shearing forces in the brain and by implication also at the vitreoretinal interface.” They add that, “The notion that shearing forces underlie the genesis of RH is further brt into q by obs of similar or identical patterns of haemorrhage in situations where no shearing is implicated. Rapidly increasing ICP and a number of other conditions are well-documented causes of RH. It seems entirely feasible, therefore, that in shaking with brainstem injury and diffuse cerebral hypoxia with oedema, the RH arise as a consequence of intracranial pathology alone and are not a result of traumatic damage to the virterorrtnal interface...” There is a letter to the editor, and an authors’ reply. See also critical comment by Punt et al in *Pediatric Rehabilitation*, 2004.

Neuropathology of inflicted head injury in children. Parulekar MV, Elston JS. Brain 2002 Mar; 125(Pt 3): 676-677 (letter). See also Clark et al., above. Criticizing Geddes I and Geddes II (see under “Shaken”) on the ground that while hypoxia induced by damage to the craniocervical junction could account for the diffuse brain changes seen in SBS, it does not account for RH. Believing that RH has to be traumatic. “Of the theories that have been proposed for the mechanism of RH in non-accidental shaking injury, [vitreoretinal traction] is the likeliest mechanism.” Citing Duhaime, 1987, Massicotte, 1991, Green, 1996. Tracking through the optic canal “has been disproved” (citing Giangiacomo, 1988, Riffenburgh, 1991). Pointing out that RH are rarely seen after witnessed or independently verifiable single-impact accidental trauma. Therefore, RH must be due to severe angular velocity change. To which Geddes and Whitwell reply, see below.

Reply. Jennian Geddes and Helen Whitwell. Brain 2002 Mar; 125(Pt 3): 678. Replying to the above two letters, their reply is so short that it can be quoted almost in its entirety: “The fact that members of the same specialty [meaning the two correspondents, above] can hold such differing beliefs highlights the lack of certainty about the pathogenesis of intraocular bleeding in infant head injury. Our motive...was merely to point out that the NP of these cases does not always support the idea that severe traumatic injury has occurred, and to suggest that we should re-examine the scientific basis for current beliefs about what causes subdural and retinal bleeding. It is likely that in doing so, we might find that the evidence is not as strong as is often claimed.” See also Dr Geddes’ later published comments on SBS (2003) under “Shaken.” Dr Geddes has evolved the general view that shaken babies may have minimal trauma leading to apnea, with the RH resulting from increased ICP. The SDH resulting also from minimal trauma.

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. Had RH evaluated by Dr. Walsh. She discusses RH in GA-1.

Correlation between retinal abnormalities and intracranial abnormalities in the shaken baby syndrome. Morad Y, Kim YM, Armstrong DC, Huyer D, Mian M, Levin AV. *Am J Ophth* 2002 Sep; 134(3): 354-359. From the abstract and the review by Dr Reece in the *Quarterly* for January 2003: 75 cases of children 2 mos to 48 mos admitted with apparent non-accidental head trauma. No autopsies. CT and MRI findings:

Ev of impact	40%
EDH	1%
SDH	93%
SAH	16%
ICH	8%
Cerebral edema	44%

Papilledema	5%	
Confluent multilayer RH	70%	
Bilateral		81%
Traumatic retinoschisis	32%	
Ev of increased ICP*	39%	

* defined as papilledema, widespread bilateral cerebral edema; midline shift; or large intracranial hemorrhage with mass effect

Sidedness of intracranial hemorrhage and sidedness of RH were not correlated. 10% had abnl skel survey.

Conclusions:

1. Direct tracking of blood through the optic nerve sheath

Lack of correl between laterality of ICH and RH was against this.

2. Increased ICP?

Pts with “evidence of increased ICP” as above defined did not differ from the other pts.

3. Purtscher’s?

No white patches seen, even in the pts with rib fractures

4. Vitreoretinal traction?

The lack of RH correlation with any other finding, including ev of impact, can be interpreted to support this theory.

Conclusions: “Our study supports previous observations that the severity of retinal and intracranial injury is correlated in SBS. We cannot support the suggestions that in most children with SBS retinal bleeding is caused by sustained elevated intracranial, elevated intrathoracic pressure, direct tracking of blood from the intracranial space, or direct impact trauma [meaning to the eyeball --JKR]. The correlation in severity of both eye and head findings may suggest, however, that retinal abnormalities are the result of mechanical shaking forces.”

JKR: 1. It appears this is an entirely clinical-radiographic study with no autopsies. 2. Did they correlate traumatic retinoschisis with increased ICP? This article is commented on, with authors’ reply, in: Correlation between retinal abnormalities and intracranial abnormalities in the shaken baby syndrome. (letter) Raj A. Am J Ophth 2003 Oct; 136(4): 773-774, and in Retinal hemorrhages in children. (letter) Gardner HB. Ophthalmology 2003 Sep; 110(9): 1863.

Ophthalmology of shaken baby syndrome. Levin AV. Neurosurg Clin N Amer 2002 Apr; 13(2): 201-211. See also under “Shaken” for other articles in this book. Here Dr Levin again rehearses the need for a thorough description of the number, type, and distribution of RH and that “numerous pre-retinal, intraretinal, and subretinal hemorrhages extending out to the edges of the retina and/or splitting of the retina seem to be particularly indicative of shaking with a very narrow differential diagnosis. Shaking appears to be a key element in creating hemorrhagic retinopathy.”

Retinal findings in children with intracranial hemorrhage. Schloff S, Mullaney PB, Armstrong DC, Simantirakis E, Humphreys RP, Myseros JS, Buncic JR, **Levin AV.** Hospital for Sick Children, Toronto. Ophth 2002 Aug; 109(8): 1472-1476. (have) See under Terson’s syndrome, below.

Ocular injuries in shaken baby syndrome. Tsao K, Kazlas M, Weiter JJ. Int Ophth Clin 2002 Summer; 42(3): 145-155.

Prevalence of retinal hemorrhages and child abuse in children who present with an apparent life-threatening event. Pitetti RD, Maffei F, Chang K, Hickey R, Berger R, Pierce MC. Pediatrics 2002 Sep; 110(3): 557-562. Prospective study of 128 ER admissions under 24 months with ALTE, covering a two-year study period. Obvious child abuse victims were excluded. They did full child abuse workups on all, with dilated funduscopy performed on 73 of the pts. Their diagnostic yield was:

3	abuse cases	=	2% of all subjects (128)
1	RH	=	1% of all who had funduscopy (73)

Of these results, the authors conclude, "We recommend considering the dx of child abuse in all pts who present with an ALTE. ...We strongly recommend performing a dilated funduscopy examination on children who present to the ED after an ALTE."

Details: Patient 1 a 2 week old neonate with a complaint of apnea and color change, normal on examination, funduscopy examination normal, admitted for rule out sepsis, CT showed acute and chronic (!) SDH's. A small old bruise on the face and a sublingual abrasion. No history of difficult birth. Child abuse ruled in by ? (no confession is mentioned, nor any judicial/administrative outcome). Patient 2 a 6 week old with a complaint of apnea, alert in ER, dilated funduscopy revealed bilateral RH not otherwise described. CT revealed bilateral SDH, skull surv rev healing rib fxx, a distal femur fx, a corner fx, forearm healing fxx, etc. Father confessed to shaking. This was the only patient to have RH in this study. Patient 3 a 6 month old with a history of multiple ALTE's, ER physician observed his mother trying to smother him. MSBP diagnosed. Eye exam not mentioned.

Fifteen (13%) of the 128 subjects had a family history of SIDS. The authors did not run a P-value on this, but it looks to me to be highly significant. The authors do not comment on it. This incidental finding needs to be looked into further re SIDS.

Infantile subdural hematomas due to traffic accidents. Vinchon M et al. (Lille) (see under "Time of Injury -- Head"). Pediatric Neurosurgery 2002 Nov; 37(5): 245-253. The main finding of this retrospective CT study of 18 infantile acute SDHs was that most of them were mixed-density on the first day. Three cases had RH, "of a type distinct from that found in SBS."

Infantile subdural hematomas due to traffic accidents. Vinchon M, Noizet O, Defoort-Dhellemmes S, Soto-Ares G, Dhellemmes P. Pediatric Neurosurg 2002; 245-253 (Lille). Had 18 infants with CT scans and 16 had ophth. Three had RH, all flame hemorrhages limited to the posterior pole.

Bilateral retinal hemorrhages in an 18-year-old woman. Abdul-Rahim AS, Brown SM, Shami MJ. Surv Ophth 2002 Nov-Dec; 47(6): 590-593. Bilateral flame hems and a foveal subhyaloid hem due to ALL.

Extension of retinal hemorrhage into the vitreous of a shaken baby through a break in the internal limiting membrane. Arch ophth 2003 Jan; 121(1): 91-96. Commented on in Emerson GG (letter) Arch Ophth 2004 May; 122(5): 792, with author's reply.

Retinal and subdural haemorrhages: Aoki revisited. Gardner HB. Br J Ophth 2003 Jul; 87(7): 919-920. See also his letter commenting on Levin's August 2002 article: Retinal hemorrhages in children. (letter) Gardner HB. Ophthalmology 2003 Sep; 110(9): 1863.

Nonophthalmologist accuracy in diagnosing retinal hemorrhages in the shaken baby syndrome. Morad Y, Kim YM, Mian M, Huyer D, Capra L, Levin AV. J Pediatr 2003; 142: 431-434. Retrospective chart review of 75 SBS patients seen by Dr Levin's eye service at the Hospital for Sick Children, age range 2-48 months, average age 10 months. SBS being defined as two or more of the following:

characteristic neuroradiologic abnormalities

skeletal injury
 RH
 history of shaking
 no adequate history to explain the injuries

Of their 75 SBS cases, 61 cases or 85% had RH confirmed by ophthalmology. As far as the work of the non-ophthalmologists, the authors found that out of the 61 cases with confirmed RH, the nonophthalmologists

were unable to examine the retina	14 cases
failed to document any attempt to examine the retina	26 cases
correctly diagnosed the retina	28 cases
got false-negative results	4 cases
got false-positive results	0 cases
reported details about the hemorrhages	0 cases

Intraretinal hemorrhages and chornic subdural effusions. Glutaric aciduria type I can be mistaken for shaken baby syndrome. Gago LC, Wegner RK, Capone A Jr., Williams GA. Retina 2003 Oct; 23(5): 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.

Lens-sparing vitreous surgery for infantile amblyogenic vitreous hemorrhage. Capone A. Retina 2003 Dec; 23(6): 792-795. Reports results on eleven eyes with VH due to SBS. Results of vitrectomy were improvement in several patients, but "Visual outcome of SBS may be limited as a consequence of structural damage to the retina, optic nerve, or posterior visual pathways."

Traumatic brain injury in childhood: intensive care time-series data and outcome. Jones PA, Andrews PJD, Easton VJ, Minns RA. Br J Neuros 2003; 17: 29-39. Prospectively followed 54 head-injured children. Normal ICP values were

1 year old	6 mm Hg
8 year old	8 mm Hg
15 year old	15 mm Hg

Cerebral perfusion pressure

1 year old	45 mm Hg
15 year old	58 mm Hg

Pts with very high ICP all did poorly.

Ophthalmologic findings in suspected child abuse victims with subdural hematomas. Pierre-Kahn V, Roche O, Dureau P, Uteza Y, Renier D, Pierre-Kahn A, Dufier JL (Paris). Ophthalmology 2003 Sep; 110(9): 1718-1723. From the abstract: A prospective observational case series designed to determine whether ophthalmology can help distinguish between shaken babies and children with accidental impact head trauma. Consecutive infants hospitalized with subdural hematoma.

group 1	186 children presumed to have been shaken
group 2	38 children with ev of head impact, no history of trauma
group 3	7 children with severe accidental head trauma

Results

group 1	77% had RH
group 2	20% had RH

group 3

0% had RH

The shape, laterality, and size of the hemorrhages did not differ between groups 1 and 2.

82% of the hemorrhages resolved within 4 weeks.

Conclusion: RH were not specific for shaken baby syndrome. But in conjunction with a subdural hematoma, they are strongly suggestive of shaking.

Comment by JKR: This is an example of how the diagnostic entity of SBS has caused confusion in research. These authors placed shaken babies in a separate group from impact babies. Query whether there was adequate factual basis for doing so. Their results become uninterpretable.

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/>
Dr Reece has a brief summary of the RH literature in his contribution entitled, "The differential diagnosis of inflicted childhood neurotrauma." Here is what he says:

Increased ICP. Cites the reports of Muller & Deck, 1974, Khan & Frenkel, 1975, Lambert, Johnson & Hoyt, 1986, Munger et al., 1993, McRae et al, 1994, and Emerson et al., 2001. Says that the problem with this line of argument is that RH are rare in nonabuse cases of increased ICP, and even when present are few and posterior. (citing Levin in Reece & Ludwig, 2001).

Birth hemorrhages. Up to 45% of vaginal deliveries, citing Levin and Emerson et al., 2001.

Purtscher's syndrome/ CPR. CPR does not cause RH. Citing Kanter, 1986, Gilliland & Luckenbach, 1993, and Odom et al., 1997.

Seizures. Citing Sandramouli, 1997 (33 children with sz, no RH) and Tyagi et al., 1998 (32 children with sz, no RH).

Meningitis. Case report by Fraser et al., 1995 of one case of a 12-year-old with meningococcal meningitis who was found to have a large subretinal hemorrhage with vitreous hemorrhage.

Then Dr Levin has a contribution (pp. 127-159, including 26 pages of references), entitled "Ophthalmic manifestations of inflicted childhood neurotrauma." Dr Levin states at the outset that in evaluating the literature, he gives greater weight to those papers in which the RH were fully characterized following an examination by an ophthalmologist. Most of the papers in the 26 pages of references were not. Dr Levin's concise and condensed summary of the literature divides the subject of abusive RH into topics:

Epidemiology: Most of the papers report about 80% overall incidence of RH in SBS (defined to include both impact and non-impact AHT). He points out a reason for the discrepancy in published incidence: the documentation of RH is higher in autopsied fatal cases, where even microscopic RH can be found, and lower in survivors. Traumatic retinoschisis: "Macular retinoschisis with or without paramacular folds (traumatic retinoschisis) has been well documented clinically, at postmortem, and by electroretinography as a distinctive finding that has not been reported due to any other cause in children younger than 5 years." (p. 128) [This is as of 2003. -JKR] Four groups have shown a correlation between the severity of the RH and the severity of the brain injury. Sidedness is not conclusively demonstrated because of small sample sizes. [This is an important point with regard to the mechanism of RH. If vitreoretinal traction is the mechanism, there should not be sidedness. -JKR]

Pathophysiology:

Purtscher's syndrome: There is no correlation between rib fractures and the frequency or severity of RH. But it can occur.

Terson's syndrome. The apparent lack of sidedness is against Terson's. Plus Terson's "seems to be uncommon in children."

Vitreous shaking: "has strong support in theory and autopsy findings." Also the high frequency of hemorrhage at the vitreous base favors this mechanism. [But Terson's is also at the vitreous base. JKR] "The body of literature suggests that it is shaking itself, with resultant shearing injury, that is the primary factor in the generation of RHs seen in SBS." (p. 129) Says that the optic nerve atrophy is primary optic nerve atrophy, [even though the usual cause of visual loss is cortical blindness. -JKR]

Differential diagnosis: The long list of systemic and ocular diseases that can cause RH can be dismissed from this differential because of the lack of supportive features in the history and physical. That leaves some conditions that are worth discussing.

1. Unintentional head trauma. There is overwhelming evidence that unintentional head trauma causes RH in less than 3% of instances. “When RHs do occur, they are almost always confined to the posterior pole, few or moderate in number, and rarely subretinal.” [This knowledge, which now seems commonplace, is entirely owing to the educational efforts of Dr Levin toward our profession, and we owe him a permanent debt of gratitude for making it part of our diagnostic acumen. – JKR] “Short falls associated with significant brain injury and intracranial hemorrhage can rarely result in RH of the type seen in other unintentional injuries.” (p. 130) But [massive RH of the type seen in abuse] and documented by an ophthalmologist has never been reported in unintentional injury.

2. CPR. [As to which, see *Knight’s Forensic Pathology*, 2004, below] RH rarely occur and when they do they are small posterior pole. Only one convincing case in the literature.

3. Increased ICP. There is little evidence in the published pediatric literature. Notes that increased ICP hemorrhages generally are due to papilledema (which occurs in about 10% of SBS cases) or retinal vein occlusion, and are peripapillary.

4. Vaccination. No evidence.

Knowledge gaps:

Epidemiology: The time course of RH -- generation and resolution. A uniform grading schema for type and number. The specificity and sensitivity of various types and numbers re intentional versus unintentional head injury (although the sens and spec of TR is already known to approach 100%).

Pathophysiology: [This paragraph is of extreme importance, both as to its content and its wording. It shows that Dr Levin’s thinking is evolving from the doctrinaire, almost propagandistic positions of a few years ago to a more scientific approach. As a result, I am going to quote it in full. –JKR] *“The role of vitreous traction and tissue shearing must be further explored. The postmortem orbital findings at our center suggest a role for autonomic dysregulation and direct vessel damage that is yet to be explored. We do not understand the compounding effects of anoxia/ hypoxia, anemia, thrombocytopenia, mild coagulopathy, obstruction of retinal venous flow, or possible age-related anatomic variations in the retinal vasculature. The adjunctive role of increased ICP needs further exploration. Although the role of vitamin C deficiency has been suggested exclusively in lay and legal literature, this has not been formally explored. We do not know the minimal forces required to generate RH, or more specifically, the reason why shaking seems to be unique in the generation of severe RH.”*

Differential diagnosis: Gives a short list of some conditions that are of interest because they *have not been systematically excluded* as possible rare causes of RH in infants:

Anoxia
Increased ICP
SIDS
Coagulopathies
Anemia
Hypotension
Hypertension
Mild vitamin K deficiency*
Mild vitamin C deficiency*
Carbon monoxide poisoning
Immunizations
Glutaric aciduria type I
Meningitis
Drowning

* [Why “mild?” Because severe deficiency is a known cause and is easily excluded on clinical grounds in a given abuse case. Mild (subclinical) deficiency is the type that comes up in court. It is not so easily excluded and has not been researched. –JKR]

Knight's Forensic Pathology, Third Edition. Pekka Saukko and Bernard Knight. London: Arnold, 2004. "Petechiae in the eyes and intra-ocular haemorrhages can occur after **CPR**, as well as after violent sneezing or coughing; they are well known to occur during whooping cough." p. 40. "Retinal haemorrhages, classically a sign of raised intracranial pressure and of head injury, have also been described in whooping cough and after CPR." p. 41 [Compare Dr Levin's views as of 2003, just above. –JKR]

Extension of retinal hemorrhage into the vitreous of a shaken baby through a break in the internal limiting membrane. Emerson GG. Arch Opth 2004; 122(5): 792. Author reply 792-293.

Characteristics that distinguish accidental from abusive injury in hospitalized young children with head trauma. Bechtel K, Stoessel K, Leventhal J, Ogle E, Teague B, Lavietes S, Banyas B, Allen K, Dziura J, Duncan C. Pediatrics 2004 Jul; 114(1): 165-168. Prospective clinical study of 82 chil aged under 2 yr admitted to Yale New Haven with head injury. 15 abusive and 72 accidental. all subjects had dilated ophthalmoscopy. Abusive was determined by "historical and physical examination findings and an extensive evaluation of the child's psychosocial status," similar to the classification scheme used by Duhaime et al., Very young children (1992) and Reece & Sege (2000). In other words, they used the history and investigation, not the head injury itself, to classify. The authors say this method "could be one limitation of our study." Be that as it may, found:

	Abusive	Accidental
RH	60%	10%
Bilateral RH	40%	1%
Preretinal hemorrhages	30%	0%
Premacular RH	20%	0%
Extending to the periphery	27%	0%
Sz	53%	6%
AMS on presentation	53%	1%
Scalp hematoma	7%	49%

Conclusion: "Such characteristics [as the above] may be useful to distinguish accidental from abusive had trauma in children <24 months of age." This article is commented on in a letter to the editor by Lueder, below (2005), asking for more detail on the accidental cases. And so the authors provided the following additional detail on the 7 accidental cases:

1 month-old witnessed 3-foot fall out of father's arms as he was lying in bed. right-sided skull fx and EDH, one right-sided RH

4 month-old fell 3 feet out of sibling's arms, left skull fx, intraretinal hems in the center of the retina OS not extending to the periphery

4 month-old witnessed 4-foot fall out of mother's arms, head hit table, acute R skull fx and intracranial hemorrhage, intraretinal hems around optic disk and arcades

8 month-old witnessed 2-foot fall out of mother's arms as she was lying on a couch, L skull fx and EDH, single intraretinal hem OS

8 month-old witnessed 3-foot fall off a bed, acute R SDH, bilat intraretinal hems, each of which were small, in the center of the retina, and did not extend to the periphery

9 month-old witnessed fall down stairs in a walker, R frontal skull fx, acute R SDH, single intraretinal hem OD

10 month-old head run over by a car in the driveway, witnessed, intracranial air with orbital fx, intraretinal hems in the center of the retina OS not extending to the periphery

Shaken baby syndrome without intracranial hemorrhage on initial computed tomography. Morad Y, Avni I, Capra L, Case ME, Feldman K, Kods SR, Esernio-Jenssen D, Lukefahr JL, Levin AV. Journal of AAPOS 2004 Dec; 8(6): 521-527. (American Academy for Pediatric Ophthalmology and Strabismus). Working off of the listserv, they found eight (8) cases of SBS from around the world in which the initial CT scan failed to demonstrate any intracranial hemorrhage. Age range from 2 to 27 months. Added to eight previously reported cases of SBS with no intracranial hemorrhage, that makes 16. For a detailed summary of this article, see below under "Shaken." All eight cases had extensive bilateral RH, one with a perimacular fold, one with schisis cavities. They give diagnostic criteria for SBS which do NOT require intracranial hemorrhage:

the presence of at least two of the following criteria:

- (1) altered state of consciousness with or without seizure and an abnormal CT scan showing characteristic findings of SBS [other than hemorrhage]
- (2) typical skeletal injury
- (3) retinal hemorrhage with or without macular schisis
- (4) history of child abuse that included shaking with or without blunt head trauma or an inadequate history to explain the observed injuries

A new cause for retinal haemorrhage and disc oedema in child abuse. Raman S, Doran RM. Eye 2004 Jan; 18(1): 75-77.

Retinal hemorrhages in an 8-year-old child: an uncommon presentation of abusive injury. Mierisch RF, Frasier LD, Braddock SR, Giangiacomo J, Berkenbosch JW. Pediatric Emergency Care 2004 Feb; 20(2): 118-120. Case report of severe abusive injury, including shaking, causing fatal intracranial hemorrhage and RH in an eight year-old boy who died of intractable intracranial hypertension. "Police investigation confirmed that the injuries were caused by severe abusive injury, including shaking." Conclusion: The diagnosis of SBS is not limited to babies.

Patterns of presentation of the shaken baby syndrome: subdural and retinal haemorrhages are non necessarily signs of abuse. (letter) LeFanu J, Edwards-Brown R. BMJ 2004 Mar 27; 328(7442): 767.

Images in clinical medicine: circumpapillary retinal ridge in the shaken baby syndrome. Hylton SC, Goldberg MF. (Johns Hopkins) NE J Med 2004 Jul; 351(2): 170.

A new cause for retinal haemorrhage and disc oedema in child abuse (letter). Raman S, Doran RML. Eye 2004; 18: 75-77. (Leeds). From Dr Levin's review in the *Quarterly* for Autumn 2004, an abused 3 year old with FTT, multiple bruises and scratches, fractures, a thin SDH with hydrocephalus, and multiple flame hemorrhages around both optic nerves, with papilledema in one eye. BP 150/110. The authors suggest that the flame hemorrhages were due to hypertension, which was due to anxiety. Dr Levin states that it is clear these hems were due to inflicted neurotrauma, not hypertension.

Retinal haemorrhages as proof of abusive head injury. J Pediatr 2004 Jun; 144(6): 719-722.

Hemorrhages after minor trauma. Ophth 2004 Jul; 111(7): 1428-1431.

Update from the ophthalmology child abuse working party: Royal College of Ophthalmologists. Adams G, Ainsworth J, Butler L et al. Eye 2004; 18: 795-798. According to Dr Levin's review in the Winter 05 *Quarterly*, this consensus-development statement poses questions and answers:

1. Can normal handling cause RH? No.

2. Do cervical injuries cause RH? No.
3. Does hypoxia cause the clinical picture of SBS? No.
4. Can short-distance falls cause RH? Rarely.
5. Does Terson's syndrome happen in children? Rarely, and only around the optic nerve.

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. Discusses RH. 70 – 90% of cases. Unilateral in 14 – 20%. RH can be categorized as preretinal, intraretinal, or subretinal. Subretinal hems are distinguished by seeing the retinal vessels coursing over them. Preretinal hems are distinguished by seeing that they cover up the retinal vessels. Preretinal hems are also called subhyaloid hems. Intraretinal hems are described by appearance as flame, dot, or blot. As to the mechanism of RH, says that there are several theories. "The most likely explanations are the orbital and vitreous shaking theories."

a. The orbital shaking theory proposes that the eye shakes forward and backward within the orbit, in which it is only loosely fixed, and this stretches the optic nerve, causing ONSH. This ONSH in turn causes RH. "It is unclear how this intradural hemorrhage translates to retinal hemorrhage, but it may be the result of autonomic dysregulation or central vessel damage impairing blood return from the eyeball." (citing Levin in *Recent Advances No. 18*, 2000).

- b. The vitreous traction theory, which Dr Starling describes as more likely.

Discusses the differential diagnosis of RH. Birth hemorrhages affect up to 59% of normal newborns, with increased incidences in prolonged labor and difficult delivery. Even the deepest birth hemorrhages disappear in 14 to 21 days. The further differential diagnosis of RH includes sepsis, leukemia, meningococcal meningitis, coagulopathy, and vasculitis. But those hems are minor and generally limited to the posterior retina. Seizures do not cause RH. (citing Sandramouli et al., 1997 and Tyagi et al., 1998). Accidental trauma is in the differential because 2% of serious accidental head injury produces RH. Citing DiScala et al. (2000) who found RH in 0.6% of accident victims and 22.8% of abuse. As to CPR, there are a few case reports of CPR causing small posterior RH. As to Terson's syndrome, says "Terson syndrome in children is very rare; when seen it usually is associated with shaking injuries." (citing Levin, 2000). As to Purtscher's syndrome, it is retinal exudates or white patches after severe compressive chest trauma; it is also very rare in children. Cites the case report of McEniery et al. (1991) on a child with inflicted chest compression who had no RH.

Junk science and glass houses. Patrick Lantz (letter) *Pediatrics* 2004; 114: 330. Excoriating an editor of *Pediatrics* (Dr Lucey) for having written a letter characterizing as "junk science" the author's art in *BMJ* reporting perimacular folds in a natural case. (See below under "Traumatic retinoschisis.") Refers to the AAP's official statements (1993, 2001) that "retinal and vitreous hemorrhages and nonhemorrhagic changes, including retinal folds and traumatic retinoschisis, are characteristic of shaken baby syndrome." Says that the total published basis for this dogma consists of "a nonsystematic review article, a noncomparative case series, and a book chapter (Greenwald, 1986, Levin, 1990, and Levin, 2000). Citations in those articles and book chapter...consist solely of noncomparative, observational reports." Says, "The vested dogma that vitreoretinal traction causes traumatic retinoschisis and perimacular retinal folds during a presumed shaking episode is a faith-based assumption, not a scientific fact."

In reply, Dr Block of the Academy says Dr Lantz fails to appreciate the difference between the words "characteristic" and "diagnostic." In a somewhat convoluted reply, he seems to imply that this wording was reached advisedly during discussions by the Committee. Appears to concede that the lesions are not diagnostic. Says, "There is no doubt, based on accumulated scientific experience with hundreds of examples, that RH and other ocular manif are characteristic findings, along with other biologic and sociologic markers, in cases of AHT. Although there may be some physicians who falsely assume all RH or retinal folds are indicative of abuse, most have evolved medical opinions based on an accumulation of

reports, including the single case reported by Dr Lantz and his colleagues.” Does not, however, defend the editor’s use of the term “junk science,” and appears to concede by silence that it was inappropriate.

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. Comes up with 41 cases exclusive of the 13 cases of Hadley, Sonntag, Rekate (1989); in Hadley’s cases it is not clear where the shaking information came from, whether it was from the history or just inferred by the clinicians, so those cases are treated separately. Of the 41 historically shaken cases, there were 11 pure shakings, 12 shaken-plus-impact, and 18 in which impact could not be determined from the information provided. Of the 41 admittedly shaken cases without Hadley’s material, had average age 7 months, 33 had some comment on the presence or absence of RH. Of these 33, all 33 (100%) had RH. Ten (10) had some comment as to vitreous hemorrhage, and all ten of these had VH. As to retinal folds, only two (2) cases had them, and these were cases of Massicotte (1992) and Kivlin (1999). Greenwald’s macular cysts or domelike lesions with their residual “circular cicatrix” are not taken to represent retinal folds for purposes of this article. Retinal detachment and papilledema are also categorized, but were very rarely recorded.

Retinal hemorrhage and pediatric brain injury: etiology and review of the literature. Aryan HE, Ghosheh FR, Jandial R, Levy ML. J Clin Neurosci 2005; 12(6): 624-631. From the abstract: “The presence of RH is neither necessary nor sufficient for the diagnosis of child abuse. Additionally, RH are also associated with an ever-expanding list of conditions, each of which carries important implications for patients and their families. To correctly interpret a patient’s retinal hemorrhages, the physician requires a broad knowledge base, including child abuse, the differential dx of RH, and the types of RH...”

RH in accidental and nonaccidental injury (letter). Lueder GT. Pediatrics 2005 Jan; 115(1): 192. Comments on Kirsten Bechtel’s 2004 art (above), asking for more detailed data on the RH observed in 7 pts with accidental injury. 3 of them had a single hem. What about the other 4? Says this could be significant given the heated medicolegal debate that has erupted in the field. Asks: what were the mechanisms of injury in those 4 pts, how many RH did they have, and where? Cites Lantz, Junk science (2004) for the heated debate. I have incorporated into the summary of Bechtel’s art (above) the additional data which the authors provide in response to Dr Lueder’s letter.

Accidental and nonaccidental head injuries in infants: a prospective study. Vinchon M, Defoort-Dhellemmes S, Desuremont M, Dhellemmes P. (Lille) J Neurol (Pediatrics 4) 2005; 102 (4 Suppl): 380-384. From Larry Ricci’s review in the Winter 2006 *Quarterly*. Had 56 cases thought to be abuse and 73 thought to be accidental. Here are some of the findings:

	RH	No RH
Abuse	42	14
Acc	5	68

So the specificity of any RH for abuse was 93%. The PVP was 89% and the PVN was 83%. But then they graded the RH as to mild, moderate, or severe. When that was done, they got

	Mild RH	Moderate RH	Severe RH
Abuse	5	10	27
Acc	5	0	0

So for moderate or severe RH the SENS for abuse was 66% and SPEC was 100%.

The authors also studied the epidemiology and risk factors. They found a highly significant association between abuse and a history of perinatal illness history, substance abuse, psychiatric disorder, violence, jail, child removal. The authors speculated that perinatal illness could have interfered with parental bonding.

The abused child and adolescent. Platt MS, Spitz DJ, Spitz WU. In: Spitz WU, ed., *Spitz and Fisher's Medicolegal Investigation of Death: Guidelines for the Application of Pathology to Crime Investigation*, 4th edition. Springfield, Ill., Charles C. Thomas, 2006. Their pathogenesis is not precisely understood.

Mechanisms that have been proposed:

(1) increased pressure in the central retinal vein due to ?; (2) vitreous traction; (3) Purtscher-like syndrome from chest compression (citing Goetting & Sowa, 1990); (4) Increased ICP. Says that RH caused by increased ICP are rare. He goes with acceleration-deceleration when it comes to *retinal detachment* (see below). RH are common in AHT but may occasionally be seen in car accidents, accidental falls. Gives an abbreviated differential dx for RH. Discusses birth hemorrhages, with Emerson, 2001 finding 35% of healthy newborns had RH following NSVD; 80% gone by two weeks, 100% gone by one month. Mentions other types of ocular injury associated with AHT:

- VH
- ONSH
- retinal detachment*
- retinoschisis*
- optic nerve infarction
- cataract
- dislocated lens
- optic nerve atrophy

Retinoschisis and retinal detachment can only be dxd via indirect ophthalmoscopy. “The artifact created by removing and bisecting the globes make postmortem diagnosis of these entities virtually impossible.” (p. 387). “Whenever a child is suspected of being abused, the ocular fluid should not be drawn from the eye. .. Aspirating the ocular fluid prior to examination of the retina may cause artifacts that are difficult to interpret.” (p. 387, no citation). [I should note that comments on NAME-L (2006) from around the country including the chief of ophthalmic pathology at the AFIP disagree with the foregoing, and state that aside from the removal of vitreous hemorrhage, alteration of RH by aspiration of vitreous humor is unlikely or does not occur. Some pathologists advocate reinflating the globe with formalin through the same needle after aspiration. –JKR]

The retina in forensic medicine: applications of ophthalmic endoscopy: the first 100 cases. Davis NL, Wetli CV, Shakin JL. Am J Forens Med P 2006 Mar; 27: 1-10. Used an ophthalmic endoscope to get around the problem of postmortem corneal clouding. Found RH or retinal petechiae in 15 cases, of which 10 had been subjected to either CPR or ventilatory support (67%). “This clearly exceeds the 48% of our total pop which were exposed to these forms of life support, thereby indicating a 19% greater likelihood of retinal petechiae or hemorrhagic activity in cases of resuscitative effort.” As to ICP, Terson’s, head trauma, etc., found that 9 of the 15 hemorrhagic retina cases did have some form of head trauma, neck compression, chest compression, or rapid rise in ICP. But 6 cases did not have these events. “Therefore, retinal petechiae do not necessarily occur in cases of head trauma, Terson syndrome, and neck/chest compression, although they are more prevalent in this population. Then gives a lengthy discussion of the following topics:

- Shaken baby syndrome
- Determination of postmortem interval
- Exsanguination
- Postmortem position
- Carbon monoxide
- Terson’s syndrome
- Natural disease
- Ideas for the future

As to SBS, reports one case in their series: a two month-old who died of cardiac disease, in whom they did this endoscopy pre-autopsy to allay concerns about possible SBS, not clear why.

As to postmortem interval, because the retina tends to lose adhesiveness and become folded postmortem. The authors tried to correlate the degree of folding with PMI, with poor results.

As to exsanguination, the authors looked for pale vessels. They only had one case, but it did have extremely pale to undetectable vessels.

As to postmortem physical position, the authors gained the “overall impression” that there is such a thing as retinal livor mortis.

As to carbon monoxide poisoning, they found bright red coloration.

As to Terson’s syndrome, they present one case of a fatal ruptured berry aneurysm that showed flame hemorrhages radiating from the optic disc.

As to natural disease, the authors suggest that AV nicking and diabetic retinopathy might be seen.

As to ideas for the future, just keep doing it.

Ocular manifestations of crush head injury in children. Gnanaraj L, Gilliland MGF, Yahya RR et al. Eye, November 2005. According to Dr Boos’s review in the Spring 2006 edition of the *Quarterly*, the Toronto group reports two groups of accidental-injury children; 16 toddlers who had televisions fall on their heads (no deaths), and 9 who suffered complex skull fractures in MVA’s (all died). Of the TV’s, 1/11 who had oph exams was found to have RH (9%), which was preretinal and intraretinal (blot) hemorrhages extending to the equator. Of the MVA’s 4/9 had RH (44%), three confined to the posterior pole and one extending to the ora serrata. No retinoschisis or folds. The authors caution meticulous investigation of crush injury cases.

Dr Boos comments that this article seems to be a commentary on Pat Lantz’s case report of TR from a TV case. The present authors consider his case to be atypical, but admit that they themselves might have encountered the same findings if their sample size had been larger. “Clearly, a significant minority of head crush injuries are likely to include RH.” Dr Boos in his comment goes on to consider the implications for the biomechanics and pathogenesis of RH. The authors correctly make the point that all of these injuries initially involved impact of the moving head -- dynamic loading. But is that the biomechanically significant event, or is rapid ICP? he asks. Some pts had orbital fractures, implying direct dynamic loading of the eyeball, but not all. Keenan’s study (Pediatrics 2004) found 25% incidence of RH in accidental injury, the highest incidence reported, but many of his pts had direct ocular trauma.

Retinal hemorrhage and pediatric brain injury: etiology and review of the literature. Aryan HE, Ghosheh FR, Jandial R, Levy ML. J Clin Neuroscience 2005; 12: 624-631. According to Dr Levin’s review in the *Quarterly* for Summer 2006, this article deals only in part with child abuse, and discusses many adult diseases. The article apparently gives an exhaustive differential DX of RH, most of which is irrelevant to child abuse. Dr Levin makes in his review the useful observation that, “I also must challenge the authors’ statement that blood underneath the internal limiting membrane (ILM) is ‘preretinal.’ I believe most authors would consider this a form of retinoschisis, since preretinal blood would be more correctly termed as blood *in front of* the ILM.” [See Dr Levin’s definition of retinoschisis under “Traumatic Retinoschisis,” below.]

Protective ocular mechanisms in woodpeckers. Wygnanski-Jaffe P, Murphy CJ, Smith C et al. Eye 2005;doi:1038/sj.eye.6702163:1-7. (Toronto) According to Randy Alexander’s review in the Autumn 2006 issue of the *Quarterly*, found, aside from the straight-line motion of the woodp’s head, found four special things about the woodp orbit and eyeball: (1) the globe is tightly packed in the orbit. (2) woodp’s close their eyes at each peck, restraining the globe. (3) their ocular structures are rigid. (4) they do not have vitreoretinal coupling. Randy also notes that the mass of the woodp brain is very small, and $f=ma$, so the *force* on the brain is very small.

Histology

A method for immediate gross sectioning of enucleated globes. Smith ME. Am J Ophth 1974; 77: 413-414. Reference on ocular sectioning technique courtesy of Dr Scott Denton. See also J Clin Path, 2001; Arch Path, 2001.

Hemorrhagic retinopathy in infancy: a clinicopathologic report. Richard R. Ober, MD (USC) J Pediatr Ophth Strab 1980 Jan/Feb; 17(1): 17-20. (have) This is a case report of a case that was actually signed out as SIDS by this office (!), which may account for the four years' delay in publishing the case. Case of a ten month old female admitted to LAC-USC in 1976 with a history of being found lethargic by the mother when she came home from work after leaving the baby in the father's care for several hours. There was a small chin bruise which the mother attributed to a fall from a walker. Hospital found a torn frenulum, diminished pupillary light reflex, on the right, and retinal detachment on the right, with diffuse retinal hemorrhages and vitreous hemorrhage, the left eye showing extensive intraretinal and vitreous hemorrhages as well. WBC 11,600 Hgb 11.6, x-rays normal. On the day after admission the neurological status deteriorated, seizures developed fol by resp depr. Subdural taps were negative. Cerebral edema was

SUSPECTED WHIPLASH SHAKEN INFANT SYNDROME

195

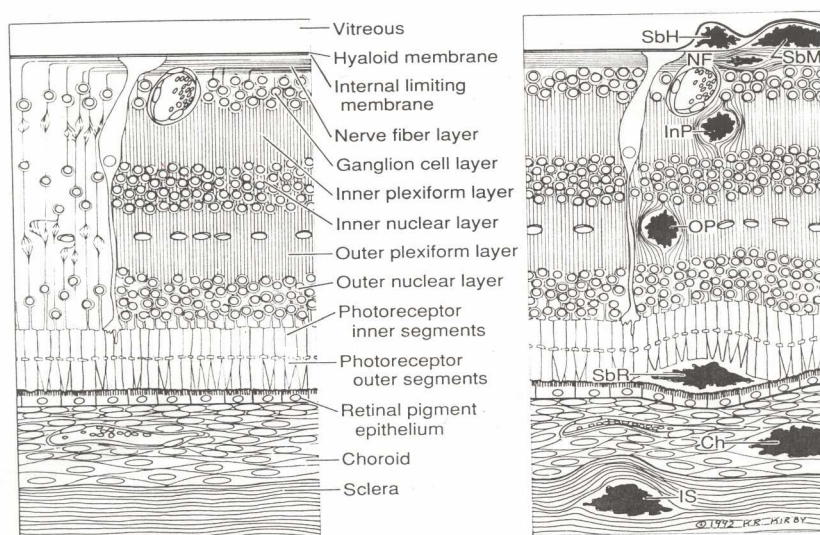


FIG. 1. Schematic anatomy of the retina (left) with associated schematic representation of the location of hemorrhages encountered in the shaken infant syndrome. SbH, subhyaloid hemorrhage; SbM, submembranous hemorrhage; NF, nerve fiber layer hemorrhage; InP, inner plexiform layer hemorrhage; OP, outer plexiform layer hemorrhage; SbR, subretinal hemorrhage; Ch, choroidal hemorrhage; and IS, intrasceral hemorrhage. In actuality, hemorrhages were likely to extend from one layer into another, with involvement of nuclear layers as well. Intrasceral hemorrhages were encountered only in the peripapillary area.

present. Died after five days on a ventilator. Autopsy: resolving lac of the frenulum, resolving contusions of the left mandible and the occipital, No skull fx, 10 cc of blood present in the right parietal subdural space, severe hypoxic enceph. Eyes: Right eye: total retinal detachment (whole-mount photomicrograph given) overlying masses of subretinal hemorrhage. Left eye: numerous preretinal and intraretinal hems and a small scleral hem next to the optic n. Micro on the eyes: Right eye: Numerous RBC's in the vitreous humor. Hemorrhage and fibrinous exudate beneath the retina, retinal edema. Left eye: multilayered RH (whole-mount photomicrograph given). No comment on any ONSH except the statement that on the left eye "The choroid, sclera, and optic nerve posterior to the lamina cribrosa were unremarkable." The author reviews the literature on retinal hemorrhage: Friendly in 1971 reported RH in 46% of 28 children with unexplained fractures and 35% of 26 pts with unexplained soft-tissue injuries. Jensen in 1971 reported ocular trauma in 40% of 48 battered children. "In both series, the most common ocular finding was RH that was often assoc w intracranial bleeding." No mention of ONSH. Friendly also had reported retinal detachment with apparent direct eye trauma in his patients. Weidenthal and Levin (DB Levin) in 1976 reported retinal detachment and giant retinal tears from apparent direct eye trauma in an 8 week old. Mushin in 1971 reported two cases of retinal detachment in infant head trauma. Harcourt and Hopkins in 1971 reported one case with vitreous hemorrhage and probable tractional retinal detachment with severe head trauma. Kiffney in 1964 reported traumatic retinal detachment in a 7 month old with repeated head trauma. The author goes on to discuss whether traumatic retinal detachment can result from indirect trauma

rather than solely from direct eye trauma. "Some reports have implicated an indirect mechanism of injury to explain retinal hemorrhage seen in the battered child. (citing Gilkes & Mann, 1967 and Caffey, 1974). Consisting of "a sudden, extreme rise in both intracranial and intraocular venous pressure instead of direct cranial trauma," suggesting a similarity to Purtscher's retinopathy as also suggested by Tomasi & Rosman, 1975 in two battered infants with chest compression trauma. Refers to Mushin's case of a possibly strangled baby with RH, 1971. Brings in Caffey's hypothesis of shaking injury, and refers to a study of human volunteers on a rocket sled deceleration model of supersonic ejection which caused RH (Lyle et al., 1957; Stapp, 1971) and similar observations in jet pilots who bailed out from supersonic aircraft. Suggests that in his present case, acceleration-deceleration forces could have caused the retinal detachment and RH.

Terson's syndrome: clinicopathologic correlations. Weingeist TA, Goldman EJ, Folk JC, Packer AJ, Ossoinig KC. Ophth 1986; 93: 1435-1442. This article presents photomicrographs and electron micrographs from two cases of Terson's syndrome of adults. One case (the authors' case 8) shows a clearly subhyaloid dome hemorrhage. The other case (the authors. Case 4) shows findings consistent with a sub-internal limiting membrane hemorrhage. Levin relies on this study for his assertion that the histopathology of TS and TR are "quite different." (2000a, p. 165.) The only other histopathologic studies of TS are by Keithahn (1993) and Friedman & Margo (1997.) These only show the epiretinal membrane; the findings are those of subinternal limiting membrane hemorrhage, and these authors so state. See also the work of Kuhn's group, under TS.

Autopsy findings in the eyes of fourteen fatally abused children. Rao N, Smith RE, Choi JH et al. FSI 1988; 39: 283-299.

Ocular and associated systemic findings in suspected child abuse. Elner SG, Elner VM, Arnall M, Albert DM. Arch Ophth 1990 Aug; 108(8): 1094-1101. (have) See under "Other." Includes **histology** of schisis cavities.

RH in an infant after CPR. Weedn VW, Mansour AM, Nichols MM. Am J Forens Med Path 1990 Jan; 11(1): 79-82.

Age-related differences in the human vitreoretinal interface. Sebag J. Arch Ophth 1991; 109: 966-971. This is not really histology but a clinicopathologic study relating gross dissection with attempts to dissect the vitreous off the posterior retina being difficult due to adherence in pts under 20 y o.

Ocular findings at autopsy of child abuse victims. Riffenburgh RS, Sathyav, L. Ophth 1991 Oct; 98(10): 1519-1524. (have) 55 autopsy cases of child abuse, 43 cases of possible abuse, 92 cases of nonabuse deaths. Total 9 cases had RH without ONSH. 4 cases had ONSH without RH. They feel that this casts doubt on the idea that RH are caused by ONSH. Fig. 6 shows "a dome-shaped hemorrhagic lesion," "which elevates the internal limiting membrane," with what looks to me like a retinal fold next to it. "All of our cases were studied for 'dome-shaped lesions' in the macula. (after Greenwald --JKR) Several typical lesions were found, but only one was located in the macula. These lesions are caused by hemorrhage that elevates the internal limiting membrane; this in turn allows the blood to pool beneath the elevated membrane (Fig. 6.) All stages of dome development were found, from small puddles of blood beneath the internal limiting m to those where the membrane had broken and the blood had accumulated under the vitreous face, to those in which the blood was infiltrating into the vitreous. It is our conclusion that these dome-shaped lesions do not represent a specific entity, but, rather, reflect stages in the evolution of hemorrhage within the internal layers of the retina. If the hemorrhaging is profuse and prolonged, the accumulation of blood will elevate the internal l m, gradually leading to the dome-shaped appearance...." [The authors do not seem to realize that what they are saying contradicts the whole theory of traumatic retinoschisis proposed by Greenwald et al. --JKR.]. A parallel article on the same material appears in JFS, next below.

The eyes of child abuse victims: autopsy findings. Riffenburgh RS, Sathyav L. JFS 1991; 36: 741-747.

Vitreoretinal traction and perimacular folds in the eyes of deliberately traumatized children. Massicotte SJ, Folberg R, Torczynski E, Gilliland MGF, Luckenbach MW. Ophth 1991; 98: 1124-1127. (have) Eye

pathology in three cases of AHT with acute perimacular folds. The authors refer to the previous work of Gaynon et al. (1988) proposing vitreoretinal traction caused by shaking alone as a cause of perimacular folds, the work of Elner et al. (1990) saying that retinoschisis is probably due to direct blunt head trauma rather than shaking. The authors state that their purpose is to test Elner's hypothesis that impact rather than shaking is the cause of traumatic retinoschisis. Three babies dying with acute AHT. Case 1 a 7 month old with SDH/SAH, +hern, no skull fx, no subgaleal hemorrhage. +ONSH. Gross eye photos show a large posterior retinal fold that could be a perimacular fold. PAS shows submembranous RH collection with separation of the ILM and ILM remaining attached to the apex of the retinal fold, described by the authors as a hemorrhagic schisis cavity. Case 2 a 9 month old hx shaken and thrown on the floor by BBS. Multiple occipital fxx. Interh SDH. +ONSH. Has perimacular folds. Section of the fold PAS sh "The ILM has been stripped from the inner surface of the retina. The vitreous remains focally attached to the ILM. The vitreous was partially separated from the ILM, but remained so adherent to this structure at the apex of the fold that the membrane was torn away from the retina. The v was attached to the free end of the ilm over the fold..." Case 3 a 13 mo w subgaleal hem at the occiput and contusions to both ears. No fx. +B SDH. Perimacular folds. Multil RH, subhyaloid h, VH. "The vitreous, partially separated from the retina, remained adherent to the ilm at the apex of the retinal folds..." +ONSH. Discussion: "Our study confirms the role of vitreous traction in formation of perimacular folds, as evidenced by partial vitreous detachment except at the apices of the folds..." But does their study decide anything as between Gaynon's pure-shaking hypothesis and Elner's impact hypothesis? They say yes. "The formation of perimacular folds in both eyes of an infant who was violently shaken, but whose eyes and skull were not directly traumatized [case 1], proves that shaking alone is sufficient to produce these folds." The authors state that if subsequent work confirms that perimacular folds are specific to shaking, it will help prosecutors rule out alleged accidentally skull trauma as the cause of these infants' injuries, which would otherwise remain a possibility if only blunt force trauma and not shaking were demonstrable.

Retinal folds in Terson syndrome. Keithahn MAZ, Bennett SR, Cameron D, Mieler WF. Ophth 1993 Aug; 100(8): 1187-1190. See under "traumatic retinoschisis." Gives EM of the epiretinal membrane spanning the perimacular fold on these two chronic perimacular folds in adults with head trauma and vitreous hemorrhage undergoing PPV and finds that this epiretinal membrane contains basement membrane, nerve fiber layer, and old blood, and is therefore the internal limiting membrane. Therefore, this perimacular fold/ macular cyst is morphologically identical to those of Greenwald, Gaynon, Elner, and Massicotte seen in child abuse. Therefore, those perimacular folds are not specific for child abuse.

Ocular and associated neuropathologic observations in suspected whiplash shaken infant syndrome: a retrospective study of 12 cases. Munger CE, Peiffer RL, Bouldin TW, Kylstra JA, and Thompson RL. Am J Forens Med Path 1993 Sep; 14(3): 193-200. Eye histopathology on 12 infants with fatal SBS. 12/12 had RH, 5/12 had retinal folds, 10 had VH, 3 had subhyaloid. The hems are broken down by layer. Shows histol of a separation of the internal limiting membrane in association with NFL hem, ONSH, gross and microscopic of an acute perimacular fold with overlying separation of the internal limiting membrane and focal subretinal hemorrhage under the fold, macular edema, and small cystic hems within the outer plexiform layer. Only 5 infants had head impact. 10 had SDH, 9 had SAH, 10 had cerebral edema pathologically, 9 had ONSH. Separation of the internal limiting membrane by blood was observed in all that had RH. "Our observations fail to define further the pathogenesis of rh in the sbs, which hv bn post to res from ...thorax compr (citing Tomasi, 1975), central retinal v occl by disc edema (citing Vanderlinden, 1974), vitreoretinal traction (citing Toosi, 1987), increased ICP also called Terson's syndrome (citing Weingeist, 1986; Toosi, 1987), or direct head [eye?] trauma (citing Harcourt & Hopkins, 1971; Weingeist, 1986.)" But says "The parallel incidence of intracranial hemorrhages and cerebral edema with RH ... implicates an increase in ICP as a poss pathog mech of the RH."

Ocular manifestations of child abuse. Andrews A. Penn Med 1996; 99S: 71-75. ?

Morphometrical analysis of retinal hemorrhages in the shaken baby syndrome. Betz P, Puschel K, Miltner E, Lignitz E, and Eisenmenger W. FSI 1996; 78: 71-80. See under "Other." Gives two photomicrographs of full-thickness RH

Bilateral subinternal limiting membrane hemorrhage with Terson syndrome. Friedman SM, Margo CE. Am J Ophth 1997 Dec; 124(6): 850-851. Reports bilateral dome-shaped posterior-pole subhyaloid hems in a pt with TS, examined histologically

Ocular pathology of child abuse] (GER). Rohrbach JM, Benz D, Friedrichs W, Thiel HJ, Wehner HD. (Tubingen). Klin Monatsbl Augenheilkd 1997 Mar; 210(3): 133-138. Case report of the eye histology on a 2 year old girl with severe intracranial hemorrhage due to abuse who survived two days. Preretinal, choroidal, and scleral hemorrhages as well as ONSH. One eye had a circular perimacular fold and hemorrhagic retinoschisis. A craterlike appearance of the central retina, hemorrhagic retinoschisis, and scleral hemorrhage in the Circle of Zinn – this combination suggests child abuse almost with certainty.

Terson syndrome: results of vitrectomy and the significance of vitreous hemorrhage in patients with subarachnoid hemorrhage. Kuhn F, Morris R, Witherspoon CDS, Mester V. Ophth 1998 Mar; 105(3): 472-477. ?

The spectrum of postmortem ocular findings in victims of shaken baby syndrome. Marshall DH, Brownstein S, Dorey MW., Addison DJ, Carpenter B. (Ottawa) Can J Ophth 2001 Dec; 36(7): 377-383. Retrospective review of the autopsy material on six patients aged 1 to 34 months from 1971 to 1995. From the abstract: “.. ranged from a focal globular hem at the posterior pole to extensive intraocular hem involving the entire retina with perimacular folds.” All had ONSH. All had intracranial hemorrhage, 2 had skull fx, 3 had rib fx, 4 had high spinal cord hemorrhage.

The spectrum of postmortem ocular findings in victims of shaken baby syndrome. (letter) Brownstein S, Dorey MW. Can J Ophth 2002 Feb; 37(1): 4. Discussing Can J Ophth 2001 Dec; 36(7): 377-383.

[Article on technique for sectioning the eyes.] J Clin Path 2001 Jun; 54(6): 417-427. Reference on ocular sectioning technique courtesy of Dr Scott Denton. See Am J Ophth, 197; Arch Path, 2001.

Unexpected findings arising from psotmortem ocular examination. Butnor KJ, Proia AD. Arch Path Lab Med 2001 Sep; 125(9): 1193-1196. Reference on ocular sectioning technique courtesy of Dr Scott Denton.

Retinal haemorrhages in infant head injury. Clark BJ, Adams GG, Luthert PJ. Brain 2002 Mar; 125(Pt 3): 677-678. See also Neuropathology of inflicted head injury in children. Parulekar MV, Elston JS. Brain 2002 Mar; 125(Pt 3): 676-677.

Why do histology on retinal haemorrhages in suspected non-accidental injury? Gilliland MG, Luthert P. Histopathology 2003 Dec; 43(6): 592-602. Gives a short review of the published histologic studies. Briefly summarizes her grading system for histology of RH

- | | |
|----|--|
| 1+ | visible only at high power |
| 2+ | visible at low power |
| 3+ | slightly distorting the retinal architecture |
| 4+ | markedly distorting the retinal architecture |

Says that contributing to the diagnosis of the individual case as accidental or non-accidental requires that the hemorrhages be documented as to size, depth and topographic location. This is also required for comparison to the published literature.

Says that the reason to do histology on retinal hemorrhages is to identify the size and location of hemorrhages in individual cases in order to properly compare them with other cases and the literature

Says that histology can help document the distinction described by Levin, 2000 (below under “Traumatic Retinoschisis”) between a schisis cavity and a subhyaloid hemorrhage -- remarkably similar in appearance particularly when the roof of the schisis cavity happens to be the internal limiting membrane.

Says that “Review of the literature demonstrates that differences exist between histopathological appearances of the retinas of non-accidentally injured children and those with non-abusively sustained retinal haemorrhages. Such review demonstrates the need for detailed histopathological examination of the retina to identify the size and location of haemorrhages within the retina of individual cases to properly compare them with other cases and the literature.”

Says that iron stains can show evidence of previous injury, but since iron can be present after as little as two days survival, any longer survival than that renders iron nondiagnostic as evidence of previous injury.

Goes on to include a monograph by P. Luthert of London, as to which see next below.

Why do histology on retinal haemorrhages in suspected non-accidental injury? Luthert P. *Histopathology* 2003 Dec; 43(6): 595-602. See also Luthert’s previous work with Clark et al., *Brain* 2002, above. Here he discusses the following topics:

- (1) present areas of ignorance
- (2) the question of possible mechanisms (can we determine the mechanism from looking at the hemorrhages?)
- (3) the question of determining the amount of force from the extent of hemorrhage
- (4) the question of timing the injury from the hemorrhages
- (5) retinal detachment
- (6) perimacular folds
- (7) retinoschisis

(1) Present areas of ignorance: “Our knowledge base is seriously incomplete.” “[W]e do not really know how retinal haemorrhages form in cases of alleged non-accidental injury.” The injuries are unwitnessed, and both confessions and denials are unreliable, and this affects the validity of judicial convictions; therefore, judicial outcomes cannot be used as an endpoint for eye research. Use of judicial outcomes creates the risk of circular argument: judicial conviction will determine which cases we consider abuse for research purposes; yet the judicial convictions were brought about by the fact that we diagnosed them as abuse. Also, pathologists see only the most serious and suspicious injuries, and there are no control retinas from other situations.

(2) Can the hemorrhages tell us the mechanism of the injury? This is most contentious when there is no injury elsewhere, and there is a swollen hypoxic brain with thin-film subdural hemorrhages and widespread retinal hemorrhages. Discusses the vitreoretinal traction theory. Apart from experimentally shaking infant primates with one eye having been vitrectomized, “I can see no way of proving this [vitreoretinal traction] hypothesis.” Says that the most direct evidence for vitreoretinal traction comes from cases of RH associated with posterior vitreous detachment, described by Cibis et al. in 1975, or with operative cataract extraction, described by Mets et al in 1986. “It is appropriate to maintain an open mind.” Goes on to discuss the increased ICP hypothesis as “an alternative, plausible, and not mutually exclusive explanation of [RH].” Citing Gilkes & Mann, 1967, and Kaur, Taylor, 1992. “[I]t is well recognized that massive intracranial haemorrhage can lead to appearances remarkably like those seen in cases of alleged shaking.” (citing McLellan, Prasad, Punt, “Spontaneous subhyaloid and retinal haemorrhages in an infant,” 1986, a case of Terson’s syndrome due to a ruptured MCA aneurysm). Raised ICP with ONSH with compression of the retinal vessels can also lead to retinal bleeding (citing Hupp et al., 1984). Notes that in central retinal vein occlusion, retinal hemorrhages may be seen out to the far periphery, as may birth hemorrhages. Says that we need studies of retinal hemorrhage extending out to the far periphery in non-traumatic cases. So far, assessment of this finding lacks an objective basis. “From all of the above, I would argue that we do not know how retinal haemorrhages form in most cases of alleged non-accidental injury. The vast majority of cases in my experience have or have had evidence of raised intracranial pressure...” Mentions that there are examples of retinal hemorrhages following simple falls. (Citing Plunkett, 2001). Mentions RH caused by rocket sleds and ejection seats and bungee jumping and says that all of these could be explained by hydrostatic pressure in the vessels of the eyes and face. Takes note of the argument (Levin et al.) that raised ICP causes only peripapillary hemorrhages. Says “This may be the case, but:

-- In the case of pyogenic meningitis mentioned above [in which Luthert's patient a child died of fatal pyogenic meningitis and had diffuse RH with equatorial sparing] there were haemorrhages extending to the ora serrata.

-- Most of the knowledge base concerning peripapillary haemorrhages comes from adults and the retinal circulation in infants may be different.

-- Examination of the back of the eye of an infant is not easy and so the full peripheral extent of haemorrhage may not be appreciated in many instances.

-- In central retinal vein occlusion, retinal haemorrhages may be seen out to the far periphery.

-- Birth haemorrhages may extend to the retinal periphery."

Goes on to discuss failure of autoregulation in the presence of systemic hypoxemia as a possible mechanism. Mentions high-altitude retinopathy as a failure of autoregulation. Says that there has been inadequate study of RH extending to the far periphery. This type of study is of great importance. Luthert himself comes out in favor of the increased ICP mechanism, but wonders about the possible shaking significance of extent to the far periphery. All in all, as to the vitreoretinal traction mechanism and the ICP mechanism, "There is an urgent need for research in this area."

(3) The amount of force from the extent of hemorrhages. For one thing, the patients with the more extensive hemorrhages do worse; this argues that they suffered more force. And Green et al., (1996) documented a correlation between the extent of hemorrhages and the severity of intracranial injury. But other factors may influence the extent of hemorrhage. "Little is known about the temporal evolution of retinal haemorrhages, but clearly they take a finite time to appear and disappear, so timing of examination in relation to onset is important." [In other words, the extent of RH may say more about the timing of the examination than it does about the force of the injury.] Also there is the problem that the force of the injury is usually unknown. He suggests a new hypothesis for the pathogenesis of RH: fluctuating cardiac output and hypoxia following circulatory arrest. he suggests that brain injury or brain stem injury (as per Geddes) would cause circulatory arrest. Restoration of this would lead to "complex evolving pathology that differs from simple hypoxia." [True, but SDH and RH are not seen in children and adults who suffer circulatory arrest and hypoxia from near-drowning or heart attack. -JKR] Mentions the birth process as an example of hypoxia with fluctuating cardiac output that is associated with RH.

(4) Time of injury. It takes 48h for hemosiderin to form following RH. The clearing of RH can be quite variable. Judging by birth hemorrhages, flame hemorrhages probably clear in a few days. Larger deep-seated hemorrhages probably take a few weeks. Resolution of hemosiderin logically may take several months. Speculates about using GFAP and immunostains.

(5) Retinal detachment. Many of these in eye pathology are artifactual.

(6) Perimacular folds. Many retinal folds seen in pathology are artifactual. But a true perimacular fold is recognized as NAI. Discusses whether such a fold indicates vitreoretinal traction, as per Greenwald. Posits retinal edema plus "the bow-string effect of the posterior vitreous, which seems better able to remain attached around the edge of the macula than at the centre, leads to puckering up of the edge of the macula." Plus Lantz's case with Terson's syndrome.

(7) Retinoschisis. Is it caused by traction, or by tracking of blood between the layers? Perhaps weakness of the Muller cell processes in infancy makes retinoschisis from either mechanism more likely in infants.

Conclusions: Why do histology on infant eyes?

- (a) to confirm or refute clinical observations.
- (b) need for quantitative studies
- © timing this or previous injuries -- a definite role.

Inflicted childhood neurotrauma (shaken baby syndrome): ophthalmic findings. Forbes BJ, Christian CW, Judkins AR, Kryston K. Journal of Pediatric Ophthalmology & Strabismus 2004; 41: 80-88. Literature review aimed at ophthalmologists.

ONSH

Optic nerve sheath hemorrhage. The Jackson Memorial Lecture. Walsh FB, Hedges TR, Jr. Am J Ophth 1951; 34: 509-527. See also under Terson's.

Pre-retinal and optic nerve sheath hemorrhage: pathologic and experimental aspects in subarachnoid hemorrhage. Smith DC, Kearns CP, Sayre GP. Trans Am Acad Ophth Otol 1957; 61: 201-211. Reportedly (Plunkett, 2001) says that ONSH is due to retinal venous hypertension or ICP. Not by extension.

Intraocular and optic nerve sheath hemorrhage in cases of sudden intracranial hypertension. Muller PJ and Deck JHN. J Neurosurg 1974 Aug; 41: 160-166. ONSH in 87% of eyes examined after a sudden rise in ICP while only 37% of those eyes had RH.

Optic nerve sheath and retinal hemorrhages associated with the shaken baby syndrome. Lambert S, Johnson TE and Hoyt CS. Arch Ophth. 1986 Oct; 104: 1509-1512. Claims to be the first published report of ONSH in SBS. Found old and recent RH in a 13 mo girl who had vomited during the past four days; babysitter admitted shaking four days earlier. Admitted in tonic-clonic sz, with extensive intraretinal and subretinal RH, retinal detachment, and a large subhyaloid hem obscuring optic n, + interh SDH, midline shift, expired in less than one day. Autopsy: scalp & skull findings not mentioned.

Pathogenesis of RH discussed: In 1967 Gilkes and Mann postulated a sudden rise in intraocular pressure similar to Purtscher's syndrome. Ober in 1980 proposed rapid acceleration/deceleration from shaking. Burton in 1980 proposed air microembolism. Others have proposed a combination of increased intraocular venous pressure, angiospasm, and retinal hypoxia. [Note: Dr. Levin says that in NAT/SBS it is vitreoretinal traction.] Valsalva's hemorrhagic retinopathy distinguished.

ONSH: "ONSH are also commonly observed when the intracranial pressure rises suddenly; in fact, Muller and Deck (infra) noted ONSH in 87% of eyes examined after a sudden rise in ICP*, while only 37% of these eyes had intraocular hemorrhages. Walsh and Hedges demonstrated that ONSH are rarely due to direct extension of SAH from the intracranial to the intraorbital space; rather, they occur secondary to sudden distention of the subarachnoid space from an acute rise in ICP, which ruptures the dural and bridging vessels in the optic nerve vaginal sheaths**. In contradistinction, intraocular hems occur only when the ICP rises still higher, resulting in occlusion of the central retinal vein and its chorioretinal anastomoses. (citing Hayreh, 1977) ONSH probably occur more frequently with SBS than has previously been recognized." P. 1512.

Munger et al. (1993) cite this article for the proposition that "ONSH are seldom extensions of subdural or subarachnoid hemorrhages, [but] occur subsequent to an acute increase in ICP, which suddenly distends the subarachnoid space, damaging the dural and bridging vessels..." But in their work, 100% of the ONSH cases had SDH or SAH.

** Note that this discussion occurred before the discovery of concurrent hemorrhages in the ocular adnexa away from the optic nerve, a finding which would not be explained by these authors' increased ICP hypothesis.

Note that Munger et al found ONSH in 75% of 12 SBS cases, always associated with SDH or SAH.

Ocular and associated neuropathologic observations in suspected whiplash shaken infant syndrome: a retrospective study of 12 cases. Munger CE, Peiffer RL, Bouldin TW, Kylstra JA, and Thompson RL. Am J Forens Med Path 1993 Sep; 14(3): 193-200. Eye histopathology on 12 infants with fatal SBS. See under "Other."

Ocular and optic nerve hemorrhages in abused infants with intracranial injuries. Budenz D, Farber M, Mirchandani H, Park H, Rorke LB. Ophthalmology 1994; 101(3): 559-565. See above.

Ruptured vascular malformation masquerading as battered/ shaken baby syndrome: a nearly tragic mistake. Weissgold DJ, Budenz DL, Hood I, Rorke LB. (Penn). Surv Ophth 1995 May-Jun; 39(6): 509-512. (have) A seven week old infant brought in with a bulging fontanelle and jerking movements. CT showed Intracerebral and subarachnoid hemorrhage and cerebral edema. LP showed fresh blood and elevated ICP. Lived on a vent for a few days post ventriculostomy. The parents seemed unusually stoic and uninvolved to the attending pediatrician. Immediately after the funeral they departed on a vacation cruise. Autopsy showed no evidence of blunt trauma. It showed ONSH, and this finding initially added to suspicions of child abuse. There were no RH(!) There was no SDH. There was a large blood clot in the ventral interhemispheric fissure between the frontal poles. Histology of this disclosed a complex vascular malformation composed of variably-sized, generally thin-walled blood vessels. The ONSH was found to be entirely confined to the subarachnoid space of the optic nerve. The authors make much of the fact that the ONSH was confined to the anterior portion of the optic nerve sheath, and was absent posteriorly. (This is relevant to the possible mechanism of the ONSH.) The authors note that RH have been previously reported from cerebral aneurysms in children, but this is the first report of ONSH. Say that there are three theories of the pathogenesis of ONSH:

- (1) Manschot (1954) postulated direct extension.
- (2) Muller & Deck (1974) postulated transmitted ICP causing rupture of dural feeder vessels.
- (3) Smith (1957) postulated venous hypertension due to ICP, similar to the proposed mechanism of TS, except this time involving the venous drainage of the optic nerve sheath itself.

Bilateral periopticointrascleral hemorrhages associated with traumatic child abuse. Lin KC and Glasgow BJ. Am J Ophth 1999 Apr; 127(4): 473-475. Postmortem examination of the eyes of a 6 month old who died from SDH. Bilateral intrascleral hemorrhages around both optic nerves. Multiple RH. Described in detail.

Optic nerve damage in shaken baby syndrome: detection by beta-amyloid precursor protein immunohistochemistry. Gleckman AM, Evans RJ, Bell MD, Smith TW. Arch Path Lab Med 2000 Feb; 124: 251-256. Axonal injury was documented by b-APP staining in six out of eight AHT infants under 1 yr, and was absent in all of four non-traumatic control infants. "Rapid acceleration-deceleration of an infant's head during intentional shaking should in theory exert stretch or shear forces upon the optic nerves sufficient to cause axonal injury." They studied the optic nerves by H&E stain, neurofilament stain, and b-APP immunostain.

5	pure shake	3 were +
3	shake-slam	3 were +
1	pure slam	1 was +

Controls: 4, all negative.

They could not observe any axonal injury changes by H&E or neurofilament stain. Only by b-APP.

They did observe retraction balls and beta-APP deposition in the NFL of the retina, but only in areas involved by RH. They attribute this retinal axonal injury to disruption of the NFL by RH, and deny a primary mechanical injury of the retinal axons. They make this proposal in the context of arguing that the optic nerve axonal damage is mechanical. Their argument becomes somewhat tortured in this area.

Note by JKR: This finding could also have implications for the visual sequelae, particularly amblyopia, in SBS survivors. Maybe it's not all cortical blindness.

Further note by JKR. Mary Case (Child Abuse Quarterly, July 2000) agrees that this finding implies stretching of the optic nerve and suggests that such a mechanism is responsible for RH.

Levin, 2000a, discussion at 164-165. "The exact mechanism by which ONSH occurs is unknown. It may be due to tracking of blood from the subarachnoid or subdural space and/or due to sudden elevation of intracranial pressure...which is then transmitted down the ONS causing the rupture of bridging vessels within the sheath. Whether the ONS blood has a role in the generation of RH is also unknown... I believe that orbital shaking...may be a major factor in causing direct trauma to the optic nerve resulting in haemorrhage within the sheath, perhaps unrelated to the intracranial bleeding."

Discussion by Dr. Mary Case at San Diego 2000: ONSH is not specific for trauma. It is seen in spontaneous intracranial hemorrhage from any cause, in any age group.

Levin, 2000a, at 164-165: Says that ONSH is characteristic of Terson syndrome and is also commonly seen in SBS. The mechanism of ONSH is unclear. Whether ONSH has a role in generating RH is unknown. Probably blood in the optic nerve sheath does not enter the retina. Discusses retinal venous hypertension due to ONSH. But the characteristic radiating pattern of RH seen in adult retinal vein occlusion is usually absent in SBS.

Optic nerve lesion following neuroborreliosis: a case report. Burkhard C, Gleichmann M, Wilhelm H. (Ger) Eur J Ophth 2001 Apr-Jun; 11(2): 203-206. A 58 y o female devel facial paresis six weeks after an insect bite. Then got NFL hems near the optic disc. Had titers to Borellia burgdorferi, improved on ABX. Final Dx: ischemic lesion (?infarct) of the optic disc.

Unilateral

[Note by JKR: Unilateral is more important on theoretical grounds than it might at first appear. If shaking causes retinal damage through vitreoretinal traction, it should damage both eyes equally. But the fact is that the eye on the side of the (unilateral) SDH is always the one with more severe, sometimes the only one with any RH. This observation suggests that the RH is due to an intracranial mechanism, not an intraocular one.]

Symptoms and signs of intracranial aneurysms with particular reference to retinal haemorrhage. Fahmy JA. Acta Ophth (Scand.) 1972; 50: 129-136. Finding that RH is common in SAH. According to Khan & Frenkel (below), Fahmy reports intraocular hemorrhage in 50 of 154 patients with SAH (32%.) 29 cases were unilateral. See also New England Journal article by

Unilateral RH in non-accidental injury. Tyagi AK, Willshaw He, Ainsworth JR. Lancet 1997; 349: 1224. Three cases of AHT:

5 wk	SDH	ext RH OD
-----	SDH	Unil RH, mult fxx
-----	SDH	Unil RH, humerus metaph

Neuroimaging, physical, and developmental findings after inflicted and non-inflicted traumatic brain injury in young children. Ewing-Cobbs L, Kramer L, Prasad M et al. Pediatrics 1998 Aug; 102(2): 300-307. See under "Shaken." 20 inflicted and 20 noninflicted. 14 inflicted had RH NOS -- 13 bilateral and 1 unilateral.

Unilateral RH in documented cases of child abuse. Drack AV, Petronio J, Capone A. Am J Ophth 1999 Sep; 128(3): 340-344. Four cases examined for suspected child abuse and one 6 month-old examined on routine followup examination for ROP. All four of the abuse cases had extensive retinal or preretinal hemorrhages in one eye only.

6 mo	Infarction	ext RH OS, L neck injuries
------	------------	----------------------------

5 mo	R SDH	ext RH OD, old rib fx
17 m	R>L SDH	ext RH OD
6 mo	Bil SDH	macular and postr RH OS; mult fxx

The one child with ROP had unilateral RH with no cranial injury, not further described in the abstract. They just present these to show that abuse RH is not always bilateral.

Unilateral RH and ipsilateral cranial bleeds in nonaccidental trauma. Paviglianti JC, Donahue SP. JAAPOS 1999 Dec; 3(6): 383-384. Three infants believed to be AHT:

5 mo	R fr SDH w ml shift	numerous RH OD only
4 wk	R fr SAH	numerous RH OD only
10 m	Small L convx EDH	numerous RH OS only

Just presents these to show that abuse RH is not always bilateral.

Shaken baby syndrome. Kivlin JD, Simons KB, Lazoritz S, Ruttum MS. Ophthalmology 2000 Jul; 107(7): 1246-1254. Found that 15% of the RH were unilateral in this series of 123 abused children.

Diffuse unilateral hemorrhagic retinopathy associated with accidental perinatal strangulation: a clinicopathologic report. Shaikh S, Fishman ML, Gaynon M, Alcorn D. Retina 2001; 21: 252-255. A severely asphyxiated 35 week newborn with a tight nuchal cord, possible sepsis, and renal anomalies and R IVH had diffuse RH and ONSH in the R eye only. The authors attribute to strangulation but do not explain why this would be unilateral. [I suggest more likely due to the IVH. -JKR]

Retinal hemorrhage asymmetry in inflicted head injury: a clue to pathogenesis. Gilles EE, McGregor ML, Levy-Clarke G. J Pediatr 2003 Oct; 143(4): 494-499. Argues that RH are definitely related to ICP, because in 5 cases, where the SDH was unilateral, the RH was also unilateral. A pure deceleration model would not predict this; indeed, it would predict that RH should seldom be unilateral at all, let alone correlated with the laterality of an SDH. The authors retrospectively reviewed 14 AHT patients of whom 10 had RH (71%). Of these, 9 had asymmetric or unilateral RH early in their admission. ("Asymmetric" means markedly different retinal injury scores of 4.89 vs 2.56, P=.006.) Of these nine, __ also had asymmetric or unilateral brain lesions. The initial RH were maximal on the side of greatest cerebral injury in 7/10 with RH. One interesting case initially had unilateral RH ipsilateral to the brain injury, and later (within 24 hours) progressed to bilateral RH. They also report two cases where ONSH was unilateral and matched the laterality of the brain pathology. Conclusion: "The distribution of RH after inflicted head injury correlates with acute and evolving regional cerebral parenchymal injury patterns." RH are caused by ICP and hypoxia, with regional pressure gradients and the rate of increase of ICP playing a role.

This article is reviewed by Dr Levin in the Spring 2004 issue of the *Quarterly*. Dr Levin argues that "The conclusion of the authors is not supported by the data they present." Dr Levin cites his own work (Morad et al., Am J Ophth 2002; 134: 354-359) for the proposition that asymmetry of brain findings and asymmetry of RH were not statistically correlated. He goes on to argue that RH caused by increased ICP/retinal vein obstruction have a different pattern than those caused by "typical SBS." He states that ICP/retinal-vein RH "has a very characteristic appearance much different than that demonstrated in this paper and in shaken babies." But Dr Levin is more cautious when it comes to hypoxia causing RH. He states, "The role of hypoxia remains unclear."

Characteristics that distinguish accidental from abusive injury in hospitalized young children with head trauma. Bechtel K, Stoessel K, Leventhal JM, Ogle E, Teague B, Laviates S, Banyas B, Allen K, Dziura J, Duncan C. Pediatrics 2004 Jul; 114(1): 165-168. See above under "In general."

RH	60%	10%
Bilateral RH	40%	1%

The authors make a big deal out of this.

CPR

Extensive retinal haemorrhages in infancy -- an innocent cause. Bacon C, Sayer G, Howe J. BMJ 1978; 1: 281.

Retinal hemorrhages after CPR: literature review and commentary. Levin AV. Pediatr Emerg Care 1986; 2: 269. The first review of this problem. 51 children had CPR: 6 had RH, of which 4 were child abuse, 1 severe HTN, 1 auto vs. ped.

RH after CPR or child abuse. Kanter R. J Peds 1986; 108: 430-432 (have). Did dilated fundoscopy on 54 pediatric pts who had CPR for both trauma and non-trauma. Found only one case of RH in the non-trauma group.

RH in an infant after CPR. Weedn VW, Mansour AM, Nichols MM. Am J Forens Med Path 1990 Jan; 11(1): 79-82. An infant with severe scald, sepsis, cerebral edema, 45 minutes of CPR, found at autopsy histology to have "several large patches of RH situated in the NFL in the equator and posterior pole of both eyes." No intracranial hem or ONSH.

RH after CPR in children: an etiologic reevaluation. Goetting MG and Sowa B. Pediatrics 1990 Apr; 85(3): 585-588. Reports twenty children without trauma who had CPR, whose eyes were examined by direct ophthalmoscopy without dilation, and found 2 with RH -- one a 2 y o drowning with multiple large RH bilat and the other an infant with near-SIDS who had one small posterior RH. Dr. Levin (2000) accepts the latter as consistent with CPR but thinks that the former case must be Valsalva's hemorrhagic retinopathy rather than CPR because "children suffer drowning primarily due to laryngospasm." [???] The work has also been criticized because Goetting and Sowa are not ophthalmologists and they used nondilated direct ophthalmoscopy. Also because their findings in patient 1 have never been replicated elsewhere. See also *letter*, next below.

RH after CPR. Hertle RW, Quinn GE, Duhaime A-C. (letter) Pediatrics 1990 Oct; 86(4): 649-650. Comments on Goetting & Sowa. Says that because they used nondilated direct ophthalmoscopy, they may have underdiagnosed or underestimated the incidence of RH in this setting. "Although a higher incidence of RH due to increased intrathoracic pressure or head trauma occur in the posterior pole than in the peripheral retina, a significant number of hems occur in the peripheral retina and are not likely to be observed by direct ophthalmoscopy." (citing two different authors in *Duane's Clinical Ophth*, 1988). Goetting & Sowa reply accepting the point, but noting that direct opth is more clinically relevant because it is the only method that is available at the bedside.

Ocular findings at autopsy of child abuse victims. Riffenburgh R and Swathyavagiswaran L. Ophth 1991; 98: 1519-1524. One hospitalized child with gram negative sepsis was thought to have Purtscher's syndrome due to CPR.

RH in newborn piglets following CPR. Fackler JC, Berkowitz ID, and Green R. Am J Dis Child 1992; 146: 1294-1296. The piglet model showed no RH after 50 minutes of CPR.

Are RH found after resuscitation attempts? A study of the eyes of 169 children. Gilliland MGF and Luckenbach MW. Am J Forens Med Path 1993; 14: 187-192. Autopsy study. All children who had RH had a condition that would be expected to cause RH independent of having CPR. All children who did not have such a condition and still had CPR did not have RH. Conclusion: CPR does not cause RH.

RH following CPR. Kramer K, Goldstein B. Clin Pediatr 1993; 32: 366-368.

Ocular and optic nerve hemorrhages in abused infants with intracranial injuries. Budenz DL, Farber MG, Mirchandani HG, Park H, Rorke L. Ophth 1994 Mar; 101(3): 559-565. Includes 4 SIDS victims who had CPR. One had "a few erythrocytes" in NFL. None of the SIDS infants had clinical RH.

Retinal hemorrhage in the young child: a review of etiology, predisposed conditions, and clinical implications. Gayle MO, Kissoon N, Herd RW, Harwood-Nuss A. J Emerg Med 1995; 13(2): 233-239. Covers the CPR issue as of mid-1994, considering it a serious DDX issue of RH, although he discusses in detail only the studies of Gilliland (1993) and Fackler's piglet model (1992) which found no ev for CPR RH. He concludes that "The evidence ... for the role of CPR in the genesis of RH is sparse and would suggest that even if CPR is administered, RH should be considered secondary to craniocerebral trauma."

Prevalence of RH in pediatric patients after in-hospital CPR: a prospective study. Odom A, Christ E, Kerr N. et al.. Pediatrics 1997 Jun; 99(6): e3. Studied retrospectively the charts of 43 nontrauma pts who survived in-hospital CPR. 62% had low platelets. 1 had multiple small punctate RH. Study n=too small to achieve statistical significance.

Note on RH/CPR: As of mid-1998 there were only the above four papers in print, plus Gayle's discussion.. These have been discussed by Dr. Alex Levin previously and also at the Second National SBS Conference 1998. His opinion is that these papers show conclusively that CPR does not cause diagnostically significant RH in infants. A meta-analysis of these four papers prepared by Dr. William Perloff of Madison, Wisconsin shows the following statistics from these papers:

Total patients observed	288	
No RH	209	73%
Some RH	79	27%
Due to other causes	74	26%
Possibly due to CPR	4	1%
Unknown cause	1	-%

Child abuse and SIDS. Botash AS, Blatt S, Meguid V. Current Opinions in Pediatrics. 1998; 10: 217-223. Dr. Levin gives this article high marks as a review of RH and CPR.

Child abuse and the eye. 1999 British Working Party. See below under "other." Says very unlikely.

Retinal haemorrhages and child abuse. Levin AV, 2000. (q.v.) "Several studies have now been done all of which come to the same conclusions: CPR-CC only very rarely results in retinal haemorrhages and, when it does, the haemorrhages are few in number and confined to the posterior pole. One group calls the appearance of extensive RH attributed to CPR-CC as 'inconceivable,' and I support this view." (182a)

Bilateral retinal hemorrhages in a preterm infant with retinopathy of prematurity immediately following cardiopulmonary resuscitation. Polito A, Eong, K, Pepka MX, Pieramici DJ. Arch Ophth 2001 Jun; 119(6): 913-914. A 24 week preemie had multiple flame and white-centered RH throughout the retinal area following CPR. No other underlying cause.

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."

Prevalence of retinal hemorrhages and child abuse in children who present with an apparent life-threatening event. Pitetti RD, Maffei F, Chang K, Hickey R, Berger R, Pierce MC. Pediatrics 2002 Sep; 110(3): 557-562. See under "In General, above." The authors prospectively did dilated funduscopy on 73 out of 128 ER admissions for ALTE. Found one (1) case of RH, and this was a 6 week old presenting with ALTE, found to have bilat RH and multiple healing fractures, bilateral acute & chronic SDH. All of their pts had

CPR. Gives a discussion of RH ddx, including a two-paragraph review of the literature on RH-CPR, citing Kanter, 1986; Kramer, 1993; Gilliland & Luckenbach, 1993; Odom, 1997.

Ophthalmic manifestations of inflicted childhood neurotrauma. Levin AL. In: *Inflicted Childhood Neurotrauma: Proceedings of a Conference...*, Bethesda, Maryland, October 10 and 11, 2002. AAP, 2003. With responses by Jane Kivlin, Brian Forbes. See also above under "In general." Says, as to CPR, that six studies have concluded that RH rarely occurs from CPR and that when it does, the RH are few in number and confined to the posterior pole. "I am aware of only one convincing case in the literature. That child had 1 peripapillary intraretinal RH." (p. 130)

The retina in forensic medicine: applications of ophthalmic endoscopy: the first 100 cases. Davis NL, Wetli CV, Shakin JL. *Am J Forens Med P* 2006 Mar; 27: 1-10. Found RH or retinal petechiae in 15 cases, of which 10 had been subjected to either CPR or ventilatory support (67%). "This clearly exceeds the 48% of our total pop which were exposed to these forms of life support, thereby indicating a 19% greater likelihood of retinal petechiae or hemorrhagic activity in cases of resuscitative effort." [This statistical argument from such small numbers seems weak. -JKR]

Seizures

Can convulsions alone cause retinal haemorrhages in infants? Tyagi AK, Scotcher S, Kozeis N, Willshaw HE. *Br J Ophth* 1998 Jun; 82(6): 659-660. 32 consecutive pediatric admissions under age 2 for convulsions were examined by indirect ophthalmoscopy within 48 hours of admission. None had RH. "Therefore, the finding of RH in a child admitted with a history of convulsion should trigger a meticulous search for other causes of these haemorrhages, particularly non-accidental injury."

Hyponatremic seizures in infancy: association with retinal hemorrhages and physical child abuse? Krugman SD, Zorc JJ, Walker AR. *Pediatric Emergency Care* 2000; 16: 432-434. According to Dr. Ricci's review in *Child Abuse Quarterly* for Jan 2002, the authors conclude that hyponatremia does not cause cerebral edema or RH. They conclude that the two cases presented are both shaken babies. Two cases are presented. One a ten week-old shaken baby with SDH, cerebral contusions, rib fractures, RH, Na 122, sz, and a confession of shaking. The other a four month-old with Na 116 due to water intoxication, cerebral edema, sz without papilledema and no injury history, had severe RH. Dr. Ricci comments that this second case is perplexing.

Retinal hemorrhages in infants with hyponatremic seizures. Rubin D, Christian C. *Pediatric Emergency Care* 2001 Aug; 17(4): 313-314. Here Cindy Christian and her colleague present the case of a six month old with posterior-pole RH in the presence of Na 116 sz with no trauma. In the *Quarterly*, Dr Ricci comments that "The mechanism of these RH remains unclear."

Convulsions and retinal haemorrhage: should we look further? Mei-Zahav M, Uziel Y, Raz J et al. (Tel Aviv). *Arch Dis Child* 2002 May; 86(5): 334-335. Versus child abuse. Prospective study of 153 children aged 2 mos to 2 years seen in the ER for sz. Child abuse investigation was undertaken in all cases with RH. They only found one child with RH after a febrile sz, and those were unilateral. Conclusion: "RH after a sz are rare. Such a finding should trigger an extensive search for other reasons, including child abuse."

SIDS

See Levin, 2000a. Dr Levin reviews the literature on this point and finds four references:

1. Odom et al., 1997, two SIDS with RH attributed to CPR (see under "CPR," above)
2. Betz, 1996 finding no RH (in study of shakens) i 10 SIDS at autopsy
3. Budenz et al, 1994 finding only a few NFL erythrocytes in one of 6 SIDS at autopsy

4. Gilliland et al, 1994, finding no RH in

13 SIDS autopsies.

The old (1994) AAP position statement, stating that any RH rules out SIDS. The 2001 position statement does not mention RH.

Note Dr. Krous's opinion (and that of most other forensic pathologists) that any finding of RH rules out SIDS.

Retinal haemorrhages in infancy. Stanton AN. BMJ 1979 Mar; 1(6163): 616. Note Dr Stanton's later publication in 2003 (after her retirement) on the low incidence of substantiated maltreatment in SIDS families. (Under SIDS v. Suffocation).

Traumatic retinoschisis

Definition of retinoschisis. Provided by Dr Levin on NAME-L, July 7, 2006:
The citation in Dorlands refers to two kinds of retinoschisis that are

quite different than the traumatic retinoschisis of SBS. The term retinoschisis means retinal splitting. The ILM is the most superficial

layer of the retina and is firmly attached to the vitreous in childhood.

SBS traumatic retinoschisis most often involves only the splitting away of

the ILM. However, the retina can be sheared at any deeper layer as well. I

have even seen one case in which the inner and outer photoreceptor segments were sheared away from each other. At autopsy, this shearing

must be distinguished from cystoid macular edema as well (which is rarely

seen in SBS). SBS retinoschisis may or may not be found with a retinal

fold at its border and there may or may not be disruption of the RPE underlying that fold (clinically seen as a circumlinear white line).

At

autopsy, the central part of the schisis dome may be collapsed to create a

"crater-like" appearance within the confines of the surrounding fold.

SBS

retinoschisis may or may not be associated with a true focal detachment of the sensory retina.

The terminology must also be distinguished from subhyaloid blood which is

blood between the posterior vitreous face and the ILM. Preretinal blood

is technically the same as subhyaloid but some ophthalmologists, especially in caring for adults, will use the term preretinal interchangeably with sub-ILM.

Hoping this is helpful. You may feel to post this message on your NAME

listserv.

Alex

Alex V. Levin, M.D., MHSc, FAAP, FAAO, FRCSC
 Staff Ophthalmologist
 Co-Director, Ocular Genetics Programme
 Director, Pediatric Ophthalmology Fellowship Programme
 Staff Paediatrician, Suspected Child Abuse and Neglect (SCAN)
 Programme
 The Hospital for Sick Children
 Department of Ophthalmology, M158
 555 University Avenue
 Toronto, Ontario M5G 1X8
 Phone 416-813-6524, Fax 416-813-6261

Joint Centre for Bioethics
 Professor, Departments of Paediatrics, Genetics, and Ophthalmology and

Vision Sciences
 Director, Postgraduate Bioethics Education
 University of Toronto

The discussions by Dr. Levin (2000a: 163-165, 169 and in Reece, 2d edition, 2001) are to be consulted.

Circular fixed fold of the retina. Wolter JR. Am J Ophth 1965; 60: 805-811. Cited by Dr. Tongue (1991) describing a circumferential retinal fold in one eye obtained from an adult who died in a motor vehicle accident. The fold was located anteriorly...and was associated with [an epiretinal membrane.]”

The vitreous, its structure, and relation to the ciliary body and retina. Hogan MJ. Invest Ophth 1963; 2: 418-445. Cited by Massicotte et al. (1991) for the proposition that the vitreoretinal attachments between the posterior hyaloid face and the internal limiting membrane are particularly tenacious in infant eyes. See also the independent anatomical work of Sebag, 1991.

Traumatic retinoschisis in battered babies. Greenwald MJ, Weiss A, Oesterle CS, and Friendly DS. Ophthalmology 1986; 93: 618-625. Paper presented at the American Academy of Ophthalmology in 1985. Presents five patients aged 2m to 9m who presented with RH and stupor. One had a skull fx, the others had no soft tissue or skull injury clinically. All were nonaccidental head trauma, basically SBS. He proposes traction on the retina as the mechanism of RH, giving a separation of tissue planes within the retina. “The pathophysiological mechanism of this syndrome may be quite unlike those of other hemorrhagic retinopathies.” (By this they mean that this retinopathy is not caused by increased intracranial pressure or by vasculopathy, the commonly presumed mechanisms of other hemorrhagic retinopathies.) They report their followup observation of these patients over a period of years.

Case 1 a 9 mo BF SBS noted bil RH on adm, had boat-shaped hems seen on the 3d day in postr pole. CT sh no ICH but LP + rbc's. 6 mos later the child was clinically blind and had retinal gliosis.

Case 2 a 8 mo BM w confession to shaking. +SDH, postr RH and preretinal hems; sent home; readm w large SDH's, extensive intraretinal hems; two weeks later on reexam the vitreous had become filled with blood. One year later there was optic nerve atrophy (clinically blind), and “in the R macula there was a large intraretinal cyst or schisis cavity surrounded by a ring of apparent gliosis....”

Case 3 10 mo BF w AHT, fx, large SDH, CVA, oph exam on day 17 sh dense vitreous hem; at one year had optic nerve atrophy and “Both retinas had a gliotic appearance, which was especially marked in a ring-shaped distribution surrounding the right macula...”

Case 4 a 5 mo WF SDH, bilat RH on adm. On day 2 a large dome-like elevation of the retinal surface in the posterior pole...The retina immediately surrounding this structure, which was described as retinoschisis, appeared thick and opaque.

Case 5 a 2 mo BM bilat SDH +RH bilat in ER, no opth until 5 mos later had disc pallor, and on the right "A large elevated apparently cystic lesion occupied the macula and the papillomacular area, surrounded by gliotic-appearing retina.

The authors note that the eyes that failed to recover vision were distinguished in the acute phase by "a large cystic or dome-like lesion in the macular region." 622 "We propose the following explanation to account for the features that characterize traumatic retinoschisis in battered babies: when an infant is shaken, its head is subjected to repetitive accelerations and decelerations as it whiplashes back and forth...traction on the retina..." 623.

The authors briefly review the previous literature on RH, including Gilkes & Mann, 1967; Friendly, 1971; Harcourt, 1971; Mushin, 1971; Mushin & Morgan, 1971; Jensen, 1971; Tomasi, 1975; Eisenbrey, 1979; Harley, 1980; Ober, 1980; San Martin, 1981. They rely on Caffey's postulate of a shaking mechanism with respect to the numerous cases in which "there is no skull fracture or external signs of a blow to the head." They begin by saying that, "The pathogenesis of intraocular hemorrhages in battered babies remains uncertain. Retinal and vitreous hemorrhages are found frequently in patients with intracranial bleeding of any etiology, probably in consequence of acute elevation of intracranial pressure." (citing Walsh FB and Hoyt WF, *Clinical Neuroophthalmology*, 3d ed. Williams & Wilkins, 1969 at 1786-7 and Khan SG, Freinkel M. Intravitreal hemorrhage associate with rapid increase in intracranial pressure (Terson's syndrome), *Am J Ophth* 1975; 80: 37-43.) "Their occurrence has been documented in abused infants with neither blood nor elevated pressure intracranially, however." (citing Tomasi, 1975). They say that some abusive hemorrhages resolve, while others lead to optic nerve atrophy or macular cysts. The authors discuss birth hemorrhages: "RH occurring in infants in other circumstances (e.g. in newborns following vaginal delivery) virtually always resolve to leave the fundus completely normal." (In other words, birth hemorrhages behave differently than abusive hemorrhages, another fact that could imply a different mechanism for abusive hems.)

There is a discussion by another opth who doubts that the mechanism of the dome-like lesion is retinoschisis, but rather hemorrhage secondary to increased venous pressure. "Hems in all layers of the retina occur in a number of nontraumatic disorders associated with changes in cerebrovascular dynamics such as central retinal vein occlusion, high altitude retinopathy, and SAH secondary to ruptured intracranial aneurysms." 624. Duhaime, 1998 includes vitreoretinal traction as one possible mechanism, along with venous pressure and SAH, *infra*. Massicotte, 1991 affirms vitreoretinal traction as the mechanism.

Cases from this article, namely cases 2 and 5 (confessed shaking cases), are discussed by Leestma (2005) as follows: "In case 2, the foster mother of the child admitted to shaking the baby in an attempt to stop seizure he was having. He had had seizures since age 4 months, and may have been abused in his original home. After other injuries to the child, he was removed from the foster home and placed in another home. In case 5, the child was admitted apneic. CPR had been attempted by the father, and he had also shaken the baby in an attempt to revive him over a 10-minute period. The child had prior apneic episodes. Subdural hematomas required repeated aspirations and shunts. The child was not examined by an ophthalmologist until 7 months of age..."

Retinal folds in the SBS. Gaynon MW, Koh K, Marmor MF and Frankel LR. *Am J Ophth* 1988; 106: 423-425. Apparently the first description of perimacular folds in child abuse, other than the findings of "circular gliosis" described by Greenwald et al. Cited by Massicotte et al. (1991) for the proposition that "In addition to hemorrhages located in the subdural space in the optic nerve, vitreous, and all layers of the retina, peculiar circinate **perimacular folds** have been described in cases of presumed shaken baby syndrome," and for the proposition that "The folds, considered to result from lateral displacement of the neurosensory retina by vitreous traction, were attributed to the shaking episode." Cited by Munger et al. (1993) for the proposition that perimacular folds are almost always present in SBS. Munger found them in only 42% of 12 SBS infants.

Ocular and associated systemic findings in suspected child abuse. Elner SG, Elner VM, Arnall M, Albert DM. Arch Ophthalmol 1990 Aug; 108(8): 1094-1101. Notes that shaking and blunt trauma literatures have developed, with Japanese authors stressing blunt trauma. Purpose: "To assess the pathogenesis of ocular injury and death associated with alleged child abuse." Reviews the autopsy and eye findings in ten deaths from child abuse. All had evidence of blunt head trauma at autopsy, although some of it would have been clinically covert. 7 had RH with VH and ONSH. These 7 are presented in detail. Ages 17m, 4y, 5m, 7m, 7m, 12m, 4m. The histories were, "17m fell from a high chair," 4y no hx, "5m found dead in crib," "7m fell from a changing table," "7m fell," "12m found unr," "4m no hx." 4 of these had hemorrhagic retinoschisis and 3 had tractional retinal folds temporal to the macula. Hemosiderin in 3. 5 cases had intrascleral hemorrhage from the circle of Zinn at the opticocleral junction. 4 cases demonstrated anterior segment injuries characteristic of direct ocular trauma. Discussion notes the report of Greenwald (1986) as to traumatic retinoschisis and Gaynon (1988) as to retinal folds. "in 4 of our cases, massive intraretinal hem dissected the internal limiting membrane or necrotic nerve fiber layer, producing varying degrees of retinoschisis. Perimacular folds were also noted in 3 cases... The folded retina was hemorrhagic and often necrotic, with either taut vitreous fibrils emanating from the crest of the fold or the inner retinal layers torn from the retinal surface. The histopathologic appearances of the retinal hemorrhages, folds, and schisis in our cases were strikingly similar to those reported by Cox in experimental blunt, nonpenetrating eye trauma." (citing Cox MS, Retinal breaks caused by blunt nonpenetrating trauma at the point of impact. Trans Am Ophthalm Soc 1980; 78: 414-465.) Goes on to discuss theories of the mechanism of RH/Vh in abused children. Theories of (1) Acute intracranial hypertension -- retinal venous hypertension (citing Khan & Frenkel, 1975); (2) transvenous transmission of intrathoracic pressure (Purtscher's syndrome); (3) direct head trauma. "The full-thickness hemorrhagic retinal necrosis... and hemorrhagic retinoschisis and perimacular retinal folds... suggest that severe anteroposterior acceleration-deceleration forces as proposed by Greenwald et al directly produce retinal injuries in abused children who die of blunt head injury... Blunt head trauma, [? as opposed to shaking or chest compression? -- JKR] ... may be necessary to produce significant vitreoretinal traction..." Discusses the possible pathogenesis of ONSH with no definite conclusion. Possible direct optic nerve trauma. Gives a lengthy discussion of the evidence of shaking versus impact as of 1990, and points out that head trauma can be missed or not evident during life. Photomicrographs: a large intraretinal schisis cavity with the ILM separated from the rest of the retina. A tractional retinal fold temporal to the macula; the inner limiting membrane has been torn from the fold and surrounding retina... There is a comment at Arch Ophthalmol 1991 Mar; 109(3): 321-322.

Vitreoretinal traction and perimacular retinal folds in the eyes of deliberately traumatized children Massicotte SJ, Folberg R, Torczynski E, Gilliland MGF, Luckenbach M. Ophthalmology 1991; 98: 1124-1127 Peculiar ringlike perimacular folds have been described by Gaynon et al. (1988) and Elner et al. (1990) in child abuse and attributed by those earlier authors to head impact (Elner) or to shaking (Gaynon.) These present authors describe such folds in three children with fatal abusive head trauma both with and without evidence of impact. Case 1 a shaken 7.5 month old with acute SDH and brainstem herniation, lived a matter of hours, no skull fracture or subgaleal hemorrhage, had perimacular folds in both eyes, "focal serous retinal detachments in the posterior pole," scattered intraretinal and vitreous hemorrhages, plus ONSH. "Vitreous was separated focally from the ILM of the retina but remained adherent to the apex of the fold; a hemorrhagic retinoschisis cavity was identified beneath the ILM (fig. 2)." Case 2 a 9 month old who was shaken and thrown on the floor, multiple occipital fractures, lived 15 hours to brain death, subdural hemorrhage, brain edema, rib fractures, etc., eyes showing ONSH, perimacular folds, and "intraretinal hemorrhages just posterior to the ora serrata." "The vitreous was partially separated from the ILM, but remained so adherent to this structure at the apex of the fold that the membrane was torn away from the retina (fig. 8). The vitreous that was attached to the free end of the ILM over the fold could be traced anteriorly to the retina just posterior to the ora, the site of subinternal limiting membrane hemorrhages..." Case 3 a 13 month old admitted comatose with ear contusions, SDH, RH, died in 29 hours, had occipital subgaleal hemorrhage at autopsy. "The vitreous, partially separated from the retina, remained adherent to the ILM at the apex of the retinal folds and could be traced anteriorly to the sub-ILM hemorrhage in the retina just posterior to the ora serrata" +ONSH.

The authors reach three conclusions: (1) "Our study confirms the role of vitreous traction in formation of perimacular folds, as evidenced by the partial vitreous detachment except at the apices of the folds. Additionally, the massive retinal hemorrhage at the vitreous base, a finding not previously described

in eyes of deliberately traumatized children, also may be explained by vitreous traction: a strand of vitreous connects the hemorrhage to the ora to the apex of the folds. The vitreoretinal attachments between the posterior hyaloid face and the internal limiting membrane are particularly tenacious in infant eyes (citing Hogan, 1963), and account for the posterior location of the perimacular folds as well as the formation of sub-ILM hemorrhages at the vitreous base.” (2) Shaking alone can cause perimacular folds. (3) Hypothesis that perimacular folds may be specific for child abuse. Testing this hypothesis would require a study comparing abused and non-abused children.

Age-related differences in the human vitreoretinal interface. Sebag J. Arch Ophthalmol 1991 Jul; 109(7): 966-971. (have) Dr. Sebag, who had already performed extensive previous studies of the vitreous humor, was studying the aging process in the human vitreoretinal interface, because lesions of this interface, particularly in the nature of pathologic vitreous traction on the retina, cause eye disease in aging. Such traction at the periphery causes retinal detachment, and at the macula causes macular holes and premacular membrane formation and proliferative vitreomembranopathy. The most common lesion at the macula is dehiscence of the vitreous cortex from the internal limiting membrane, resulting in collapse of the vitreous (posterior vitreous detachment (PVD), which causes retinal detachment via vitreoretinal traction at points of adhesion.) The present study compares the vitreoretinal interface in eyes from young individuals and older individuals. The specific questions addressed are as follows: Does the strength of vitreoretinal adhesion differ at the equator as compared with the posterior pole? Does the strength of vitreoretinal adhesion vary with age? Is strong vitreoretinal adhesion focal (limited to the central macula, optic disc, and along blood vessels), or is the adhesion more diffusely distributed in the posterior pole? He dissected 59 fresh normal donor eyes from persons of the following age groups: 0-10: n=7; 11-20: n=8; 21-40: n=7; 41-60: n=10; 61-80: n=14; 81-100: n=13. and examined the vitreous and vitreal attachments with dark field microscopy and by dissecting the retina off the vitreous. He dissected away the outer posterior layers of the eyeball (the sclera, the choroid, and the retina.) The retina was peeled off the vitreous by finding a surgical plane between the vitreous and retina at the equator and by peeling the retina backward to the posterior border of the vitreous base and posteriorly to the optic nerve. The entire fresh specimen was examined by dark-field slit microscopy. He collected samples of the retina and the vitreous cortex from peripheral and posterior pole sites and performed SEM and TEM on them. Dark-field microscopy showed that in all individuals over 21 years of age, the posterior vitreous cortex had two holes in it, one over the macula, and one over the papilla. (Fig. 1) He performed TEM on the retina from the macular area and found that its ILM was intact and perfectly smooth on the anterior surface, confirmed by SEM; there were no vitreous fibrils attached to the ILM at this area. (Fig. 2) Similarly, the vitreous cortex from this (macular) area had no internal limiting membrane elements. There was a clean separation. BUT: In six of the 15 eyes from individuals aged 20 years or younger, including four eyes from individuals aged 10 or younger, the appearance of the posterior vitreous was entirely different. Fig. 4 shows this area from a 9 month-old infant. There is a prepapillary hole but no premacular hole. On the premacular area of the vitreous in these eyes he demonstrated the presence of adherent internal limiting membrane material. (Fig. 5) The same was true in the eye of a 14 year old boy. (Fig. 6) There was no premacular hole and there was adherent ILM and portions of Muller cell processes, with cellular disruption showing that these cells had been torn during dissection. (Fig. 7) Results: In all eyes from individuals aged 21 years or older, adhesion between the ILM of the retina and the vitreous cortex in the posterior pole was weak. However, in 40% of eyes from individuals aged 20 years or younger, adhesion between the ILM and the posterior vitreous cortex was stronger than Muller's cell itself. This topographic difference is consistent with the clinical observations by Schachat and Sommer, who suggested that stronger vitreoretinal adhesion at the macula accounted for a variety of vitreomaculopathies associated with posterior vitreous detachment. Moreover, these results also demonstrate that this strong adhesion is not focal, i.e. limited to the macula, the optic disc, and along retinal blood vessels. Rather, the phenomenon is distributed throughout the posterior pole in a sheetlike configuration. He goes on to discuss the possible implications of this for the disease entity of posterior vitreous detachment. He proposes that with aging, the ILM gradually thickens. This thickening interferes with the ability of Muller cells to secrete extracellular matrix to maintain the attachment of the ILM to the vitreous. This results in the possibility of dehiscence. Dehiscence can in turn allow liquid vitreous to dissect between the vitreous cortex and the ILM, resulting in collapse of the vitreous and traction on the retina at residual points of adhesion, thus leading to rhegmatogenous events. The age-related difference found would explain why PVD occurs more commonly in the older age groups. See also Hogan, 1963, and refs given in this article.

The ophthalmologist's role in diagnosing child abuse. (editorial). Andrea C. Tongue, MD. Ophth 1991 Jul; 98(7): 1009-1010. An invited editorial commenting on Massicotte et al. She notes their statement that controlled studies are needed to establish that perimacular folds do not occur in accidental trauma. "Of interest in this respect is an isolated case report by Wolter (1965) describing a circumferential retinal fold in one eye obtained from an adult who died in a motor vehicle accident. The fold was located anteriorly ... and was associated with [an epiretinal membrane.]" She points out that in her experience folds are always associated with massive retinal hemorrhage, as is true in Massicotte's cases. She wonders if the massive hemorrhage itself could be causing the folds, possibly by pushing the retina off the pigment epithelium. "Until it is unequivocally proven that retinal folds are secondary to dynamic vitreous traction and shaking and not some other factors, it is imperative that we not equate retinal folds with child abuse, just as we cannot equate the presence of retinal hemorrhage with child abuse." "Nonaccidental trauma is still a diagnosis of exclusion." Emphasizing that not enough studies on accidental injury have been done to obviate the need for careful differential diagnosis in each patient.

Retinal findings after head trauma in infants and young children. Buys YM, Levin AV, Enzenauer RW, Elder JE, Letourneau MA, Humphreys RP, Mian M, Morin JD. Ophth 1992 Nov; 99(11): 1718-1723. RH are strongly suggestive of AHT. Review of the literature up to 1992. Does not deal with TR.

Accidental head trauma and retinal hemorrhage. Johnson DL, Braun D, Friendly D. Neurosurgery 1993; 33: 231-235. 140 accidentals with severe enough injury to have either skull fracture or ICH were examined prospectively. Only 2 had RH, both side-impact MVA's with severe head injury. "The vitreous of the young eye is firmly attached by a dense web of collagen fibrils along major retinal vessels. The retinal capillary networks (one in the nerve fiber layer and the other between the inner nuclear and outer plexiform layers) are suspended between the retinal precapillary arterioles and postcapillary venules. A firm attachment also exists between the vitreous and the lens. Shaking and abrupt deceleration impels the vitreous complex back and forth, exerts traction forces on the retina, and tears the vascular attachments. The magnitude of the forces determines the extent of the hemorrhage, but the threshold for hemorrhage is not known."

Retinal folds in Terson syndrome. Keithahn MAZ, Bennett SR, Cameron D, Mieler WF. Ophth 1993 Aug; 100(8): 1187-1190. See below under "Terson's syndrome."

Outcome and prognosis of whiplash shaken infant syndrome: late consequences after a symptom-free interval. Bonnier C, Nassogne M, Errard P. Dev Med Child Neurol 1995; 37: 943-956. Increased ICP is present in 55%-85%. See also the same group's art Neuroimaging of intraparenchymal lesions predicts outcome in SBS (2003).

Bilateral rhegmatogenous retinal detachments with unilateral vitreous base avulsion as the presenting signs of child abuse. Gonzales CA, Scott IU, Chaudry NA, Oster AS, Hess DJ, Murray TG. Am J Ophth 1999 Apr; 127(4): 475-477. A 7 year old boy with no external signs of trauma but with retinal detachments as the only presenting sign. Required vitrectomy.

Optic disc neovascularization following severe retinoschisis due to shaken baby syndrome. Brown SM, Shami M. Arch Ophth 1999 Jun; 117(6): 838-839.

[Ocular pathology of child abuse] (GER). Rohrbach JM, Benz D, Friedrichs W, Thiel HJ, Wehner HD. (Tubingen). Klin Monatsbl Augenheilkd 1997 Mar; 210(3): 133-138. Case report of the eye histology on a 2 year old girl with severe intracranial hemorrhage due to abuse who survived two days. Preretinal, choroidal, and scleral hemorrhages as well as ONSH. One eye had a circular perimacular fold and hemorrhagic retinoschisis. A craterlike appearance of the central retina, hemorrhagic retinoschisis, and scleral hemorrhage in the Circle of Zinn – this combination suggests child abuse almost with certainty.

Retinal haemorrhages in child abuse. Levin AV. In: T J David, ed., *Recent Advances in Paediatrics* 18. London: Churchill Livingstone, 2000, pp. 151-219. ISBN 044306430X. This review contains discussion of all of the RH subtopics. "Traumatic retinoschisis has never been described in children due to any entity

other than SBS so its presence is diagnostic. However, in adults, subhyaloid or sub-internal limiting membrane haemorrhages due to Terson syndrome and other disorders may appear remarkably very similar. (citing Kuhn, 1998; Keithahn, Retinal folds, 1993; Toosi, 1987.) Retinal folds (sometimes referred to as perimacular folds) may even be present. (citing references) Says that the similarity is greatest when the surface of the schisis cavity is the internal limiting membrane. But in these adults, the primary cause ... is readily apparent and other retina findings characteristic of SBS are often absent." P. 175. See p. 176-177 (summarized under Kuhn's 1998 article on TS, below) for Dr. Levin's detailed argument that TS macular cysts are different from TR macular cysts, by saying that . Describes TR as clinically recognized as "the curvilinear edge, sometimes haemorrhagic, with or without a fold of retina or an underlying depigmentation of the retinal pigmented epithelium."

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 Maltreatment and Trauma Press, 2001, p. 143-144. "Another distinctive but infrequent ocular finding in SBS is the circular retinal fold around the macula... There is still some controversy over the cause of these folds. The two possibilities are vitreous traction on the retina versus intraretinal bleeding as in Terson's syndrome... [further discussion of dome-like hemorrhages...] At the Children's Hospital of Wisconsin, circular retinal folds have been found infrequently."

Perimacular retinal folds from childhood head trauma. Lantz PF, Sinal SH, Stanton CA, Weaver RG Jr. BMJ 2004 Mar; 328: 754-756. Case report of a 14 month old male driven by private car to the hospital after being found by the father on the floor with a 40-lb big-screen TV on top of him. Pediatric ophthalmologist documented and photographed extensive RH with bilateral perimacular folds. The 3 year-old sibling was taken out of the home; in custody, the sibling volunteered statements that corroborated the father's account. Study of the telephone records and travel distances corroborated the father's account. The father's account remained consistent. Experiments proved that the toddler could have caused the TV to fall on him by climbing on the tray on the front of it. Survived 18 hours. Autopsy showed complex skull fracture, thin right-sided convexity and interhemispheric SDH, dural laceration, SAH, EDH, bilateral cortical [fracture] contusions, severe cerebral edema, traumatic retinoschisis, ONSH, extensive RH, and perimacular folds. Includes a review of the literature with a complete table of all the articles and book chapters pertaining to perimacular folds. Found that these consist of noncomparative observational reports and unsystematic narrative review articles. Concluded that "Statements in the medical literature that perimacular retinal folds are diagnostic of shaken baby syndrome are not supported by objective scientific evidence." Also that "Clinical and autopsy studies with appropriately matched controls are needed to determine the causal mechanism of perimacular retinal folds and their specificity for abusive head injury. Until good evidence is available, we urge caution in interpreting eye findings out of context."

This article is commented on critically by Gnanaraj et al. (including MGFG and Dr Levin) in their Ocular manifestations of crush head injury in children (2005), (see below) in which they retrospectively studied the eye findings in 17 children admitted with crushing head injuries and did not find retinal folds or retinoschisis in any, although they did find RH in 5, including one with preretinal and blot hemorrhages extending to the equator and one with RH extending to the ora serrata. These authors emphasize the fact that the report of Lantz et al. is unique in all the literature and say that a case of alleged crushing head injury with retinal folds should be accepted as non-inflicted only after the most rigorous scrutiny. They also criticize Lantz et al. for not dissecting the cervical spine to RO inflicted trauma.

Images in clinical medicine: circumpapillary retinal ridge in the shaken baby syndrome. Hylton C, Goldberg MF. NE J Med 2004 Jul; 351(2): 170. A color fundus photograph showing an early perimacular fold in a 5 month old female whose father shook her vigorously, allegedly in an attempt to rescue her from a choking spell.

Ocular manifestations of crush head injury in children. Gnanaraj L, Gilliland MGF, Yahya RR, Rutka JT, Drake J, Dirks P, Levin AP. Eye 2005 Nov 25; <http://www.nature.com/eye/journal/v21/n1/full/6702174a.html> retrospectively studied the eye findings in 17 children admitted with crushing head injuries, including 11 from television tipovers, and did not find retinal folds or retinoschisis in any, although they did find RH in

5, including one with preretinal and blot hemorrhages extending to the equator and one with RH extending to the ora serrata.

Conclusion: "Intraretinal and preretinal haemorrhages, predominantly in the posterior pole, can occur in crush injury to the paediatric head. Haemorrhage under the internal limiting membrane or extending to the ora serrata were only seen in situations where crush injury was part of a fatal trauma scenario related to motor vehicles. Retinal folds and the typical macular retinoschisis associated with abusive head injury were not observed."

Reviews the literature on crush injuries of the head in children re RH. All the published reports do not report any eye findings.

The authors criticize the 2004 BMJ case report of Lantz et al. (above). The authors admit that Lantz et al. did an extremely thorough factual investigation to rule out child abuse but still argue that (1) it is unique. (2) the absence of fractures and injuries typical of crushing head injury, such as orbital fractures, hemotympanum, otorrhea, (3) the authors failed to do a neck dissection to rule out SBS. (4) speculate that that particular toddler might have had an undiagnosed coagulopathy, such as vitamin C deficiency. (5) They (Gilliland et al.) cannot posit a pathophysiologic mechanism from a TV crush that would be expected to cause TR or perimacular folds, given that in their belief these lesions can only be caused by vitreoretinal traction. But they admit that their own sample was small, and indeed they might have observed some retinal folds if they had had more subjects. They also admit that in their series, they did see small areas of blood under the ILM, but emphasize that these were small. (Retinoschisis "may be either deep to the nerve fiber layer or superficial (involving only the internal limiting membrane)"). They explain these areas by speculating that "It is ... possible that the eye, like the brain, could be subject to differential shearing forces as a result of the crush resulting in small areas of focal vitreous traction with tension on the ILM." So, since it's just a question of the *size* of the sub-ILM hemorrhages, "we cannot rule out the possibility that a larger sample size would reveal other ocular findings not noted here." However, the authors emphasize the uniqueness of Lantz's case and caution the profession, particularly nonophthalmologists, that "Before attributing retinal haemorrhages or perimacular folds to a crush injury, as opposed to inflicted neurotrauma, there must be history and physical findings consistent with a crush mechanism, and meticulous investigation must be undertaken."

Retinal hemorrhage and pediatric brain injury: etiology and review of the literature. Aryan HE, Ghosheh FR, Jandial R, Levy ML. J Clin Neuroscience 2005; 12: 624-631. Dr Levin's review in the *Quarterly* for Summer 2006 makes two useful statements: "I also must challenge the authors' statement that blood underneath the internal limiting membrane (ILM) is 'preretinal.' I believe most authors would consider this a form of retinoschisis, since preretinal blood would be more correctly termed as blood in front of the ILM." [See the diagram under "Histology," above.] Also, "The authors incorrectly ascribe the circumlinear white curved line that outlines the schisis cavity as a result of resolved hemorrhage when, in fact, this has been histologically shown to represent traumatic disruption of the underlying retinal pigment epithelium."

Retinoschisis. Electronic correspondence between Dr Levin and MGF Gilliland received via NAME-L, July 7, 2006. The citation in Dorland's refers to two kinds of retinoschisis that are quite different than the traumatic retinoschisis of SBS. The term retinoschisis means retinal splitting. The ILM is the most superficial layer of the retina and is firmly attached to the vitreous in childhood. SBS traumatic retinoschisis most often involves only the splitting away of the ILM. However, the retina can be sheared at any deeper layer as well. I have even seen one case in which the inner and outer photoreceptor segments were sheared away from each other. At autopsy, this shearing must be distinguished from cystoid macular edema as well (which is rarely seen in SBS). SBS retinoschisis may or may not be found with a retinal fold at its border and there may or may not be disruption of the RPE underlying that fold (clinically seen as a circumlinear white line). At autopsy, the central part of the schisis dome may be collapsed to create a "crater-like" appearance within the confines of the surrounding fold. SBS retinoschisis may or may not be associated with a true focal detachment of the sensory retina. The terminology must also be distinguished from subhyaloid blood which is blood between the posterior vitreous face and the ILM. Preretinal blood is technically the same as subhyaloid but some ophthalmologists, especially in caring for adults, will use the term preretinal interchangeably with sub-ILM.

Hoping this is helpful. You may feel to post this message on your NAME listserve.

Alex

Alex V. Levin, M.D., MHSc, FAAP, FAAO, FRCSC
 Staff Ophthalmologist
 Co-Director, Ocular Genetics Programme
 Director, Pediatric Ophthalmology Fellowship Programme
 Staff Paediatrician, Suspected Child Abuse and Neglect (SCAN)
 Programme
 The Hospital for Sick Children
 Department of Ophthalmology, M158
 555 University Avenue
 Toronto, Ontario M5G 1X8
 Phone 416-813-6524, Fax 416-813-6261
 Joint Centre for Bioethics
 Professor, Departments of Paediatrics, Genetics, and Ophthalmology and Vision Sciences
 Director, Postgraduate Bioethics Education
 University of Toronto

"MGF Gilliland" <mggillil@pcmh.com>

2006-07-07 10:00

To

NAME-L@listserv.cc.EMORY.EDU

cc

"MGF Gilliland" <MGgillil@pcmh.com>

Subject

Re: Retinoschisis

SILM hemorrhages can be quite large and do "split the retina". As I understand the use by ophthalmologists, retinoschisis is visible clinically and is not necessarily a traumatic condition. An older Dorland's (1988) describes the condition as occurring in the nerve fiber layer in the juvenile form and in the external plexiform layer in the adult form. It goes on to say that "The disorder is usually more benign and slowly progressive than retinal detachment".

The retinoschisis in Vitreoretinal Traction and Perimacular Folds in the Eyes of Deliberately Injured Children published in *Ophthalmology* 1991;98:1124-1127 was visible grossly but had a delicate microscopic finding of vitreal attachment to the ILM.

mg

MGF Gilliland MD

Professor

Brody School of Medicine at East Carolina University

Department of Pathology and Laboratory Medicine

Forensic Division

Greenville, NC 27858-4354

Perimacular retinal folds simulating nonaccidental injury in an infant. Lueder GT, Turner JW, Paschall R. Arch Ophth 2006 Dec; 124(12): 1782-1783. According to MGFG's summary in the Spring 2007 *Quarterly*, case report of a four month old who was having his diaper changed on the floor when a 12 year-old fell on his head. Immediately unconscious, had a displaced comminuted R parietal skull fracture with SDH and IVH. R eye had VH, L eye had perimacular folds, extensive multilayer RH. Photomicrograph of the retinal folds given. "The authors postulate that the mechanism of the retinal folds was a rapid rise in ICP transmitted through the optic nerve, resulting in extravasation of fluid from the intraretinal vessels and separation of the ILM. The authors cite similar findings in adults with Terson syndrome (citing Keithahn, 1993), omitting mention of the rarity of Terson syndrome in children (citing Schloff, Levin et al., Retina findings in children with intracranial hemorrhage, 2002)." MGFG calls this art a valuable addition to the literature. MGFG notes the editorial by Dr Levin

Retinal hemorrhages of crush head injury: learning from the outliers (editorial). Levin AV. Arch Ophth 2006 Dec; 124(12): 1773-1774. Editorial comment on Lueder and Lantz, both of which were crushing

head injuries. I purchased online access to this editorial. "Perimacular folds appear to be unique to injury only in the setting of head crush and SBS. If we hypothesize that there is no biologic anomaly, then we must try to identify what is unique about head crush injury as compared with other forms of trauma. Lueder et al. are correct to identify the unique severe acute compressive rise in ICP as a possible factor. Although ICP appears not to be correlated with retinal hem in SBS (citing his earlier 2002 article), Terson syndrome appears to be uncommon in children (citing Schloff, Levin et al., Ophth 2002 -- see below under "Terson's syndrome"), and experience tells us that children with raised ICP rarely have RH and when they do they are mild and confined to the pp -- the type of ICP rise in crush head injury is extraordinary. Perhaps more importantly, the common presence of orbital fracture in head crush injuries suggests that there may be acute, severe shearing forces applied to the globe..." That's Dr Levin's argument. As I read it, he doesn't deny that the abrupt ICP theory may be correct, but he puts forward his own theory of direct orbital trauma with ocular shearing as an alternative theory. A potential fatal flaw in his argument is Terson's syndrome. If that too causes perimacular folds in infants, the vitreoretinal-traction theory will go out the window, and clearly Dr Levin is aware of that. Dr Levin acknowledges that Terson's syndrome does occur in children (as we know it does, since I have documented it). So we are down to exactly what form it takes, whether that includes perimacular folds. In my opinion, it is only a matter of time. There is ongoing discussion of this topic.

An animal model to study retinal hemorrhages in nonimpact brain injury. Binenbaum G, Forbes BJ, Reghupathi R, Judkins A, Rorke L, Margulies SS. Journal of the American Association for Pediatric Ophthalmology and Strabismus (JAAPOS) 2007 Feb; 11(1): 84-85. They gave 16 infant piglets one good hard inertial rotation at <15ms. Peak angular velocity averaged 191 radians per second and ranged up to 250. In the higher velocity piglets, they found extensive brain injury including SAH, SDH, and white matter tears. No RH and only one minimal ONSH.

Natural animal shaking: a model for inflicted neurotrauma in children? Brown S, Levin AV, Ramsay D, Serbanescu I. JAAPOS 2007 Feb; 11(1): 85. Autopsied three small animals that were shaken to death by a medium sized dog. Found no RH.

Terson's syndrome (see also under "Histology")

Comment by JKR: Judging by the titles of Terson's two articles, it appears that he was specifically referring to (a) VITREOUS hemorrhage, and (b) SPONTANEOUS intracranial hemorrhage. Dr. Levin agrees with this interpretation. Levin AV, personal communication, May 15, 2000. So does Luthert. See "Why do histology on RH?" (2003), above under "Histology."

De l'hémorragie dans le corps vitre au cours de l'hémorragie cérébrale. Terson, Albert. Clin Ophthalmol 1900; 6: 309-312. A 60 year old man.

Le syndrome de l'hématome du corps vitre et de l'hémorragie intracrânienne spontanée. Terson, Albert. Ann Oculist 1926; 163: 666-673

Optic nerve sheath hemorrhage. The Jackson Memorial Lecture. Walsh FB, Hedges TR, Jr. Am J Ophth 1951; 34: 509-527. See under ONSH. Argues that the mechanism of TS is rapid rise in ICP, not retinal vein compression by ONSH. See letter, Ophth 1992 May; 99(5): 647, below.

Follow-up report with clinical and anatomical notes on 280 patients with SAH. Timberlake WH, Kubik CS. Trans Am Neurol Assn 1952; 77: 26-30. Cited by Kuhn et al., 1998, for the proposition that the incidence of true TS in pts with SAH is 3% to 5%. Referred to also by Khan & Frenkel, 1975, as describing one or more characteristic anatomic distributions of hemorrhages in TS.

Subarachnoid hemorrhage: intraocular symptoms and their pathogenesis. Manschot WA. Am J Ophth 1954; 38: 501-505. According to Hedges (1992,) this article can be read, or misread? as saying that the mechanism of TS is retinal vein compression by ONSH.

Pre-retinal and optic nerve sheath hemorrhage: pathologic and experimental aspects in subarachnoid hemorrhage. Smith DC, Kearns TP, Sayre GP. Trans Am Acad Ophth Otol 1957; 61: 201-211. Plunkett (AJFMP 2001Mar) cites this art for the proposition that RH can be produced experimentally by ligating the central retinal vein or by abruptly increasing ICP. This article is not discussed by Muller & Deck in their discussion of RH mechanisms (1974.)

Ocular signs and prognosis in subdural and subarachnoid bleeding in young children. Hollenhorst RW, Stein HA. Arch Ophth 1958; 60: 187-192. Reported RH in 51% of infants with intracranial hemorrhage. Kuhn et al. (1998) cite this source for the proposition that the incidence of TS in children with ICH is higher (up to 70%) than it is in adults (18 to 41%).

Intracranial arterial aneurysm in childhood. Matson DD, J Neurosurg 1965; 23: 573 Reviewing 2000 cases of SAH, found only three cases of spontaneous intracranial bleeding in children ages 1 – 5 yrs.

Walsh FB and Hoyt WF: Clinical Neuro-Ophthalmology, 3d ed. Baltimore: Williams & Wilkins, 1969. Vol 3, p. 2348. "When preretinal hemorrhages are found in an infant with or without cerebral symptoms, such a finding reliably indicates the presence of intracranial hemorrhage."

Vitreous haemorrhage in SAH -- Terson's syndrome. Report of a case with macular degeneration as a complication. Fahmy JA. Acta Ophth (Scand.) 1972; 50: 137-143.

Symptoms and signs of intracranial aneurysms with particular reference to retinal haemorrhage. Fahmy JA. Acta Ophth (Scand.) 1972; 50: 129-136. Finding that RH is common in SAH. According to Khan & Frenkel (below), Fahmy reports intraocular hemorrhage in 50 of 154 patients with SAH (32%). 29 cases were unilateral. 9 had VH (6%). As Khan & Frenkel state, "The severity of the hemorrhage ranged from streak or flame-shaped papillary, peripapillary, and peripheral hemorrhages to large preretinal or vitreous blood accumulation, in a distribution similar to that reported by Timberlake and Kubik."

Fundus haemorrhages in ruptured intracranial aneurysms I. Material, frequency, and morphology. Fahmy JA. Acta Ophth (Scand.) 1973; 51: 289-298. True TS (vitreous hemorrhage) is much less common (3% to 5%).

Intraocular and optic nerve sheath hemorrhage in cases of sudden intracranial hypertension. Muller PJ and Deck JHN. J Neurosurg 1974 Aug; 41(2): 160-166. (have) Department of Pathology, Toronto Western Hospital. This autopsy study of the eyes of 23 patients dying with sudden intracranial hypertension and 12 control patients. The authors do not state what caused the sudden intracranial hypertension in any of their decedents; it appears from the introduction, which discusses SAH, that these were adults with SAH. RH was not an inclusion criterion; 37% had RH and 63% did not. The purpose of the study was to address the controversy about the pathogenesis of intraocular and optic nerve sheath hemorrhages in ICP cases (which the authors use as synonymous with SAH.) (1) How are the effects of intracranial hypertension transmitted to the orbit? -- by the veins or the optic nerve sheath? (2) What is the origin of ONSH? Is it intracranial blood tracking through the optic canal, or is it caused by local rupture of vessels within the optic nerve sheath? Results: of 46 eyes from ICP cases, 87% had ONSH and 37% had RH or VH. Most of the RH were located close to the disc, but some were peripheral. (Fig. 1 shows a large dome-shaped preretinal hem close to the disc and scattered dot hemorrhages in the posterior area.) Their sections of the optic nerve included the part within the optic canal. They found that ONSH was sometimes multifocal and often included intradural hemorrhage. To this finding they attribute significance. They found that the hem was denser in the subdural space than in the subarachnoid space. It was larger in the immediate retrobulbar area. They concluded that ONSH is of local, not transmitted, origin because (a) "intraocular and optic nerve sheath hemorrhages have been reported in cases of sudden intracranial htn in which there has been no intracranial bleeding (citing Weaver & Davis, 1961, and one eye from their material in which there was cerebral swelling without intracranial hemorrhage); (b) the subdural component was greater than the

subarachnoid component in their optic nerve sheaths, implying to them (given that their patients had SAH and not SDH) that this had to be local breakthrough bleeding within the sheath. “We therefore propose that ONSH originates from the rupture of intradural vessels and bridging vessels running from the dura to the pia mater of the optic nerve. The dilatation of the optic nerve sheath suggests this phenomenon, and the demonstration of perivascular intradural hemorrhages that communicate with the subdural space of the optic nerve sheath confirms it.” In other words, distension of the sheath spaces ruptures vessels within the sheath. They go on to argue further that intraocular hems result from obstruction of the central retinal vein within the orbit. They state that there are extensive venous anastomoses in the posterior orbit (giving a diagram,) and therefore, “for retinal venous htn and subsequent [intraocular] hemorrhage to occur, the central retinal vein must be obstructed before it communicates with anastomotic channels which could re-route the retinal venous drainage.” In fact, it must occur proximal to or at the anastomosis of the retinal vein with the choroid, since the choroid communicates with the ciliary veins. Therefore, the retinal vein obstruction producing RH must occur very close to or within the opticocylar junction. They believe they showed that such compression does occur via the effusion of clear or bloody CSF through the optic canal into the ONSH and down to the retrobulbar location, compressing the retinochoroidal anastomosis that lies just anterior to the subarachnoid cul-de-sac. Noting the work of Hayreh (1964) which failed to produce RH in monkeys by ligating the retinal vein back where it comes out of the posterior optic nerve, and the work of Fujino et al. (1969) which *did* produce RH by obstructing the retinochoroidal anastomosis. They further adduce some experimental work they did in cadavers in which they raised the ICP to 250 mmHg while producing an artificial carotid-cavernous sinus fistula and found the cavernous sinus flow was maintained, showing that anastomotic venous drainage from the orbit and hence from the optic nerve and retina will be maintained regardless of ICP. Schultz (1992) states that this work “demonstrated retrobulbar optic nerve sheath distension in 46 eyes from cases of sudden intracranial hypertension,” supporting a theory that RH is caused by ICP being transmitted down the optic nerve sheath via effusion of CSF or blood in the sheath, compressing the central retinal vein. (As opposed to direct venous hypertension transmitted through the vein itself from the cranial cavity.) This theory would require that optic nerve sheath *distension* (not necessarily *hemorrhage*) be present in all cases of RH. These guys do not mention the article by

Intravitreal hemorrhage associated with rapid increase in intracranial pressure (Terson’s syndrome). Khan SG, Frenkel M. Am J Ophth 1975; 80: 37-43. (have) Distinguishes frank intravitreal hemorrhage from the more common retinal, preretinal, and subhyaloid hemorrhages. TS is frank intravitreal hemorrhage in association with bleeding in the subarachnoid space. Presents four patients with fundus photographs. All were adults. Patient 4 was a state prisoner who was assaulted, strangled, and hit over the head with unconsciousness. He had left to right shift, but bilateral burr holes failed to reveal any subdural blood. In this patient, fundus photographs show papilledema, radiating peripapillary hemorrhages, and intravitreal debris. “We thought the vitreous hemorrhages were caused by rapid increase in intracranial pressure due to trauma, with strangulation or thoracic compression acutely increasing the intravenous pressure.” Compares a criminal assault-strangulation case presented by Walsh & Hoyt (1969) as being very similar. Suggests the same ICP mechanism for birth hemorrhages. Notes that vitreous hemorrhage is often delayed by hours or days. “The internal limiting membrane of the retina is apparently able to contain most preretinal hemorrhages, and only occasionally does the blood pass forward into the vitreous cavity, resulting in Terson’s syndrome.” Discusses Fahmy’s morphologic findings (1972) and compares them with Timberlake & Kubik’s (1962.) Apparently these earlier researchers found both radiating peripapillary hemorrhages and larger preretinal hemorrhages. Comments on the mechanism as being controversial. Discusses the direct-extension theory at some length and rejects it. “Hemorrhages that lie so far from each other can only be explained on the basis of a suddenly increased intracranial pressure. This pressure, transmitted rapidly to the optic nerve sheaths, produces a rapid venous stasis.”* Distinguishes the case of hydrocephalus, which does not cause RH because, they theorize, the venous channels have time to dilate and compensate for increased ICP and venous pressure. Quotes Castren (1963) as saying the same thing. Quotes Miller and Cuttino (1948) as proposing compression of the choroidal anastomotic channels.

* This mechanism is disputed on the ground that the venous anastomoses with the facial and pterygoid systems preclude much of an increase in intraocular venous pressure from an intracranial source. The vein compression would have to occur distal to these anastomoses. The alternative theory is distension of the optic nerve sheath by CSF or blood forced into it from the cranial cavity. See the debate between Hedges and Schultz in Ophth 1992 May; 99(5): 647.

Terson's syndrome: clinicopathological correlations. Weingeist TA, Goldman EJ, Folk JC, Packer AJ, Ossoinig KC. Ophth 1986 Nov; 93(11): 1435-1442. "At the turn of the century, Terson reported the occurrence of vitreous hemorrhage after spontaneous subarachnoid hemorrhage. Today, the syndrome is ascribed to patients with vitreous hemorrhage from all forms of intracranial bleeding. Before 1962, Terson's syndrome was considered relatively uncommon, since only 15 cases were recorded in the world literature. In the interim, numerous reports have been published..." (citing eighteen articles, three of which (1974, 1975, 1979) refer to rapid increase of intracranial pressure rather than specifically to intracranial hemorrhage. Nearly all of them refer to "vitreous hemorrhage," "vitrectomy," or "intraocular hemorrhage.") They note that extracranial hemorrhage (e.g. orbital hemorrhage or scleral hemorrhage) can occur with severe SAH. As far as intraocular hemorrhage, it can occur "in the subretinal space, within the sensory retina, and in the vitreous cavity." "The blood may be located in one or more of the following anatomical sites: (1) beneath the retina (subretinal;) (2) within the retina (intraretinal;) (3) between the internal limiting membrane of the retina and posterior vitreous face (subhyaloid;) or (4) within the vitreous cavity (intravitreal.) When blood escapes into the vitreous cavity, the condition is referred to as Terson's syndrome." The authors specifically state (p. 1441) that subinternal limiting membrane hemorrhage is *not* subhyaloid hemorrhage. [For another description of the same terminology, see Levin, *supra*.]

The authors note that previously reported findings in the posterior pole of eyes with TS have included "retinal and preretinal hemorrhage, ... and preretinal membrane formation." But not previously reported is these authors' finding, in the posterior pole (in fact, in the macula) of eight adult patients with chronic SAH-related vitreous hemorrhage, of "**a peculiar dome-shaped membrane** ... overlying the posterior pole." Plastic-embedded histology and TEM showed that in the chronic cases this dome-shaped membrane was "a complex epiretinal membrane" consisting of basement membrane material with glial cells. But the authors feel they have established that this dome-shaped membrane is NOT the internal limiting membrane of the retina. They feel they have established this by doing light and electron microscopy on a comparable dome-shaped preretinal hemorrhage (my term, not theirs) found in the posterior pole of their only acute case, a patient who died acutely with "subhyaloid and vitreous hemorrhage" related to spontaneous "intracranial hemorrhage" (NOS) presenting acutely with severe frontal headache and with shifting of the sulcus seen on CT. (From this brief description it is in my opinion permissible to assume that the hemorrhage in this patient was probably an *intracerebral* hemorrhage, possibly with associated SAH.) In this enucleated right eye, they use a PAS stain to demonstrate the presence of what they believe to be the internal limiting membrane UNDER the posterior pole hemorrhage (figs. 7 and 8.) The low- and high-power paraffin sections show a dome-shaped preretinal hemorrhage on top of the retina. The low-power paraffin section of this area shows a thin line ABOVE the hemorrhage which could be thought to be the (elevated) internal limiting membrane. But TEM of this formation (fig. 10) shows that it is merely a compressed collection of fragments of collagen fibers "typical of those that exist within the vitreous." So what we have is a true subhyaloid hemorrhage, just as they describe. The implication is that if this lesion had become chronic, as in the other cases, an epiretinal glial membrane similar to the others would have eventually formed over the hematoma, between it and the vitreous humor, including deposition of basement membrane material laid down on the scaffolding of those compressed collagen fibers at the back surface of the vitreous humor.

[Of note, in this acute enucleated eye there was no ONSH. The chronic cases were living vitrectomy patients, so no ONSH could be looked for. Also note that in this article, the authors do not describe or demonstrate any retinal fold. That will have to wait for Keithahn et al., 1993.]

What is the significance of these findings? Primarily, it implies that these chronic epiretinal membranes can safely be excised without doing any damage to the retina, since they are not the internal limiting membrane. Their excision will leave the internal limiting membrane perfectly intact. And it may help visual recovery, because over time these membranes may opacify. They note that "Most patients who survive TS have good macular function and no evidence of intraretinal hemorrhage." (But compare the case presented by Friedman & Margo, 1997, *infra*.)

The authors give a discussion of what they believe to be the pathogenesis of TS. (Although in my judgment their discovery of an epiretinal membrane does not in itself really shed any light on this pathogenesis.) "TS is usually associated with a precipitous intracranial hemorrhage. Several mechanisms have been proposed to explain why hemorrhage occurs in the retina and vitreous cavity. The most likely hypotheses is that blood or [pressure] transmitted through the CSF travels anteriorly within the intervaginal space of the orbital portion of the optic nerve and obstructs venous drainage from the eye. [Noting that in

their one enucleated case, no ONSH or intraneural hemorrhage extension was found.] This observation supports the hypothesis that blood does not have to extend directly from the intracranial cavity to the globe, but instead may interfere with venous outflow, perhaps at the point where the central retinal vein exits the optic nerve sheath. The abrupt rise in venous pressure ruptures intraretinal vessels and the hemorrhage spreads into adjacent tissues. (naming the spaces, as above). When blood escapes into the vitreous cavity, the condition is referred to as Terson's syndrome." In light of this discussion, the only finding that they authors adduce that sheds light on the possible pathogenesis of TS or RH is their finding of a lack of ONSH or ONH in their case 8, the enucleated acute case. And that's just one eyeball. Hence they do not press the point on the pathogenesis being secondary retinal venous hypertension, but only suggest it. Other scholars have taken up the cudgels more forcefully for and against this theory versus the major competing theory in the child abuse context, vitreoretinal traction. (Smith, 1957; Greenwald, 1986; Massicotte, 1991; Levin, 1998; Plunkett, 2001.) Having read Sebag's work and Massicotte's, I am convinced that Weingeist is wrong. The basement membrane material he studied is unquestionably internal limiting membrane, not a neomembrane. JKR.

Features of intracranial aneurysms in infants and report of a case. Crisostomo EA, Leaton E, Rosenblum EL. Dev Med Child Neurol 1986; 28: 68-72.

Spontaneous subhyaloid and retinal haemorrhages in an infant. McLellan NJ, Prasad R, Punt J. Arch Dis Child 1986; 61: 1130-1132. MCA aneurysm causing intracerebral hemorrhage and RH in a 6 week old term female admitted for possible abuse. Developed seizures at home, sz in ER, full fontanelle, "extensive bilateral RH and a large R subhyaloid hem" on adm. (no fundus photo given) No history or signs of direct injury or neglect. Coags & plts normal, skel surv nl, LP bloody w xanthochromic supernatant. Initially dx'd as probable SBS. CT showed a large localized intracerebral hematoma (fig. 1). Cerebral angiography disclosed an MCA aneurysm. Comments in the abstract that "These findings were not the result of shaking." Cited by Luthert in "Why do histology on RH?" (2003) to the effect that "[A]lthough Terson's syndrome was originally defined in terms of vitreous haemorrhage associated with intracranial subarachnoid bleeding, it is well recognized that massive intracranial haemorrhage can lead to appearances remarkably like those seen in cases of alleged shaking." See also Hupp et al., 1984. Medele et al., 1998 for reports of RH caused by raised ICP and by optic nerve sheath trauma causing compression of the retinal vessels.

Terson's syndrome: significance of ocular findings. Toosi SH and Malton M. Ann Ophth 1987; 19: 7-11

Intracranial hemorrhage and non-accidental injury. Newton RW. Arch Dis Child 1989; 64: 188-190. Review of 10,000 cases of intracranial hemorrhage in children, found no cases of spontaneous SAH in children younger than 1 year.

Long-term visual outcome in Terson syndrome. Schultz PN, Sobol WM, Weingeist TA. Ophth 1991 Dec; 98(12): 1814-1819. (have) A retrospective study of all eyes dx'd as TS at the Univ of Iowa between 1980 and 1990. Presents 30 eyes from 19 patients. "The syndrome of vitreous hemorrhage in association with any form of intracranial or subarachnoid hemorrhage has come to be known as *Terson syndrome*." (italics in original.) 15 pts had spontaneous SAH. 4 pts had traumatic SDH; they were age 18 to 35. Two of the four trauma patients had the dome-shaped posterior blood collection. In the introduction they refer to their earlier finding (1986), stating that "Preretinal hemorrhages often occur within the temporal vascular arcades where they may produce a **peculiar dome-shaped accumulation of blood**" (giving an ophthalmoscopic fundus photograph) and reiterating that the blood was sequestered between the internal limiting membrane and the posterior hyaloid face, and that the posterior hyaloid face was then replaced by an epiretinal membrane. Say that vitreous hemorrhage "may occur from rupture of these dome-shaped lesions but is also seen in their absence." In this long term followup study of nineteen consecutive patients aged 9 to 60 with TS, they describe relatively favorable visual outcome.

Mechanism of Terson syndrome. (letter) Hedges TR Jr. Ophth 1992 May; 99(5): 647, commenting on Schultz et al., above. (have) Hedges says that Schultz's description of the pathogenesis of TS is incorrect. His work in 1951 and 1955 (above) showed that rapid rise of ICP transmitted through the venous channels into the orbital veins causes TS. It is the rapidity and magnitude of the rise in ICP that determines the

degree and extent of the ocular hemorrh in TS. It is NOT transmission of pressure down the sheath space “causing venous stasis via compression of the intraorbital veins” as stated in Schultz’s article; this is a misconception from Manschot’s article (1954.) Schultz replies that because of intraorbital venous anastomoses of the ophthalmic vein with the facial and pterygoid venous systems, intracranial pressure is unlikely to raise *retinal* venous pressure enough to cause RH. Hayreh (1971) raised the ICP up to 50 mm Hg in monkeys and achieved only a 7 mm Hg pressure rise in the ophthalmic vein. Therefore, Schultz continues to propose that causing RH would require occlusion of the retinal vein distal to the orbital anastomoses. This could come from CSF or blood forced into the optic nerve sheath from the cranial cavity. This is the theory of Muller & Deck (1974.)

Terson’s syndrome: a reversible cause of blindness following subarachnoid hemorrhage. Garfinkle AM, Danys IR, Nicolle DA, Colohan AR, Brem S. J Neuros 1992 May; 76(5): 766-771. (have) TS refers to vitreous hemorrhage with SAH, usually due to a ruptured cerebral aneurysm. Although it is familiar in the ophth lit, it has been only rarely commented on in the neurosurgical lit. Present a prospective series of 22 consecutive pts with SAH, in 8 of whom VH developed, either at presentation (6) or within 12 days (2,) and in these two RH was already present. The initial amount of intraocular hem did not correlate with the severity of SAH. Vitrectomy gave good visual outcomes in the survivors.

Retinal folds in terson syndrome. Keithahn MAZ, Bennett SR, Cameron D, Mieler WF. Ophth 1993 Aug; 100(8): 1187-1190. Background: Perimacular folds have been reported in the shaken baby syndrome, but have not been described in adults with Terson syndrome. Presents two adults with Terson’s syndrome, defined as “the presence of vitreous hemorrhage with any form of intracranial hemorrhage.” Case 1 a 19 year old man with a traumatic SDH from an MVA, with dense vitreous hemorrhage noted in the L eye. Four months post injury he underwent vitrectomy for severely diminished VA on the left (the VH failed to clear.) At operation, it was found that the posterior hyaloid was firmly adherent to the retina. When the hyaloid was surgically peeled away, there was a membrane spanning a fold of retina encircling the macula (a perimacular fold.) Case 2 a 26 year old man with a GSW to the L forehead, requiring frontal lobe debridement, fol by a postoperative ICH. Two months post-injury he was found to have VH on the right, and underwent vitrectomy at 3 months. When the posterior hyaloid was stripped away, a retinal fold 360 degrees surrounding the macula was noted. A thin, glistening membrane was found spanning the macula and attached to the apices of the fold. A small amount of hemorrhage was located inferiorly between the membrane and the retina. Did EM on this membrane, showing basement membrane material with hemosiderin macrophages. My comments: (1) This is clearly the same epiretinal membrane that was found by Weingeist et al. in their chronic cases of TS in 1986, although it does not comment specifically on the presence of collagen fibers typical of the vitreous. (2) What are the implications of case 2 for the theory of Massicotte et al. (1991) to the effect that this patter of attachment of the membrane to the apices of the perimacular fold signifies vitreoretinal traction? This art is commented on in Ophth 1994 Jan; 101(1): 1.

Spontaneous and traumatic vitreous hemorrhage. Dana M, Werner M, Viana M, et al. Ophth 1993; 100: 1377-1383. Cited by Levin in Reece, 2d ed., 2001, for the proposition that nontraumatic causes of vitreous hemorrhage in infants and young children are quite uncommon.

RH associated with spinal cord AVM. Clark RSB, Orr RA, Atkinson CS, Towbin RB, Pang D. Clinical Pediatrics 1995 May; 34: 281-283. (Pittsburgh) (have) 4 year old boy presented with acute LOC and sz. with onset during sleep. Eye exam showed papilledema and bilateral RH. CT of the head showed SAH in the interpeduncular cistern and periventricular cistern and a blood clot in the fourth ventricle with mild enlargement of the cerebral ventricles. Found by MRI to have a ruptured AVM of the thoracolumbar spinal cord at T9 to L2. This patient survived; there is no comment on ONSH. A fundus photograph of the left eye shows a single dot-blot hemorrhage and a small preretinal hemorrhage right over a vessel of the temporal arcade in the posterior pole. Discussion: “Intraocular hemorrhages are recognized complications of sudden increases in intracranial pressure, thought to be caused by ‘transmission of ICP into the ONSH through the subarachnoid communication in the optic canal.’ (quoting Muller & Deck, 1974.) A possible mechanism to explain the constellation of clinical findings in our patient is that rupture of his avm produced an acute SAH that extended into the basal cisterns and ventricles. The combined effect of the bleeding and secondary acute hydro causes a sudden rise in ICP that was subsequently transmitted through

the optic sheath into the venous drainage of the retina, precipitating RH.” The authors do not mention or discuss Terson’s syndrome, possibly because there was no vitreous hemorrhage. Compare Weissgold et al. (1995, below) for a case with ONSH but no RH.

Ruptured vascular malformation masquerading as battered/ shaken baby syndrome: a nearly tragic mistake. Weissgold DJ, Budenz DL, Hood I, Rorke LB. (Penn). Surv Ophthalm 1995 May-Jun; 39(6): 509-512. (have) See under “ONSH.” This seven week old infant with an AVM had ONSH but no RH. Accordingly, he is not TS. He did have ONSH, and that led to suspicion of child abuse until the AVM was discovered histologically.

Terson’s syndrome in spontaneous subarachnoid hemorrhage: a prospective study in 60 consecutive patients. Pfausler B, Belcl R, Metzler R, Mohsenipour I, Schmutzhard E. (Innsbruck). J Neurosurg 1996 Sep; 85(3): 392-394. Prospective study of 60 consecutive pts with spontaneous SAH. Ten of them (16%) had TS. Case fatality: with TS -- 90%. Without TS -- 10%. TS is common and unfavorable, may herald a rebleed.

Bilateral subinternal limiting membrane hemorrhage with Terson syndrome. Friedman SM, Margo CE. Am J Ophthalm 1997 Dec; 124(6): 850-851. (have) Reports bilateral **dome-shaped** posterior-pole hems in a pt with TS, examined histologically. A 53 year old woman seen one month after spontaneous SAH from an aneurysm. Both eyes had dense vitreous hemorrhage. And partial posterior vitreous detachment with an elevated retinal mass in the posterior pole of both eyes. Underwent bilateral vitrectomy. After removal of the VH and the posterior hyaloid, a dome-shaped hemorrhage was visible in the posterior pole. (Note, after removal of the hyaloid.) The tissue overlying the hemorrhage was extracted and examined histologically, found to be a PAS-positive membrane surrounded by hemosiderin macrophages (photomicrograph.) Both maculae showed a demarcation line corresponding to the edge of the hemorrhagic detachment of the internal limiting membrane. “**Most clinical reports** of patients with Terson syndrome describe dome-shaped retinal hemorrhages. There has been confusion in the literature concerning the anatomic localization of these hemorrhages.” Conclude that “Subinternal limiting membrane hemorrhage in pts with TS may be more common than has been previously reported. We postulate that initial hem in TS occurs **under the internal limiting membrane**. These hemorr may break into the subhyaloid space, resulting in vitreous hemorrhages and secondary epiretinal membranes.” Compare the similar findings of Keithahn (1993). [The reason these authors phrase their conclusion cautiously as “more common than has been previously reported” is that they are aware of the purported finding of Weingeist et al (1986) that these epiretinal membranes are not internal limiting membrane but a subhyaloid neomembrane. –JKR]

Hemorrhagic macular cysts in Terson’s syndrome and its implications for macular surgery. Morris R, Kuhn F, Witherspoon CD, Mester V, Dooner J. Dev Ophthalm 1997; 29: 44-54. These are **sub-internal limiting membrane** cysts. See the later work by the same group (2000) extending this finding.

Intracranial aneurysms in infants and children. Allison JW, Davis PC, Sato Y, James CA, Haque SS, Angtuaco EJ, Glasier CM. (Little Rock) Pediatr Radiol 1998 Apr; 28: 223-229. Retrospective chart review of 21 children with aneurysms imaged over a 20-year period. 18 saccular, 6 mycotic, 1 traumatic. 44% posterior circulation, nine aneurysms arose from distal arterial branches. 16% were giant (over 2.5 cm), 40% were “large” (1 cm – 2.5 cm). Compared to adults, noted posterior location, large size, peripheral distribution.

Terson’s syndrome in subarachnoid hemorrhage and severe brain injury accompanied by acutely raised intracranial pressure. Medele RJ, Stummer W, Mueller AJ, Steiger HJ, Reulen HJ (Munich). J Neurosurg 1998 May; 88(5): 851-854. (have) Retinal OR vitreous hemorrhage in association with SAH. Is it caused by ICP? Prospective ophthalmologic study with indirect ophthalmoscopy of 22 pts: 13 with spontaneous SAH and 9 with TBI. All pts had elevated ICP on presentation. (>20 mm Hg).

SAH	6/13	(46%)	TS
TBI	4/9	(44%)	TS

Conclusion: TS may be related to acute elevation of ICP, independent of its causes, and may occur with similar incidence in pts with trauma and spontaneous.

“This phenomenon [bleeding in posterior eye compartments] has only rarely been described in association with subdural and epidural hematomas or traumatic SAH.” (citing Espinasse-Berrod (Fr.), 1980; LeRebeller (Fr.), 1980; Shaw, 1977; Toosi, 1987; Vanderlinden, 1974.) Notes that it is possible that vision impairment in some or many trauma patients may be due to primary brain and/or optic nerve injury (injury to afferent pathways.) But notes that many pts have recovered vision after vitrectomy, and the literature on SAH/TS does not suggest injury to afferent pathways.

“Initial attempts at explaining TS suggested that, in SAH, blood traverses the subarachnoid space into its continuation within the optic nerve sheath. It was thought that blood penetrates the sclera in the porous region where the optic nerve enters the globe, and finally appears in the vitreous space within the eye. (citing Doubler & Marlow, 1917.) Many textbooks still attribute TS to this mechanism, despite evidence that there is no connection between the optic nerve sheath subarachnoid space and the vitreous body. (citing Anderson, Ultrastructure of the optic nerve head, 1970, 1980). Castren (1963) suggested that rapid increases in ICP result in TS, which is caused by venous congestion due to impairment of venous drainage to the cavernous sinus. (citing Fujimoto, 1979; Espinasse-Berrod, 1980; Hedges (letter), 1992.) Retinochoroidal connections and the central retinal vein could be compressed by pressure-induced dilation of the optic nerve sheath subarachnoid space. (citing Muller & Deck, 1974; Toosi, 1987.) Increased venous pressure results in stasis followed by vessel rupture and intraocular hemorrhage. Our own observations support the concept that ICP represents the principal impetus for the development of intraocular hemorrhage because intraocular hemorrhage was associated with higher initial levels of ICP. However, our measurements can only be taken as an indicator of the actual intracranial pressure present when intraocular hemorrhage developed.” Goes on to note that the initial phase of SAH causes dramatic increase in ICP. The same pattern can be observed in the initial phase of trauma caused by hypercarbia in the unconscious patient (citing Moskala (Pol.), 1995.) “Furthermore, posttraumatic TS may result from plateau waves, which are well described in the early posttraumatic phase. Other patterns of intracranial hypertension without extreme ICP peaks, which are observed, for instance, in hydrocephalus, cavernous sinus thrombosis, or carotid cavernous sinus fistula, do not result in TS. A typical opth finding in such pts is papilledema but not intraocular hemorrhage.”

Terson syndrome: results of vitrectomy and the significance of vitreous hemorrhage in patients with subarachnoid hemorrhage. Kuhn F, Morris R, Witherspoon CDS, Mester V. Ophth 1998 Mar; 105(3): 472-477. TS defined as VH with either SAH or SDH. Distinguished from mere RH without VH. Study A retrospectively reviewed 4 children and 23 adults, aged 6 months to 69 years, who underwent vitrectomy for dense VH. Three of the four children were under 1 year. All four of the children suffered traumatic SDH and had permanent brain damage. Accordingly, none of the children responded to vitrectomy; their vision remained in the finger-counting range, either because of amblyopia or brain damage, the authors think more likely the latter. In their reply to Monte Mills’ criticisms the authors state that these infants were “savagely hit.” (See also the commentary by Dr. Levin summarized below.) Study B was a prospective clinical series of 100 pts with SAH due to ruptured cerebral aneurysms. Study A frequently found epiretinal membranes over **hemorrhagic macular cysts** (in 13/33 eyes.) They consider it established that the epiretinal membranes they removed in these eyes were internal limiting membrane; these membranes were removed at the time of vitrectomy. They did light microscopy, EM, and immunoh. Therefore these are “**submembranous hemorrhagic macular cysts.**” In study B, they found that various types of intraocular hemorrhage were present in 17/200 eyes. 8 of these were vitreous hemorrhage (TS.) TS was more common in pts with coma. In adults, some form of intraocular hemorrhage occurs in 18% to 41% of pts; in children up to 70% (citing Hollenhorst & Stein, 1958,) although true TS (i.e. VH) is much less -- 3% to 5% in adults (citing Timberlake, Kubik, 1952 and Morris, Kuhn, 1997, above.) “Our prospective study found a lower incidence of intraocular hemorrhage (17%), but an approximately double than previously reported rate of eyes with Terson syndrome (8%).” TS was a predictor of mortality: 54% with TS vs. 20% without, and it correlated with the overall severity of the patient’s condition.

Epimacular membranes or epiretinal membranes are known to form in TS. (citing Garfinkle, 1992 and Garcia-Arumi, 1994.) This is significant because it adversely affects the visual outcome. **Hemorrhagic macular cysts** also occur; the authors found these in 40%.

Monte Mills, MD, of Madison, writes a letter to the editor criticizing this article (Ophth 1998 Dec; 105(12): 2161-2162,) in which he directs himself to SBS, and opines that “[I]t is not appropriate to use the term Terson syndrome for children with SBS,” because “[T]he *mechanisms* of intraocular injury,

associated neurologic and retinal injuries, and *prognosis* in infants with SBS are different from older patients with classically described Terson syndrome.” Specifically, he argues as follows:

Mechanisms: Arguing for a different mechanism of SBS RH from TS RH, he adduces (1) his view that the hemorrhages in SBS arise distant from the papilla rather than overlying the peripapillary vascular arcades as in TS: “The preretinal and vitreous hemorrhage does not appear to arise solely from the peripapillary region,” as in TS, but rather “includes scattered RH throughout the posterior pole as well as the more anterior retina, frequently extending to the ora serrata.” (2) the absence of papilledema or raised disc margins acutely in SBS, (3) there is ONSH in SBS, and it has not been documented in TS. (This is probably a specious argument, bc until very recently it has not been technically possible to image ONSH antemortem and there are very few autopsy cases of TS. –JKR) (4) SBS patients probably don’t have the acute rise in ICP that SAH pts have, because in SBS pts, “Intracranial hemorrhage may be minimal and is frequently less prominent than the associated traumatic neuronal injury.” And “In these infants with open fontanelles, it is unlikely that small amounts of hemorrhage could cause a sufficiently rapid increase in intracranial pressure to lead to intraocular hemorrhage by the mechanism suggested in classical TS.” (5) Vitreoretinal traction may be the mechanism of RH in SBS, because of “the correlation between the severity of intraocular and intracranial injury” (citing Wilkinson et al., 1989; Green et al., 1996; Matthews & Das, 1996; and Mills, 1998), and because histopathologic evidence (citing Massicotte et al., 1991) suggests inertial shearing within the retina and at areas of vitreoretinal attachment.

Associated brain injuries: (1) “The correlation between the severity of intraocular and intracranial injury,” including the correlation of (a) the extent of RH (citing Wilkinson et al., 1989), (b) the incidence of perimacular folds (citing Mills, 1998, in which he correlates perimacular folds with mortality), and (c) the presence or extent of VH (citing Matthews & Das, 1996) with the severity of neurologic injury including impairment of the posterior visual pathways in most cases of SBS.

Prognosis: The prognosis of VH in SBS patients is markedly different than TS patients because of (a) impairment of the posterior visual pathways, (b) the formation of perimacular folds (retinal injury).

The authors reply as follows: (1) They identify the original authors who added SDH to the definition of TS. (Shaw & Landers, 1975; Gutierrez-Diaz, 1979; Clarkson et al., 1980,) and state that since the “clinical implications” of VH from either SDH or SAH are “very similar,” the definition of the syndrome should include both types of patients, even if the underlying mechanism is possibly different. (2) They distinguish shaken infants from savagely hit infants, and argue that the RH literature of pure shaking cases may not apply to savagely hit infants. The literature of savagely hit infants shows retinal findings **identical, they say, to their adult TS cases**, including hemorrhagic macular cysts. And they find no difference between the ocular findings in adult head-trauma TS patients, adult SAH-TS patients, and savagely hit infant TS patients, suggesting to them that the SBS population is a special and distinguishable group.

Dr. Levin comments on this article at p. 175-176 of his review article (2000a). He says, in the context of the similarity of Terson’s cysts to retinoschisis cysts, that “In one large study of pts with Terson syndrome, there were only 5 children, all of whom were less than 1-year-old, who had subdural haemorrhage and ‘haemorrhagic macular cysts’ which by description (no photographs provided) sound very much like the lesion characteristic of traumatic retinoschisis. Although only one child is cited as a victim of child abuse in the paper, my personal communication with the first author reveals that all five children were suspected victims of abuse (Dr. F Kuhn, personal communication.) Whether or not these children were shaken in addition to sustaining impact abusive head injury is unclear... Dr Kuhn and co-workers believe that the presence of intracranial blood, more than a history of trauma, is the common denominator in the formation of these haemorrhagic retinal cysts, (citing Morris, Kuhn and Witherspoon, Hemorrhagic macular cysts. Ophth 1994.) yet such lesions have not been described in ch other than those who are abused. In a prior publication by the same group, presumably of the same pts, one photograph is offered which shows a large bulbous mushroom-shaped subinternal limiting membrane macular haemorrhage which does not look like the TR of SBS as it lacks a demarcation fold and is larger than any lesion I have ever seen in SBS.... If one considers just the adults in the early paper group, only 7 of 28 eyes had subinternal limiting membrane bleeding demonstrating that this is an uncommon finding in Terson syndrome. Other authors have confirmed this conclusion (citing Weingeist, 1986), yet controversy remains.” Levin goes on (p. 176-177) to discuss other reasons to believe that TR is different than the premacular haemorrhages seen in adults with TS:

1. The adult TS lesions may be associated with preretinal membrane formation (citing Kuhn), "the contraction of which may be the cause of internal l m stripping with blood accumulation. Citing Keithahn, 1993 for the finding that "In one study of two adults with subILM blood and retinal folds, the ILM or cicatricial membrane was described as taught [sic] between the folds.
2. Weingeist's finding that the TS hemorrhage is preretinal, not intraretinal. "This is distinctly different than the blooning forward of the ilm in TR." TR "has not been observed to be assoc w cicatrix or ilm contraction."
3. Optic nerve atrophy, a not-uncommon finding in SBS, is not usually present [in TS.] (citing Kuhn.)
4. Pigmentary changes at the edges of premacular haemorrhage in TS, which resemble the subretinal pigmentary alterations seen at the edges of TR lesions, are probably due to breakdown products of blood rather than the shearing trauma to the retina seen in SBS." (citing Weingeist.)

See also the comments of Tongue, 1991.

Terson syndrome: CT evaluation in 12 patients. Swallow CE et al. Am J Neuroradiol 1998 Apr; 19(4): 743-747. Hyperdense retinal crescents or nodularity on CT in the setting of SAH suggests the need for ophthalmoscopic examination -- probable RH. Subtle finding on CT. Note that they don't refer to any ONSH.

[Terson syndrome] Ophth 1998 Mar; 105(3): 472-477. Also letter, Ophth 1998 Dec; 105(12): 2161-2163.

Surgical approach in Terson syndrome: vitreous and retinal findings. Augsten R, Konigsdorffer E, Strobel J. (Jena). Eur J Ophth 2000 Oct-Dec; 10(4): 293-296. Advises early vitrectomy. Six eyes from three adult pts.

Macular rings in Terson's syndrome. (letter) Kuhn F, Morris R, Mester V. Acta Ophth Scand 2000 Dec; 78(6): 719-720. (have) Commenting on report by Sadeh et al. on a macular ring in TS, Acta Ophth 1999; 77: 599-600. We have previously shown in a large prospective study (1998) that hemorrhagic macular cysts are present in 39% of TS eyes. In 69% of these cases, the blood accumulation is below the ILM, and in 31 % it is above the ILM. In other words, **submembranous hemorrhagic macular cysts were found in 27% of TS eyes.** And it is more common than preretinal hemorrhagic macular cyst. "We proposed the term 'submembranous macular cyst' for this pathology, as opposed to a preretinal hemorrhagic macular cyst (representing 31% of eyes with a cyst.)" Also, "**Retinal folds** are not uncommon in eyes with submembranous hemorrhagic macular cysts, and are caused by the ILM exerting traction on the retina along the ILM's reinsertion." "The perimacular ring results from late pigment epithelial changes resulting from the traction by the detached/ stretched ILM." This has clinical implications, because removal of the ILM membrane results in visual improvement. Comment by JKR: Dr. Levin (2000a at 176-177) states that his personal communication with Dr. Kuhn reveals that all of the five infant cases in this study were suspected abuse cases, so Dr. Levin's point that this type of cyst is never seen outside abuse cases still stands. But Dr. Levin admits by implication that subinternal limiting membrane bleeding of this type is morphologically similar to TR. He cannot explain why it occurs in 27% of adults with nontraumatic intracranial bleeding. The obvious implication is that if it occurs nontraumatically in adults, it could occur nontraumatically in infants. But it never has, at least as far as Dr. Levin's experience and the published literature shows. But that's probably because spontaneous intracranial hemorrhage in infants is rare, and fatal hemorrhage even rarer.

Levin, 2000a, supra, 164, 175. "Traumatic retinoschisis has never been described in children due to any entity other than SBS so its presence is diagnostic. However, in adults, subhyaloid or sub-internal limiting membrane haemorrhages due to Terson syndrome and other disorders may appear remarkably very similar. Retinal folds (sometimes referred to as paramacular folds) may even be present. But, in these adults ... the primary cause ... is readily apparent and other retina findings characteristic of SBS are often absent."

“The histopathology of the retinal lesions (in Terson syndrome,) especially domed-shaped macular haemorrhages, is quite different.” (no ref cited) Also RH other than typical peripapillary flame hemorrhages due to papilledema are rare other than in SBS, “less than 5%” by his statistics.

Avoiding pitfalls in the diagnosis of subarachnoid hemorrhage. Edlow JA, Caplan LR. N E J Med 2000 Jan; 342(1): 29-36. (have) Saying that SAH is often missed in the ER. Often presents as headache, “the worst headache of my life.” Providing guidelines for primary care physicians. Gives a color fundus photograph of subhyaloid and flame hemorrhages. The flame hemorrhages are right over the temporal vascular arcade. The subhyaloid hemorrhage is large. “These [ocular] hemorrhages may be the only clue to the dx in unconscious patients.” Discusses the limitations of CT scanning and LP. LP should not be performed unless the CT is nondiagnostic; it has caused acute deterioration in patients with SAH. Discusses xanthochromia. Cited by Plunkett (Am J For Med P 2001 Mar) for the proposition that ONSH occurs with AVMs. This article does not say anything about ONSH. It does say that RBC’s disseminate “throughout the subarachnoid space” in aneurysmal SAH.

Ocular manifestations of child abuse. Alex V. Levin, MD. Chapter 5 in: Reece RM and Ludwig S, eds., *Child Abuse: Medical Diagnosis and Management*, 2d ed. Philadelphia: Lipincott Williams & Wilkins, 2001. At 100: “Likewise, intracranial hemorrhage alone, although not uncommonly associated with intraocular hemorrhage in adults (Terson’s syndrome), occurs in fewer than 5% of children with intracranial bleeding.” (citing his own unpublished data.) And note that Dr Levin refers to Weingeist’s 1986 histologic study of chronic hemorrhagic macular cysts in TS, distinguishing these subhyaloid hemorrhages from retinoschisis. To distinguish this lesion of TS from the lesion of TR, Dr Levin adduces the following considerations:

1. TR is intraretinal. Weingeist showed that TS macular cysts are subhyaloid. (But note Kuhn’s conclusion.)
2. RH from spontaneous intracranial hemorrhage in infants is rare (less than 5%).
3. TS hemorrhage occurs over vessels. TR is not localized to vessels.
4. Many SBS do not have increased ICP, at least acutely.

Retinal haemorrhage and fatal stroke in an infant with fibromuscular dysplasia. Currie ADM, Benley CR, Bloom PA. Arch Dis Child (Br.) 2001; 84: 263-264. Eleven month old had “extensive subhyaloid and retinal hemorrhages” with intracerebral and IVH. Alex Levin says (Child Abuse Quarterly for July 2001) that the fundus photograph shows peripapillary intra- and pre-retinal haemorrhages which he would not label as “extensive” because no information is given about whether they extended to the periphery. He wonders whether it was really a SBS, although the CT is not typical for that.

Reece & Ludwig, 2d edition (2001), p. 67: “In reviewing 2,000 cases of SAH, Matson found only three instances of spontaneous intracranial bleeding occurring in children ages 1 through 5 years. Reviewing additional studies from 1966 to 1973, totalling 10,000 cases, Newton found no instances of spontaneous SAH in children younger than 1 year. ... A review of recent case reports yielded 72 cases of spontaneous rupture of cerebral aneurysms in children younger than 5 years, 66 cases in children younger than 2 years, and 20 cases in infants 1 year or younger.”

A ruptured retinal arterial macroaneurysm presenting as subhyaloid haemorrhage. Vaidya AR, Shah NJ. Indian J Ophth 2002 Mar; 50(1): 56-58.

Retinal findings in children with intracranial hemorrhage. Schloff S, Mullaney PB, Armstrong DC, Simantirakis E, Humphreys RP, Myseros JS, Buncic JR, **Levin AV**. Hospital for Sick Children, Toronto. Ophth 2002 Aug; 109(8): 1472-1476. From the abstract: To identify the incidence of Terson’s syndrome in children. Did dilated ophthalmoscopy on 57 consecutive children with ICH due to nonabusive causes. Found: 55 pts had no RH. Two pts had RH. These were: one pt with infectious retinopathy had one dot hemorrhage. One pt from an automobile accident had three flame hems and two deeper dot hems. All non-normal eye exams were repeated by Dr Levin personally. Conclusion: “The maximal incidence of intraretinal hemorrhage in children with nonabuse ICH is 8%.”

Brief review of the literature on Terson's quotes it as thought to be rare until a 1954 review showed a 20% incidence (Manschot, 1954). Other studies next several decades showing more common (Morris, 1967), Khan & Frenkel, 1975, Shaw, 1975). "Today the def has been expanded to include all forms of intracr bl with vitreous or retinal hem or both (Weingeist, 1986, Levin in TJ David, Billotte (fr) 1988, Kuhn, 1998)."

"To our knowledge, there are no studies prospectively evaluating the occ of TS in chil. Two observational case report articles descrirbe RH in eight eyes of six ch w intracr hem (Shaw, 1975, Kuhn, 1998). Although there were no inv reported to rule out nonaccidental trauma in these six chil, child abuse was likely in five (F Kuhn, personal communication, April, 1998). In the sixth child, the cause of the intracr hem remains unk..." "[T]he incidence of TS in children cannot be assessed fully."

Materials: had 57 chil with nonabusive ICH, aged 6 weeks to 16 years. Of these, 17 were 4 years of age or younger. Of the 57 ICH's 27 were due to trauma, 24 to medullobl and things like that, 6 to AVM's and things like that. Of the 27 trauma, they were:

MVA	15
Sport accidents	6
Falls	6
Five story bldg	1
One flt of stairs	1
Eight foot slide	1
Six foot ravine	1
From mother's arms	1
From stroller	1

The intracranial hemorrhages were:

SDH	18 (32%)
EDH	7 (12%)
SAH	7 (12%)
Parenchymal	33 (58%)
IVH	22 (39%)
Skull fracture	13 (22%)
Craniotomy	(24) (42%)

Of the 57 included patients, 55 had normal retinal examinations (96%). The two with positive retinal findings were:

Pt 1 a 7 year-old passenger thrown 100 feet w L SDH, IPH, IVH w midline shift. Right retina had three superficial flame-shaped NFL hems and two deeper intraretinal hems, all less than 500 microns, located along the vaascular arcades. No hem in the macula. The left fundus was normal.

Pt 2 an 8 y o w IgA nephropathy and a sz, CT showed cortical infarcts. Strep sepsis. Got heparin, then devel a thalamic hemorrhage with IVH into all ventricles. Eye exam revealed bilateral small white retinal lesions consistent with infection. One eye had a slingle dot hemorrhage in one of the white lesions.

Discussion: Although incidence of TS in adults is about 16 – 27 percent, our study suggests that the incidence is much lower in children.

"Shaking is a violent act. It is now believed that shaking alone can cause most of the intraretinal and intracranial findings [in SBS]. Death can also occur from shaking alone. SBS is characterized by the triad of skeletal injury, intracranial hemorrhage, retinal hemorrhage, or a combination thereof, usually in the absence of external signs of injury. (citing Levin in TJ David, and Caffey, 1974) Some authors believe

that impact must accompany the shaking to cause the syndrome (citing Duhaime et al, 1987), but this is widely disputed (citing Levin, Hadley, 1989, Alexander, 1990, Green, Br J Ophth 1996). RH are obs in 50% to 100% of shaken babies. (Citing Levin, Ludwig, 1984, Till, 1968). The exact pathophysiologic mechanism of many of these hemorrhages are unknown. Retinoschisis, when present, is the result of shearing forces on the macular retina through its connections with the vitreous, which are particularly strong in children. (citing Levin, and Greenwald, 1986). Purtscher's retinopathy may also play a role in some children, because a rise in intrathoracic pressure would be expected especially when the infant's torso is grasped to such a degree that rib fractures occur. This pressure rise may be transmitted to the eye, causing disruption of venous return. (citing Levin and Tomasi, 1975). The optic nerve sheath theoretically may be affected by intracranial pressure or hemorrhage, as in Terson's syndrome. Repetitive shaking may also induce unique shearing trauma to the orbital contents and retina."

"We are unaware of any studies of TS specifically in children. To understand the pathophysiologic mechanism of this disorder, we must rely on several adult studies with proposed theories (Garfinkle, 1992) [discussion of the optic nerve tracking theory, compression of the central retinal vein by ONSH theory (Riddoch, 1925, Hayreh, 1964), the rapid rise in ICP theory (Walsh & Hedges, 1951, Manschot, 1954). "The most likely theory was proposed in 1954 and states that increased ICP results in excess accum of CSF within the nerve sheath communication of the subarachnoid space (Manschot, 1954, Toosi, 1987). As a result, compression of the retrobulbar portion of the optic nerve can obstruct the retinochoroidal anastomoses and, perhaps to some degree, the central retinal vein as well. This theoretically wd result in a decrease in venous drainage that wd cause stasis and hemorrhage. Because the retinochoroidal anastomoses are located near the optic nerve and scleral junction anterior or 'at the anterior limitation of' the subarachnoid space, the compression wd occur only with alrge amounts of neve sheath dilatation. As a result, only certain patents wd wxperience intraocular hemorrhage (Garfinkle, 1992). Stretching of the optic nerve sheath may also shear bridging vessels that go from the dura to the pia mater (Muller & Deck, 1974). Why TS occurs less frequently in children is unknown. ... Traditionally, studies of adult TS have included AVM as a primary cause: a rare entity in choldhhood,. Although our series had only one pt with an AVM, review of the literature also reveals that RH rarely occur in children with a ruptured aneurysm (citing Levin, and McLellan, Spontaneous subhyaloid and retinal hems in an infant, 1986). ... Intraretinal hem is rare in chil w raised ICP. Why? Suggests some ideas. Concludes that intracranial hemorrhage without shaking is a rare cause of intraretinal hemorrhage in children. As for cases where there is no clear mechanism for the ICH and there is RH, he says that on the basis of the rarity of RH in nonabusive ICH and its commonness in abusive ICH, "should lead one to consider shaken baby syndrome as the primary diagnosis."

Comment by JKR (9/11/02): interesting that this series had two small children who suffered intracranial hemorrhage from short-distance falls.

Further comment by JKR (9/11/02): In this discussion Dr Levin for the first time acknowledges that the mechanism of abusive RH is unknown, and that traumatic retinoschisis (even assuming it exists) accounts for only a small minority of cases. That leaves open the mechanism of the great majority of abusive RH. Dr Levin appears here to be edging toward a realization that either ONSH or increased ICP or both can cause intraretinal hemorrhage by a venous-drainage mechanism in Terson's cases. What he does not do yet is take the next step, and ask whether the same mechanism could account for abusive RH. But he appears to be edging toward acknowledging this also.

This article is reviewed by Dr Reece in *Child Abuse Quarterly* for January 2003. Dr Reece rehearses the usual arguments against nontraumatic mechanisms for extensive RH:

1. The theory of blood tracking through the optic canal "has been refuted anatomically..."
2. The theory of retinal vein compression "has been challenged by primate research showing that such compression does not result in RH."
3. The theory of increased ICP causing decreased venous drainage from the retina "is contradicted by the presence of another outlet for drainage of the globe: the facial and pterygoid venous

systems...”

Retinal hemorrhages in infant head injury. (letter) Clark BJ, Adams GG, Luthert PJ. Brain 2002 Mar; 125 (Pt 3): 677-678 (have). Arguing for the abrupt-increase of ICP theory of RH, and against the vitreoretinal traction theory. Commenting on Geddes I and Geddes II (2001a). Doesn't give a detailed argument. See under “RH -- in general.”

The natural history of nontraumatic subarachnoid hemorrhage-related intraocular hemorrhages. Steibel-Kalish H, Turtel LS, Kupersmith MJ (Israel). Retina 2004 Feb; 24(1): 36-40. Out of 450 pts with known intracranial aneurysms, 70 had SAH. Of these, 19 pts had intraocular hemorrhage (30 eyes).

RH only	4 eyes
Subhyaloid hemorrhage without VH	12 eyes
Vitreous hemorrhage	14 eyes

Vitrectomy was performed only in the eyes that had VH. Followup showed good VA in the nonoperated eyes with few complications. Conclusion: the natural history of these hemorrhages is benign except for VH, which may require early vitrectomy.

The authors state, “Vitreous hemorrhage in association with subarachnoid hemorrhage is termed Terson syndrome, yet other forms of posterior segment hemorrhages are known to occur with intracranial bleeding. Many authors have considered all types of intraocular hemorrhage to be Terson syndrome, although each may have a different prognosis. Additionally, traumatic and spontaneous causes of intracranial hemorrhages have been included in studies reporting the outcome of pts with intraocular hemorrhages. Bc eyes with intraocular hemorrhage after traumatic intracranial bleeding may have other traumatic ocular injuries, the prognosis may differ from the intraocular hemorrhage associated with spontaneous SAH.” Goes on to discuss the indications for early vitrectomy. “Our results show that eyes with intraocular hemorrhage associated with SAH from aneurysm rupture frequently achieve good visual recovery, even without intervention.” In other words, the natural history of the spontaneous lesion is better than that of the traumatic lesion. Cites Weingeist, Goldman, Terson's syndrome: clinicopathological correlations, Ophth 1986; 93: 1435-1442. Weingeist et al., Long term outcome in Terson, 1991; Keithan et al., Retinal folds in Terson syndrome, Ophth 1993; 100: 1187-1190; Kuhn et al, Terson syndrome: results of vitrectomy and the significance of vitreous hemorrhage in pts with SAH, 1998.

Retinal hemorrhages of crush head injury: learning from the outliers. Levin AV. Arch Ophth 2006 Dec; 124(12): 1773-1774. Editorial comment on Lueder and Lantz, both of which were crushing head injuries with TR. Dr Levin says that TS in infants is rare, citing his own Schloff art 2002 above. See under TR, above.

Vitreous hemorrhage

Lens-sparing vitreous surgery for infantile amblyogenic vitreous hemorrhage. Capone A. Retina 2003 Dec; 23(6): 792-795. Performed lens-sparing vitrectomy with long-term followup on 15 infant eyes aged 2 to 23 months with VH due to

SBS	11
Terson's	1
Birth trauma	1
Coagulopathy	2

Two of the SBS infants had bilateral nonrecordable flash VEP's preoperatively. One SBS eye had total rhegmatogenous retinal detachment with proliferative vitreopathy. Ten patients had visual improvement.

But “visual outcome in SBS may be limited as a consequence of structural damage to the retina, optic nerve, or posterior visual pathways.”

Extension of retinal hemorrhage into the vitreous of a shaken baby through a break in the internal limiting membrane. Lincoff H, Madjarov B, Lincoff N et al. Arch Ophth 2003 Jan; 121(1): 91-96. Comment, Emerson GG, 2004 May; 122(5): 792. The 2004 comment says that Lincoff showed that a vitreous cloud of erythrocyte fragments can emanate through the internal limiting membrane without any break in the ILM. They did this experimentally by injecting autologous blood into the subretinal space of rabbits, and saw the vitreous cloud appear, and then did a vitrectomy and closely examined the ILM to rule out any break. OK, well, we (Emerson) recently examined a girl with SBS. She had severe ONSH, diffuse RH, and circumferential perimacular folds. Histology revealed (gives a very nice photomicrograph showing a tiny defect) that she had a tiny microscopic break in the ILM overlying the RH, and through this tiny break, intact RBC's were seen escaping into the vitreous humor. Posits that in the case of SBS, unlike the rabbit injection experiment, there are traumatic forces to the ILM.

Vitreous hemorrhage in children. Spirn MJ, Lynn MJ, Hubbard GB III. Ophth 2006; 113: 848-852. According to Dr Strominger's review in the *Quarterly* for Autumn 2006, this retrospective series from Emory 168 cases excluding ROP cases. By far the great majority were unilateral. But when it came to the 9.5% that were bilateral, 50% of them were SBS. Overall, cause of VH was

trauma	73%	
penetrating		24%
nonpenetrating		30%
SBS		9%
birth trauma	5%	
postoperative	5%	
spontaneous	27%	

Purtscher's syndrome (Valsalva's hemorrhagic retinopathy)

Annable states that Valsalva's hemorrhagic retinopathy only causes NFL hemorrhages.

Strangulation cases are presented by Khan & Frenkel (below) and by Walsh & Hoyt (1969.) See Khan & Frenkel's discussion of mechanism.

"Valsalva's hemorrhagic retinopathy, resulting from sustained closure of the glottis and a subsequent rise in intrathoracic pressure, is associated only with superficial retinal hemorrhages." Lambert, Johnson and Hoyt, infra, 1986, p. 1512, citing Duane TD, Valsalva hemorrhagic retinopathy. Am J Ophth 1973; 75: 637-642. Annable in Reece's Child Abuse, 1st ed. "...the Valsalva mechanism is unlikely to be the only explanation [for RH in SBS] because the RH in the SBS are in all layers and not just in the superficial layers, as is true with Valsalva's retinopathy." (emphasis in original).

Some observations on Purtscher's disease: traumatic retinal angiopathy. Marr WG, Marr EG. Am J Ophth 1962; 54: 693. Cited by Gilkes & Mann, 1967 for the similarity of retinal findings to an infant who had been gripped by the chest and shaken.

Traumatic retinal angiopathy (Purtscher). Madsen PH. Ophthalmologica 1972; 165: 453-458. Referenced by Kivlin in her chapter in Lazoritz & Palusci's SBS book, 2001, p. 142.

Purtscher retinopathy in the battered child syndrome. Tomasi L and Rosman P. Am J Dis Child 1975; 129: 1335-1337. These guys reported two battered infants with RH and "exudates." One infant was

thought to have been strangled and had retinal detachment. See discussion by Ober, 1980, concerning retinal detachment. Do they diagnose Purtscher's simply because there are white lesions in the retina?

Some observations on Purtscher's disease: traumatic retinal angiopathy. Am J Ophth 1962; 54: 693-705. Purtscher's retinopathy, caused by crushing chest trauma, is characterized by cotton-wool spots and superficial hemorrhages. Classically, according to Lambert et al. (*infra*) it has cytoid bodies. Annable, in Reece's Child Abuse, states, "Typical Purtscher's retinopathy, however, which occurs after crushing injuries to the chest, is associated with cotton-wool spots and superficial hemorrhages..." (p. 144). See also Tomasi, 1975 on Purtscher's in the child-abuse setting.

Intravitreal hemorrhage associated with rapid increase in intracranial pressure (Terson's syndrome). Khan SG, Freinkel M. Am J Ophth 1975; 80: 37-43. See under "Terson's." Presents a prison patient who was hit, strangled, possibly chest compressed, and had vitreous hemorrhage.

Lung injury resulting from nonaccidental crush injury to the chest. McEniery J, Hanson R, Grigor W, Horowitz A. Pediatric Emergency Care 1991; 7: 166-168. Cited by Starling in her chapter on AHT in Giardino & Alexander, *Child Maltreatment: A Clinical Guide and Reference, Third Edition*, 2005 (p. 44) as being a case report of a six week old infant with inflicted crushing injury of the chest who did not have RH at autopsy. Showing that Purtscher's syndrome is very rare in infants.

Acute pancreatitis with Purtscher's retinopathy: case report and review of the literature. Campo SM, Gasparri V, Catarinelli G, Sepe M. Dig Liv Dis 2000 Nov; 32(8): 729-732. A 32 year old alcoholic w acute pancr developed retinal arteriolar occlusion suspected to be due to leukocyte aggregation due to complement activation.

Retinal haemorrhages in child abuse. Levin AV. In: T J David, ed., *Recent Advances in Paediatrics 18*. London: Churchill Livingstone, 2000. "Characterized by large white patches in the retina which may represent fat emboli, superficial retinal infarction, or exudate..." Given that chest compression may occur in SBS, "some authors feel that this pressure can be transmitted up to the retinal veins contributing to the formation of RH. (citing Wilkins, 1997; Duane, 1972; Tomasi, 1986) What if the caregiver states that they or someone else fell on the child? "I doubt that this type of injury would specifically result in enough compression of the chest..." Plus, the occurrence of unilateral RH in AHT is against this.

Does Valsalva retinopathy occur in infants? An initial investigation in infants with vomiting caused by pyloric stenosis. Herr S, Pierce MC, Ford H, Pitetti R. Pediatrics 2004; 113: 1658-1661. From Dr Kaplan's review in the *Quarterly*, did direct ophthalmoscopy on 100 vomiting infants under 5 mos with pyloric stenosis, found 0 RH.

Birth hemorrhages

Retinal hemorrhages in the newborn after vacuum delivery and spontaneous vaginal delivery. Schlaeder G et al. Gyn Obst 1925; 70: 98. According to Caffey (1974), these authors found RH in 41/100 vacuum (ventouse) extractions as opposed to 15/100 NSVD's, about three times more common. The findings of Emerson et al. (2001) are comparable.

Le pronostic eloigne des hemorrhagies retiniennes du nouveau-ne. Bull Soc Fr Ophth 1949; 62: 227-236.

Retinal hemorrhages in the newborn. Giles CL. Am J Ophth 1960; 49: 1005-1010.

Retinal and conjunctival hemorrhages in the newborn. Baum JD, Bulpitt CJ. Arch Dis Child 1970; 45: 344-349. Cited by Caffey (1974) in his literature review of birth hemorrhages to distinguish them from traumatic (abusive) hemorrhages. "Baum and Bulpitt compared the incidence of idiopathic retinal

hemorrhages and idiopathic conjunctival hemorrhages in the newly born infant. Retinal hemorrhages seemed to result from several causes: increase in the blood viscosity and polycythemia appeared to be the major causal factors. Conjunctival hemorrhages, on the other hand, appeared to result principally from increases in the cephalic pressures.”

Retinal hemorrhages in newborn infants. Sezen F. Br J Ophth 1970; 55: 248-253. RH were detected in 14% of the newborns, but after 5 days, only 2.6% still had them. Caffey summarizes this article as follows: “Sezen found retinal hemorrhages in 14% of 1,238 newly born infants immediately after birth. Between the third and fifth day this incidence of 14% had diminished to 2.6%. This indicates that most of the idiopathic retinal hemorrhages of the newborn infant disappear during the first weeks of life, in contrast to the observations of Aron who found that the retinal hemorrhages in abused infants persisted for ten years and in one case for 19 years.” Caffey, 1974.

Retinal hemorrhages in the newborn. Planten JT and Schaaf PC. Ophthalmologica 1971; 162: 213. According to Caffey’s literature review (1974), “Planten and Schaaf concluded that idiopathic retinal hemorrhages of the newborn infant appear in 20% to 30%, but that they cannot be causally related to increased intracranial pressure during labor, they rarely occur during breech deliveries or caesarean section, and are rarely associated with subdural hematomas or other signs of brain damage.”

Retinal hemorrhage in newborns and organic amblyopia. Van Noorden GK, Khodahoust A. Ann Ophth 1973; 89: 91-93.

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. (have) Caffey reviews the literature on birth hemorrhages, in order to provide a basis for distinguishing the “ocular bleedings” of battered infants from birth hemorrhages on the ground that birth hemorrhages do not persist beyond a few weeks, while those of battering tend to persist for many years. He cites articles by Schlaeder et al. on vacuum delivery (1925), Baum & Bulpitt (1970), Sezen (1970), Aron (1970), Planten and Schaaf (1971). From these, he concludes that (a) birth hemorrhages disappear in a few weeks, (b) birth hemorrhages are not due to trauma or to increased intracranial pressure (which Caffey seems to equate), (c) that birth hemorrhages may be due to polycythemia or hyperviscosity.

Perinatal retinal hemorrhages: morphology, etiology, and significance. Von Barsewisch B. Berlin: Springer Verlag, 1979.

Fundus hemorrhages in infancy. Kaur et al. Surveys of Ophthalmology 1992 Jul-Aug; 37(1): A frequently consulted review for non-ophthalmologists.

Magnetic resonance imaging evaluation of neonates with retinal hemorrhages. Alexander R, Smith W, Judisch G, Sato Y et al. Pediatrics 1992; 89: 332-333.

Obstetric correlates of neonatal retinal hemorrhage. Williams MC, Knuppel RA, O’Brien WF, Weiss A et al. Ob Gyn 1993 May; 81(5 Pt 1): 688-694. Moderate to severe RH was found in 18% of spontaneous, 13% of forceps, 28% of vacuums, and 50% of sequential vacuum- and forceps-assisted deliveries. Conclusions: Maternal and fetal factors other than vacuum-assisted delivery are significant correlates of moderate to severe RH. Vacuum-assisted delivery among SGA infants is closely correlated with moderate to severe RH.

Hemorrhagic retinopathy in newborns: frequency, form of presentation, associated factors, and significance. Gonzalez Viejo I et al. Eur J Ophth 1995 Oct-Dec; 5(4): 247-250. (Spain) They prospectively examined 420 neonates. Found RH in 81 (19%). Lower freq in C/S, +assoc with prolonged labor. All were normal 3 – 4 mos later.

Long-term outcome of neonatal macular hemorrhage. Zwaan J, Cardenas R, O’Connor PS. Pediatr Ophth Strab 1997; 34: 286-288.

Vascular retinal abnormalities in neonates of mothers who smoked during pregnancy. Beratis NG et al. Journal of Pediatrics 2000; 136: 760-766. 162 newborns of smoking mothers and 162 controls matched for -----? Used imaging to study the retinal artery and vein as well as the retina. 61 smokers had birth hemorrhages, while 36 nonsmokers. Statistically significant. The authors postulate that increased blood viscosity due to elevated hematocrit might be involved.

Incidence and rate of disappearance of retinal hemorrhage in newborns. Emerson MV, Pieramici DJ, Stoessel KM, Berreen JP, Gariano RF, Yale Med, Ophth 2001 Jan; 108(1): 36-39. (have abstract) Prospective study of 149 newborns at an urban hospital found these rates of intraretinal hemorrhage by indirect ophth:

overall	34%
NSVD	33%
Vacuum	75%
C/S	7%

Number of hems:

One or two	22% of eyes
More than ten	54%

The hems were dot-blot or flame, also large white-centered blots. All but one were intraretinal; one pt had subretinal.

Clearing

By two weeks	86% of eyes
By four weeks	all except the one subretinal

Incidence and rate of disappearance of retinal hemorrhage in newborns. Emerson MV, Pieramici DJ, Stoessel KM, Berreen JP, Gariano RF. Ophth 2001 Jan; 108(1): 36-39. The following abstracted from Penny Grant's review in Child Abuse Quarterly, July 2001: A prospective study in which 149 normal nb had dilated ophthalmoscopy in the first 30 hours of life and then were followed every two weeks in clinic. They stratified their hems by extent: one DD from disc, out to the equator, or out to the ora, and by number of hems. All hems were intraretinal except one subretinal. Overall 34% had RH. 75% of vacuums, 33% of NSVD, 7% of C/S. RH was not statistically associated with any labor factors or fetal factors, maternal factors. 33% had some peripheral hems. But overall the hems were overwhelmingly posterior in distribution. 54% had more than ten hems. The hems disappeared by two weeks in 86%, all but one by four weeks, which was the subretinal case. Noted that preretinal hem or vitreous hem never happened.

Obstetric vacuum extraction: state of the art in the new millenium. Mikovsky P, Watson WJ. Ob Gyn Surv 2001 Nov; 56(11): 736-751. Mentions RH. Vacuum is replacing forceps as the preferred method of delivery. [Does this mean we will be seeing more birth hemorrhages? See Williams et al., 1993 and Emerson et al., 2001, above. JKR]

Hemosiderin

The medicolegal implications of detecting hemosiderin in the eyes of children who are suspected of being abused. Gilliland MGF, Luckenbach MW, Massicotte SJ, and Folberg R. Arch Ophth 1991; 109: 321-322

Long term sequelae

Ocular damage in the battered-baby syndrome. Mushin AS. Br Med J 1971; 3: 402-404. According to Caffey's summary (1974), "Mushin found ocular changes in 12 of 19 *battered infants*, all 12 of whom had permanent impairment of vision in one or both eyes."

Electroretinographic findings in infants with the shaken baby syndrome. Fishman CD, Dasher WB, Lamber SR. J Pediatr Ophth Strab 1998; 35: 22-26

Optic disc neovascularization following severe retinoschisis due to shaken baby syndrome. Brown SM and Shami M. Arch Ophth 1999; 117: 838-839. Four months later a lot of vessels were seen growing out into the vitreous from the disc. Can progress to rubeosis irides. Needs to be watched for possible operative treatment.

Long-term outcome in infants with the shaking-impact syndrome. Duhaime A-C, Christian C, MossE, SeidleT. Pediatr Neurol 1996; 24: 292-298.

SHAKEN (= AHT)

(Some articles have been moved to the “Biomechanics” section, below.)

Diagnostic criteria, as stated by the authors of the 2007 multicenter study (Hymel KP et al., Pediatrics 2007 May; 119(5): 922-929, see below):

1. Cases in which the child’s primary caregiver admitted abusive acts that could be linked to the child’s acute clinical presentation for traumatic cranial injuries
2. Cases in which an independent witness verified abusive acts that could be linked to the child’s acute clinical presentation for traumatic cranial injuries
3. Cases in which a child not yet cruising or walking became clearly and persistently ill with signs of acute cardiorespiratory compromise linked to his/her traumatic cranial injuries while in the care of a primary caregiver who denied any knowledge of a head injury event
4. Cases in which the child’s primary caregiver provided an explanation for the child’s head injury event that was clearly developmentally inconsistent with the parent(s)’ description of their child’s developmental capabilities
5. Cases in which the child’s primary caregiver provided an explanation for the child’s head injury event that was highly inconsistent with repetition over time [meaning repetition of the history, i.e. a changing history --JKR]
6. Cases in which the head-injured child revealed at least two noncranial injuries considered moderately or highly specific for abuse

The authors state that these criteria “are free of circular logic and other inherent biases,” and that they are “conservative.” As to circular reasoning, see Donohoe M, Evidence-based medicine and shaken baby syndrome. Part I: literature review, 1966-1998. Am J Forens Med P 2003 Sep; 24(3): 239-242. Now actually, these criteria are not proposed by the particular authors as clinical diagnostic criteria, only as research criteria intended to be conservative. Compare the criteria of Duhaime et al., 1998, *infra*. --JKR

National Institute of Neurological Diseases and Stroke

<http://www.ninds.nih.gov/disorders/shakenbaby/shakenbaby.htm>

National Center on Shaken Baby Syndrome

<http://www.dontshake.com>

Brain Injury Association

<http://www.biausa.org>

Defense

<http://www.sbsdefense.com>

Lazoritz S and Palusci VJ, eds. *The Shaken Baby Syndrome: A Multidisciplinary Approach*. New York: The Haworth Abuse & Trauma Press, 2001.

Abusive Head Trauma in Infants and Children: Medical, Legal, and Forensic Issues. Two volume set. Kay Rauth-Farley, MD, Lori Frasier, MD, Robert Parrish, JD, Randell Alexander, MD, PhD. Saint Louis: G.W. Medical Publishing, Inc., 2005.

Definition (after Caffey): "...intracranial and intraocular hemorrhages, in the absence of signs of external trauma to the head or fractures of the calvaria, and are associated with traction lesions of the periosteum of the long bones in the absence of traumatic changes in the overlying skin of the extremities." From: Caffey J. The whiplash shaken infant syndrome: manual shaking of the extremities with whiplash-induced intracranial and extraocular bleeding, linked with residual permanent brain damage and mental retardation. Pediatrics 1974; 54: 396-403. Another definition is in AV Levin, 2000: "[T]he association of characteristic brain injuries and retinal haemorrhages is diagnostic of SBS." (see under RH.) A usable definition is: Inflicted traumatic brain injury and intracranial hemorrhage in a small child without evidence of impact to the head. (Notice that I didn't include retinal hemorrhage in that definition, partly bc the AAP 1993 stated they were present in 75 to 90%, not 100%; 83% in Kivlin's series (2000). "RH are not needed to make the dx of SBS." -- Kivlin, 2001. Some authorities do include them.)

Definition (after Levin): SBS being defined as two or more of the following:

- characteristic neuroradiologic abnormalities
- skeletal injury
- RH
- history of shaking
- no adequate history to explain the injuries

Definition: "Shaken Baby Syndrome is a collection of findings, all of which may not be present in any individual child with the condition. Injuries that characterize Shaken Baby Syndrome are intracranial hemorrhage; retinal haemorrhage; and fractures of the ribs and at the ends of the long bones. Impact trauma may produce additional injuries such as bruises, lacerations or other fractures. Shaken Baby Syndrome is a condition that occurs when an infant or young child is shaken violently, usually by a parent or caregiver. Some experts believe that impact trauma to the head is a necessary component of the mechanism of injury. Signs of impact may or may not be visible because the impact which produces sudden deceleration of the head may be against a soft object such as a mattress."

From the "Joint Statement on Shaken Baby Syndrome" of Health Canada, 2002. Available from the Health Canada web site at http://www.hc-sc.gc.ca/dca-dea/publications/jointstatement_web_e.html

Etude medico-legale sur les services et mauvais traitements exerces sur des enfants. Tardieu A. (Auguste Ambroise Tardieu) Ann Hyg Publ Med Leg 1860; 13: 361-398. Recognizing subdural hemorrhage as a form of child abuse. See above under "classic articles" and "fractures."

Chronic subdural hematomas in infants. Sherwood P. Am J Dis Child 1930; 39: 980. Brogdon's book *Forensic Radiology* (1998), pp. 283-284 refers to this as a "classic paper," and describes it as presenting nine cases of chronic SDH in infants, of whom Brogdon quotes the paper as concluding that the etiology of these was obscure, but noting that five of the nine infants were cared for in institutions or by foster mothers, and stating that "trauma due to injury at birth or other means is a possible factor." Brogdon points out that in 1930 medicine still was only guessing at the traumatic nature of SDH, partly because Virchow had laid down that it was of infectious etiology.

Multiple fractures in the long bones of infants suffering from chronic subdural hematoma. Caffey J, Am J Roentg (AJR) 1946; 56: 163-173. [cf Lazoritz, Has Caffey's syndrome changed... Child Abuse & Neglect 1997; 21(10): 1009-1014.] Quoted by Dr. Tongue (see under "RH") to the effect that "Caffey, in 1946, suggested that multiple fractures in long bones in infants with chronic subdural hematomas were secondary to trauma. Two of these children also had RH. Caffey recognized that only trauma could explain the spectrum of injuries, yet he failed to implicate intentional trauma and stated that the injuries could be the result of forgotten or relatively minor traumatic episodes."

SBS is a legally accepted diagnosis. *State v. Lopez*, 412 SE2d 390 (S.C. 1991); *State v. McClary*, 541 A2d 96 (Conn 1988). But it is becoming more controversial. *People v. Manjit Kaur Basuta*, Calif. Court of Appeal No. D034429, Dec. 10, 2001 (see below) and *People v. Stephen Martinez*, 2001 WL 1630083 (Colo. App No. 00CA0312, 2001) (Westlaw).

The pathology of child abuse. Weston JT. In: Kempe CH, Helfer RE, eds., *The Battered Child*, 3d edition. Chicago: University of Chicago Press, pp. 77-100. Cited by Guthkelch, 1971, as finding a 42% incidence of SDH in his series of battered children aged 2 months to 5 years with an average age of 24 mos. Cited by Kleinman (*Diagnostic Imaging of Child Abuse*, 2d ed.) and by Guthkelch as describing three infants with SDH and adult admission of violent shaking. "In only one of these was there any mark of injury on the head..." (Guthkelch). In fatal cases "examination of the galea frequently revealed numerous ... haemorrhages ... even in the absence of conspicuous external bruising, abrasion, or laceration." Also cited by Caffey (1974) as part of his literature review collecting 28 historical cases of shaking associated with injury to bones or brain.

Whiplash injury and brain damage: an experimental study. Ommaya AK, Faas F, Yarnell P. JAMA 1968 Apr; 204(4): 285-289. See below under "Biomechanics."

[SDH in two adults from impactless automobile whiplash injuries.] case report. Ommaya AK, Yarnell P. Lancet 1969; 2: 237. See Guthkelch, *infra*.

Spine and spinal cord trauma in the battered child syndrome. Swischuk LE. Radiology 1969; 92: 733. Cited by Caffey (1974), 398, as follows: "Fractures of the spine with local injuries to the spinal cord of one infant were attributed by Swischuk to manual whiplash shaking."

Morphology of brain lesions from blunt trauma in early infancy. Lindenberg R and Freytag E. Arch Path 1969; 87: 298-305. Classic description of chronic white-matter tears identified in children, so-called "contusion tears." See also 1970, below. These tears "are believed to be the result of shearing of the interface between grey and white matter." (Geddes et al., 2001). (Geddes et al. suggest that the biomechanics of shearing at the gray-white interface may be different than that of shearing in the white matter.)

Brainstem lesions characteristic of traumatic hyperextension of the head. Lindenberg R and Freytag E. Arch Path 1970; 90: 509-515. This was adults. Geddes et al. cite this work as showing findings comparable to theirs in the brainstem and upper cervical cord of infants with SBS. (2001, below).

The significance of skull fractures in children. A study of 1,187 patients. Harwood-Nash CE, Hendrick EB, Hudson AR. Radiology 1971; 101: 151-156. Quoted in Chapter 22 of *Knight's Forensic Pathology, Third Edition* (2004) as having found that less than half of children with subdural hematoma had skull fractures. SDH being twice as frequent in the non-fracture cases. This was not a study of abuse.

Infantile subdural hematoma and its relationship to whiplash injuries. Guthkelch AN, Br Med J 1971 May 22; 2(759): 430-431. Remarks that SDH is common only in infants. It has an overall incidence of only 2.7% in all head injuries, but 5.2% in a pediatric series, almost all of which was in pts under 2. Weston's 1968 series of battered children found 42% had SDH -- a remarkable disparity from the general incidence of this lesion. In adults, SDH can occur after "disproportionately slight" head injuries. It is known that SDH usually results from either a heavy moving object impacting the head or the rapidly moving head being brought up against a stationary mass. However, "direct violence [impact] is not an essential part of

the picture.” (citing case reports of SDH in adults from a fall on the buttocks, and Ommaya and Yarnell’s (1969) report of two cases of SDH from impactless automobile whiplash injuries. Remarking that in the clinical series of battered infants at Hull which the author studied, “by no means all the patients so affected have external marks of injury on the head.” “It is now submitted that the conditions which are known to exist in many cases of the battered child syndrome are particularly favourable to the production of subdural haematoma in infants by an essentially similar mechanism.” (to whiplash) Citing the relatively large head and puny neck muscles of the infant. Also saying that the symmetrical chest or limb grip of the adult would explain why these hematomas are usually bilateral and symmetrical (78%) while in adults they are bilateral in less than 50%. Citing Court’s 1969 report of a confession of shaking.

Reporting a retrospective clinical series of 23 cases of parental assault on children under 3 (and all but one under 18 months), with SDH in 13 (57%), bilateral in 10. (Apparently a chart review) “The only sort of injury which was recorded more often being bruising of the skin. One or more fractures of long bones were found in association with SDH in six cases.” [By “only...bruises of the skin” Guthkelch seems to mean the lack of skull fracture. Eight pts had skull fx, 15 did not. --JKR] Seven pts had SDH and no skull fx; of these, five had no **external** marks of injury. Of these, one was found at operation to have extensive cortical bruising, and another was found at necropsy to have surface hemorrhages on the cerebral cortex. [That would leave three with no evidence of impact. --JKR]

Presents two “personal cases:” Case 1 a 6-month boy presenting in convulsions with no marks of injury. Bilateral SDH. At necropsy, “...the surface of the underlying [i.e. superior parasagittal] brain was contused, with some small lacerations.” Mother confessed to shaking him “to try to clear his throat.” No signs of bruising of the chest wall and no rib fractures. Case 2 a 6 month boy w convulsions and no external signs of injury and no fractures of the skull or long bones. Tense fontanelle and bilateral RH, bilateral SDH. Mother admitted that they “might have” shaken him when he cried at night.

Discussion: “The mere absence of visible injury on the head does not exclude direct violence, for Weston (1968) pointed out that in fatal cases ‘examination of the galea frequently revealed numerous ... haemorrhages ... even in the absence of conspicuous external bruising, abrasion, or laceration.’ In a non-fatal case there is, of course, no opportunity to expose and examine the deep layers of the scalp... Nonetheless, one has the impression that ‘a good shaking,’ is felt, at least by British parents, to be socially more acceptable and physically less dangerous than a blow on the head or elsewhere.”

Conclusion: The discrepancy between the frequency of SDH in battered children versus other head injury victims even in the absence of skull fracture or clear ev of direct violence to the head suggests that “when the head is not the main target of attack, the likely mechanism of production of the haematoma is one in which repeated sheering (sic) strains of one sort or another are applied to the cranial contents. Warning clinicians that even if there is no external sign of injury, one must keep in mind the possibility of assault if there is a SDH.

Cited by Caffey (1974). Cited by Kleinman (1998) as describing two infants with convulsions and confessions of shaking with no evidence of external injury. Guthkelch is cited by Brogdon (1998) as having been credited with first having linked subdural hematoma to shaking. “However,” Brogdon states, “that claim of primacy may belong to Weston, who described three instances of subdural hematoma in infants who had been violently shaken.” (citing Weston’s chapter, “The pathology of child abuse,” in the third edition of Kempe & Helfer’s *The Battered Child* (1968)) (above). Summarized by Leestma (2005) as follows: “In case 1, the mother said that the baby had had several ‘fits,’ and she feared he was going to choke, so she shook him several times to try to clear his throat, whereupon he went into convulsions. The mother admitted the possibility of innocent, though thoughtless, actions on the baby. In case 2, the mother denied striking or beating the child but admitted she and her husband ‘might have’ shaken the baby after he cried at night. Oval bruises matching finger pads were found on forearms of the baby.”

Ocular injury in the battered-baby syndrome: report of two cases. Mushin A, Morgan G. Br J Ophth 1971; 55: 343-347. According to Ober (1980), Mushin presents two cases of retinal detachment in children with head trauma and comments on the considerable force required to cause direct damage to the retina. Caffey cites this article in “The whiplash shaken infant syndrome” (1974) as a report of “intraocular hemorrhages” reported in shaken infants (as opposed to “so-called battered infants”). Leestma (2005) states that “In this case, the father admitted to strangling the baby with a blanket and then spent the remainder of the night shaking the baby in an attempt to revive him. Pharyngeal bruising and hemorrhages were noted at autopsy and ‘extensive’ bruising, sites not specified, was noted as well.”

On the theory and practice of shaking infants. Its potential residual effects of permanent brain damage and mental retardation (the Jacobi lecture) Caffey J. Am J Dis Child 1972 Aug; 124(2): 161-169 This is actually two lectures, the first a long disquisition on chronic abusive long-bone fractures, and the second a disquisition on chronic subdural hematomas.

The fractures take the form of either (a) metaphyseal lesions, or (b) subperiosteal new bone formation, often repetitive. Caffey emphasizes that the metaphyseal lesions are due to “indirect, traction, stretching, and shearing, acceleration-deceleration stresses on the periosteum and articular capsules, rather than direct, impact stresses such as smashing blows on the bone itself.” He states that these “indirect, primary, acceleration-deceleration traction drags on the periosteum, its vessels, and the joint capsules from manual seizing and gripping the extremities and whiplash-shaking the head.” Describes 27 cases of confessed shaking -- one from Newsweek magazine for 1956, four previously reported by Weston and Steele and Pollock in the 1965 edition of *The Battered Child* (these were shaking plus beating or slamming cases), Guthkelch’s three shaking cases, one case from Kempe et al. (Kempe, Silverman, Steele, 1962) who was held upside down and shaken by the legs, with resulting leg bone lesions, a case from Swischuk (1969) having compression fractures of the spine believed to have come from shaking, two cases of RH and SDH reported by Gilkes (Fundi of battered babies, 1967) -- one of leg-swinging and one of shaking, and three of his own cases with traumatic involucra of the leg bones, no head injury, caused by leg-seizing. All of this he refers to as “whiplash-shaking.” He states that this type of “grabbing and gripping of an infant or younger child by the extremities or by one leg or arm and then shaking him” was common and commonly considered innocuous. He includes those grabbings done in anger and also those done in play or thought to be therapeutic, and states that all are dangerous because they are “brain jolting.” He goes on to inveigh against various vibratory toys, playslides, “contraptions,” small airplanes, trampolines, games, and even invokes endogenous seizures, such as those seen in meningitis and epilepsy, and also endogenous head-banging and body-rocking by infants “in the first months of life,” and even breath-holding, as pathogenic to the brain.

Subdural hematomas: [On this subject, note that Caffey does not have any first-hand material to present. His discussion consists of a review of the material presented by Ingraham and Matson in their treatise *Neurosurgery of Infancy and Childhood* (Springfield: Charles C. Thomas, 1954), with commentary. First of all Caffey points out that subdural hematomas are practically always traumatic in origin. (While this seems a commonplace today, it was still not universally understood in 1972.) Next he quotes Ingraham & Matson showing that the peak incidence of SDH is in the sixth month of life. Caffey goes on immediately to assert that “This high vulnerability of the neonate and the younger infant to traumatic intracranial bleeding is due to the combination of heavy head and weak neck muscles, which renders his brain especially susceptible to whiplash stresses.” Adds the suppleness of the skull which permits stretching of the veins, and the softness of the unmyelinated brain. Mentions that in the 319 SDH cases of Ingraham & Matson, fractures of the skull were found in only 9%, and bruises rarely. In that series there were many chronic SDH’s. “One can reasonably conclude that whiplash-shaking and jerkings, which are the prime causes of subdural hematoma in diagnosed cases, are also substantial causes of later mental retardation in the countless cases of unrecognized chronic SDH...” “The frequent bilaterality of SDHs also supports whiplash-shaking as the probable cause.” As evidence for **shaking rather than impact** mechanism of SDH, he adduces the following (page 169): (1) bilaterality of subdural hematomas in 85% of infants and frequent bilateral retinal hemorrhages, (2) “a striking lack of such signs of impact injuries such as blows to the head. Usually there are no bruises to the face or scalp, no subperiosteal cephalhematomas, and no fractures of the calvarium.”

As to RH, he refers to the work of Kiffney (1964), Maroteaux (1967) and Friendly (1971), and states that “The retinal lesions caused by shaking will undoubtedly become valuable signs in the diagnosis of subclinical inapparent chronic subdural hematoma...”

Goes on to discuss chronic infantile hydrocephalus with meningeal “gliosis” in idiopathic juvenile mental retardation, states that the neuropathology of this has not been studied, and raises the possibility that a proportion of these cases could be due to shaking.

In conclusion, “The radiographic bone changes in the [battered child syndrome], both their nature and distribution, indicate that they are usually caused largely by rough manhandling of the infantile arms and legs, such as grabbing and then grasping, squeezing, wringing, and jerking, and then whiplash-shaking of the head...” Reiterates that “The pathogenicity of ordinary, habitual, customary, repeated shaking of infants is generally unrecognized by physicians and parents.” He extends this point, stating that “The habitual, repeated, relatively mild whiplash-shakings which are inflicated in the ordinary training and

disciplining of infants, and which may induce undetected cumulative chronic subdural hematomas...are probably more pathogenic than the less frequent but more violent and conspicuous shakings during wilful assault, because they are consistently unrecognized and may persist to generate mental retardation and permanent brain damage.” States that the consequences of this are that “some, perhaps many” of the cerebrovascular injuries currently attributed to various causes “are undoubtedly caused by undetected, depreciated, and inapparent whiplash-shakings during the first weeks and months of life.”

“Fallen fontanelle” (*caida de mollera*): a variant of the battered child syndrome. Guarnaschelli J, Lee J, Pitts FW. JAMA 1972; 222: 1545. A 2 mo Mexican male with bilateral SDH sz, tense fontanelle, fixed & dilated, was shaken upside-down by parents to relieve a fallen fontanelle. [According to the biomechanics, this cannot happen.] This article is cited by Caffey as “a bizarre example” in his 1974 article defining the whiplash shaken infant syndrome. Caffey recites that the Mexican grandmother “had attempted to raise the sunken fontanel by a series of therapeutic maneuvers which terminated in holding the infant topsy-turvy by its ankles, with its head over a pan of water, and then shaking the infant up and down while an assistant slapped and pounded on the soles of its feet. The sunken fontanel did rise and had become bulgy when admitted to the hospital. Subhyaloid hemorrhages were found in the ocular fundi and the pupils were fixed. There were no signs of external trauma to the head, trunk, or extremities. Clonic seizures developed and the cerebrospinal fluid from the cranial subdural space and the lumbar subarachnoid space contained fresh blood. The clinical signs subsided after treatment of the subdural hematomas. The infant died 8 months later at 10 months of age from pneumonia and quadriplegia. In this case, manual longitudinal whiplash shaking of the inverted infant with concurrent pounding of the soles of its feet induced longitudinal whiplash and jerking stresses to the head which resulted in permanent severe generalized brain damage -- with cerebral palsies.”

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. Naming the syndrome. Words it in slightly different ways:

“The most characteristic pattern of physical findings in the whiplashed infant is the absence of external signs of trauma to the head and the soft tissues of the face and neck, and of the facial bones and calvaria, in the presence of massive traumatic intracranial and intraocular bleedings.” 399

“Finally, it became conclusively clear that a reasonable explanation for the pathogenesis of these common lesions -- metaphyseal avulsions and subperiosteal hemorrhages -- was traction-stretching stresses on the periosteums, induced by grabbing the infants by the extremities or by the thorax, and then shaking them, which in turn induced whiplashing of the head onto the thorax. WLS of the head was the reasonable explanation for the presence of bilateral subdural hematomas and bilateral intraocular hemorrhages, combined with concurrent absence of external signs of trauma to the head and neck and to the extremities in more than half of our early cases.” 396

Discusses what shaking is, or what he means by “whiplash shaking.” This is to be learned primarily from the list of case reports (below) that he adduces as historical support for the existence of the syndrome. As will be seen from them, these instances of shaking included both attempted therapeutic shakings, punishment shakings, and rage shakings. But interestingly, in this article, Caffey has quietly dropped a whole group of mechanisms that were included in his earlier (1972) Jacobi lecture, where he included casual and recreational shakings as well as common incidents such as playslides and motorcycles. In this present article, the only mechanisms mentioned are *manual* shakings, and indeed, that term now appears in the title. Also, he now limits his focus almost exclusively to the population of battered babies; while still insisting that the mentally retarded are a subject population, he has quietly abandoned his earlier argument that “many, if not most” retarded children are victims of unreported, possibly innocent shaking. See below for the characterization of the shakings included in this present article. Some Caffey describes as “brutal,” while others are histories that allow the inference of brutality, if not necessarily of criminal intent. He states that the critical criterion is not intent but the full-amplitude oscillation of the head -- “flexion of the head ventrad until the chin strikes the anterior chest wall (sternum), followed immediately by similar but reverse companion extensions of the head on the neck dorsad until the occiput strikes the back (upper thoracic spine).” 401 He states, as he had earlier, that these incidents are usually repetitive: “repeated during long periods,” “in paroxysms...repeated...over periods of days or weeks.” 401

Reviews all the known cases where a history or confession of manual shaking was obtained. Stating that confessions of trauma are rare, and medical histories of shaking are also rare because “it [routine shaking] is considered innocuous by both the parent-assailant and the questioning physician.”

“By far the most extensive” proof of shaking comes from the confession of that wet-nurse Virginia Jaspers back in 1956; the sources given for this material are the personal communication of the physician who obtained the original confession and news reports from *Newsweek* and *Master Detective*. The author quotes extensively from the [New Haven] coroner’s autopsy reports on two of the wet-nurse’s shaking victims: case one a 12 day old newborn admitted with gasping respirations and no external signs of trauma. There was a bulging fontanelle and diffuse hemorrhages in the ocular fundi. “Brain and head: bulging anterior fontanel, bilateral subdural hematomas, bilateral subarachnoid bleedings, subpial bleeding, lacerations of the cerebral parenchyma [the white matter? JKR], pyknosis and death of ganglion cells and large perivascular bleedings.” Case two a 11 week admitted semicomatose with a bulging fontanelle. Ocular fundi invisible (bleeding?), bloody CSF, no external signs of trauma on the face or head, “Brain: no external signs of trauma to the head; bilateral subdural hematomas with subarachnoid hemorrhages...extensive bleedings at the sites of attachment of the bridging cerebral veins to the superior sagittal sinus...” This wet-nurse’s shakings are described by Caffey as “brutal.” 397 Many of them resulted in brain damage and mental retardation.

Cites Guthkelch’s finding of subdural bleedings in 13 of 23 abused infants, 22 of whom were younger than 18 months. Five of the 13 with SDH had no external signs of head trauma or cranial fracture.

Cites two additional cases of Guthkelch’s with histories of shaking: Case one a 6 months with no external signs of trauma, at autopsy having torn bridging veins at the falx; case 2 a 6 months with no external signs of trauma, clinically treated subdural hemorrhages, discharged, readmitted with recurrent SDH’s, grab marks on the arms, confession of shaking by the father, survived.

Cites Weston’s two infants who had been shaken to death and had SDH’s at autopsy.

Cites two infants of Steele and Pollock described by Weston in the same chapter, who were shaken and apparently did not have intracranial sequelae.

Cites one case reported by Silverman of a 7 months who was shaken by the legs upside down and had metaphyseal fractures in the legs, no SDH.

Cites three cases of his own: two infants whose leg was jerked and had metaphyseal avulsions, and one with metaphyseal lesions after being shaken by an 8 year old sibling. None of these three had SDH’s.

Cites Swischuk (1969) reporting one infant who sustained fractures of the spine with local injuries to the spinal cord from manual shaking.

Cites Guarnaschelli et al. (1972) report of *caida de mollera* in a two month-old, admitted fixed & dilated with subhyaloid hemorrhages and a bulging fontanelle with a bloody tap. [Caffey does not comment on the lack of leg bone lesions in this case, which does not fit his theory. --JKR]

Cites case of Mushin and Morgan (1971) with strangulation and shaking-to-revive of a 3-months found with extensive bruising, SDH and extensive RH.

Reviews the autopsy literature, consisting of the two coroners reports from 1948 and 1956 concerning victims of that wet-nurse in New Haven, Virginia Jaspers; there the most significant findings were limited to bleedings in the brains and eyes, in the subdural, subarachnoid and subpial spaces and in the cerebral substance itself. Plus ONSH and NFL hems. “There were no external signs of trauma to the head in either of the infants.” 399 [And, interestingly, no long-bone injuries. --JKR]

Reviews again the RH literature in child abuse, extending somewhat his earlier (1972) discussion:

In Caffey’s original 1946 series of six battered babies, two had RH with SDH

The two Yale coroner’s cases (1948, 1956)

Kiffney (1964) one case of bilateral retinal detachment in a 7 mos battered infant

Gilkes & Mann (1967) stating that they had found only Kiffney as an earlier ref on the

status of the eyes of abused infants. “In their cases, they were impressed with the extensive spread and the persistence of the signs of ocular hemorrhages, both preretinal and intraretinal, and by the presence of gross papilledema in some cases. These authors cite the pateints of Wallis who suffered from subdural hematomas induced by the parents who ‘gripping the infants by the ankles

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

Maroteaux et al. (1967) found "plaques" in the peripheral temporal retinas [the word "plaques" in french does not mean plaques; it means focal injuries. -JKR] Comments that "The authors state that these lesions cannot be satisfactorily explained on the basis of battering and they question the validity of the term 'battered child' for all abused infants...' We agree with them..."

Aron et al. (1970) found similar retinal spots located in the peripheral temporal segments, some with retinal detachments in all 18 abused infants.

Mushin and Morgan (1971) reported one case.

Harcourt & Hopkins (1971) eleven cases of ocular complications -- eight with permanent visual impairment and ten with intraocular bleedings.

Guthkelch had two cases (1971)

Friendly (1971) had five cases with vitreous hemorrhages, bilateral cataracts, dislocated lenses, and retinal detachments. Caffey comments that one of Friendly's cases sounds like a shaking case bc of lack of external trauma to the head. He notes that "In these reports by ophthalmologists manual shaking was admitted in three cases and was probable in several others." 400

Phelps (1971) reported two cases of infants with numerous unexplained pale-centered RH and no history of trauma.

Guarnaschelli's case (caida de mollera) (1972)

Mushin (1971) found ocular changes in 12 of 19 battered infants

Discusses the pathogenesis of RH in SBS. Goes into a detailed discussion of birth hemorrhages in order to rule out the possibility that the RH seen in SBS are just persistent birth hemorrhages. Shows that birth hemorrhages are transient, while SBS hemorrhages are long-lasting.

Discusses the significance of SDH as to the pathogenesis of SBS. Bilateral in more than 80% of cases. They are the critical lesion that does most of the damage. Often missed or confused with other illnesses. "These facts indicate that many features of post-traumatic subdural hematomas are not satisfactorily explained or understood; namely, ... the exact causal mechanisms of the combination of subdural and intraocular bleedings..."

Discusses "the nature of the whiplash stresses and the resistance of the infantile head to these stresses." (in other words, biomechanics.) The infantile head is relatively heavier and the neck muscles of infancy are weaker. The sutures and fontanels are more stretchable, allowing more tearing forces on the bridging veins and brain surface. Soft brain. Shaking of the infant trunk causes "a two-phase cycle of rapid, repeated, to-and-fro, alternating, acceleration-deceleration flexions of the head..." Posits repeated paroxysms of shaking over a period of days or weeks. Cites the opinion of Ommaya, "an experienced investigator of whiplash stresses on the brains of small experimental animals," who "wrote me recently in a personal communication that he agreed with me on the high risk of the whiplash shaking mechanism owing to the high vulnerability of the human infantile calvaria and brain..." Citing Ommaya et al., whiplash and brain damage, 1968; Ommaya & Yarnell, SDH after whiplash, 1969; Ommaya & Hirsch, concussions from impact and whiplash in primates, 1971. This last article as supporting the hypothesis that approximately one-half of the potential for brain injury during impact on the head is causally related to head rotation. 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.

Leestma (2005) summarizes two cases of confessed shaking in this article as follows: "In the case of baby 'H,' the information about the case strongly implies that the baby's nurse, who confessed to shaking most of the babies in her care, as well as performing other physical actions, such as pounding on the back, shook this baby. Autopsy was reported to show a liver laceration with hemorrhage, as well as intracranial

injuries... Apparently, no sign of external injury was noted. In the case of baby 'K,' specifics of the shaking were provided by the same nurse involved with baby 'H.' In this case, the nurse shook the baby until her head 'bobbed' and she became faint because she did not drink her bottle. In this case, there was no sign of external trauma but intracranial injuries were present..."

For details about the New Haven coroner's autopsies and Virginia Jaspers I am indebted to Joseph H. Davis, M.D. (internet communication, 2004). --JKR

Cerebral concussion and traumatic unconsciousness: correlation of experimental and clinical observations on blunt head injuries. Ayub K. Ommaya and T. A. Gennarelli. Brain 1974; 97: 633-654 See below under "Biomechanics."

Microcephaly following baby battering and shaking. Oliver JE. Br Med J 1975; 2: 262-264. According to Leestma (2005), this report includes two cases of confessed shaking. Leestma states, "In case 1, the mother admitted to shaking the baby and throwing it, and possibly swinging it against an object. There was a 'long' delay between infliction of the injuries and when hospitalization occurred. The child had been having 'fits' and had been refusing to eat. Skull fractures and other injuries were noted ... In case 2, the baby victim had been battered by the mother with blows of the fist and by shaking. There were bruises of the arm and cheek, as well as scratches to chest and abdomen. Old rib fractures were also noted."

Computed tomography in child abuse and cerebral contusion. Ellison PH, Tsai Fy, Largent JA. Pediatrics 1978; 62: 151-154. Cited by Leestma (2005) to the effect that "In case 3, the father admitted that while shaking the baby, his head struck a hard surface. Generalized bruising was found with ecchymoses on buttocks, both forearms, and frontal scalp. A coagulation profile was normal. There were skull fractures, multiple rib fractures, and evidence of recent and older long-bone injuries."

Elevated intracranial pressure in whiplash-shaken infant syndrome detected with normal computerized tomography. Bennett HS, French JH. Clin Pediatr 1980; 19: 633-634. Cited by Leestma (2005) to the effect that "The child was admitted flaccid and in coma, with injuries... During the hospital stay, nurses observed the father to violently shake the baby by her arms because she had not finished her lunch. The father admitted to shaking in this fashion on many prior occasions. Skull films showed widened sutures but no fracture, and other radiographs showed old fractures of long bones and clavicle."

Whiplash shaking syndrome: retinal hemorrhages and computerized axial tomography of the brain. Carter JR, McCormick AQ. Child Abuse & Neglect 1983; 7: 279-286. Discussed by Leestma (2005) as follows: "In case 2, the mother of the baby admitted shaking the child to quiet its crying. There were multiple old rib fractures and other injuries as noted. In case 3, the child was said to have fallen off a bed 3 days prior to admission to hospital. The child at that time was limp but eventually recovered. Three days later, the child began having seizures. Examination revealed a bruise on the back and a scrape on the forehead but little else externally. There was a skull fracture and evidence of older long-bone injury, subdural hematoma, and retinal hemorrhages. Both parents admitted to having shaken the baby. In case 4, the father admitted to having violently shaken the baby during an apneic spell in an attempt to revive her. The child did not have fractures but had many bruises of the neck and abdomen, a minimal subdural hematoma, and retinal hemorrhages. The child was returned to the family. At 6 months of age, the child was readmitted to hospital with a hyphema of the right eye, fracture of the left radius, and bruising to the right face."

Shaking as a culpable cause of subdural hemorrhage in infants. Benstead JG. Med Sci Law 1983; 23: 242-244. From Dr Leestma's summary (2005): "In case 1, the child was admitted semiconscious with seizures and died. Four years after the child's death, a sibling was treated for a broken leg. At that time, the father was questioned again about the dead child and admitted to 'viciously' shaking the child at the time. In case 2, the child had been irritable for the day prior to admission and had been crying inconsolably. There was a 'scuff' mark across the right chest but no other external injury. Internally, there were hemorrhages in both psoas muscles and other retroperitoneal tissues. The father admitted to shaking the child during one of her 'fits' to quiet her. In case 3, the child was admitted to hospital posturing and unconscious. He had apparently been normal 2 hours before. The baby had a tiny bruise above the left eye and a bruise on the right upper arm. There was a sagittal sinus thrombosis found at autopsy. The mother

admitted losing her temper and throwing the child across the room to a sofa and also admitted violently shaking the child..."

Primary brain trauma in non-accidental injury. Calder IM, Hil I, Scholtz CL. J Clin Path 1984; 37: 1095-1100. Cited by Geddes et al., 2001, as being, along with Lindenberg & Freytag, 1969, Vowles et al., 1987, Hadley et al., 1989, Shannon et al, 1998, and Gleckman et al., 1999, one of the few published studies of the neuropathology of AHT.

Delayed deterioration following mild head injury in children Snoek JW, Minderhoud JM, and Wilmink JT.. Brain 1984; 107: 15-36. Presents 42 mostly school-aged children with seemingly trivial sports-related head injury had a lucid interval of hours to days followed by either sz or mental-status deterioration. Duhaime points out that in all of these cases the original injury was witnessed and the timeline was well documented, unlike child abuse cases, in which a lucid interval is never seen. See under "Time of injury -- head."

Infantile acute subdural hematoma: clinical analysis of 26 cases. Aoki N and Masuzawa H. J Neurosurg 1984; 61: 273-280. By the term "Infantile Acute SDH," (IASDH) as used in Japan, the term means "accidental," i.e. noninflicted. See below under "Head injury -- fall vs. inflicted."

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.

Neurological manifestation in abused children who have been shaken. Frank Y, Zimmerman R, Leeds NMD. Dev Med Child Neurol 1985; 27: 312-316. Summarized by Leestma (2005) as follows: "In case 1, the mother of the baby admitted to having violently shaken her. Papilledema may have been noted ... [3 month old female admitted apneic comatose with +RH and no SDH.] Studies indicated probable sagittal sinus thrombosis. In case 2, the child was said to have fallen off a kitchen table, but later parents admitted to having shaken the baby because she cried. The child had apparently not lost consciousness after the shaking but by the next day was feeding poorly and developed focal seizures. No evidence of external trauma was found on admission. The child survived with brain atrophy and neurologic deficits. In case 4, the father admitted to shaking the baby because she annoyed him. The child was hospitalized with seizures. There were bilateral retinal hemorrhages; dilated retinal veins; scattered red, round, and raised papilla-like bleed foci in the retina, and scattered flame-shaped retinal hemorrhages with foci of pallor. CT scan showed subdural effusions. Apparently, the child was returned to the family but was readmitted at 10 months of age with a fractured femur. A sibling was also treated for abuse."

The abuse of infants by manual shaking: medical, social, and legal issues. Eagan BA, Whelan-Williams S, Brooks SG Jr. J Florida Med Assoc 1985; 72: 503-505-507. Discussed by Leestma (2005) as follows: "In case 1, the baby was admitted comatose and not breathing. The baby was said to have fallen from a couch to the floor and become unconscious. The child had been admitted several times in the past for traumatic incidents allegedly including several falls. The stepfather of the baby admitted he had shaken the child to waken her from a nap and then had slapped her. Two months later, the child was readmitted unresponsive. The stepfather admitted to spanking the child and 'spinning her around,' after which she became unconscious. In case 3, the father reported that the child was placed in an armless chair while he took a nap. The child may have fallen from the chair and had become lodged between 2 other pieces of furniture. On questioning, the father admitted to have shaken the child back and forth to calm him. The child had healing fractures of the left arm. Siblings showed evidence of abusive injuries."

Neurological manifestations in children who have been shaken. Frank Y, Zimmerman R, Leeds NMD. Dev Med Child Neurol 1985; 27: 312-315.

Subdural hematomas in abused children: report of six cases from Japan. Aoki N and Masuzawa H. *Neurosurgery* 1986 Apr; 18(4): 475-477. The purpose of this work was to answer American critics (Rekate et al., 1985) who argued that these authors' earlier article (above) could have misdiagnosed cases of SBS as accidental IASDH, particularly since RH was present in all of them. Here the authors present six cases of AHT, of which five have head or face bruises and the other one had abdomen bruises. State that in Japan, all abused children have bruises, as in these cases. Suggest that the same is probably true in the US, and argues that US physicians need to reevaluate their tendency to diagnose AHT based solely on SDH + RH when there are no other injuries. A history of a short fall leading to an SDH with RH is not *per se* suspect. "These findings of bruises and skull fractures suggest a different mode of trauma from that causing IASDH, for which trivial head injury, such as falling down while sitting or standing, is exclusively responsible." The authors impliedly cast doubt on the Anglo-American theory of whiplash (citing Guthkelch); but how would they explain case 1 which had no head bruises and no history of a fall? [In this study, one pt had a history of a fall, and in that case the history was obviously spurious.] In this study, 2 had RH, 3 did not, 1 was not examined for RH. RH not detailed. All but one of these six cases are toddlers. "In conclusion, RH and SDH without external signs of trauma in Japan is usually attributed to accidental, trivial head injury (IASDH;) in the United States, it is usually attributed to child abuse. It is important to be aware of these tendencies when investigating the cause of SDH in infants." Case 1 age 1 yr 11 mos presented comatose after becoming drowsy and suffering a grand mal at home. Exam showed no bruises on the head or face. Had multiple bruises on the abdominal region. Had anisocoria / larger R pupil. + R RH. Craniotomy evac 30 gm clotted R SDH. Pt recovered with moderate L hemiparesis. Case 2 a 3 y o emaciated boy presented obtunded with fever and grand mal. Bruises on face and head. +RH. Sutural diastasis. Bilat SDH. Brown CSF. Treated nonoperatively, remained tetraplegic. Case 3 a 1 yr 9 mos boy history of falling off a table, had a sz several hours later. Kept at home in a comatose state for one month. No skull fx. CT sh high-density convexity SDH w interh. Observed. Remained comatose. Case 4 An emaciated 3 yo boy w hx of battering, LOC. DV. Multiple burns. No RH. No fx. Bilat SDH. Resolved without operation. Case 5 an infant age 3 mos w multiple bruises of the head and face, LOC, presenting w spasticity & a tense fontanelle. No RH. Linear fx R frontal. Bilat acute & chronic SDH's + interh. Survived w drainage. Case 6 a 3 y o girl with a R forehead bruise, coma, anisocoria, large R pupil, no skull fx, CT acute R SDH, resolved nonoperatively and recovered completely w conservative man. No RH. According to Sunderland (see Wilkins, 1997), this controversy "has also now involved British neurosurgeons," citing Howard, Bell & Uttley, 1993. Aoki further answered by Rekate et al. 1989 autopsy study of five shakens showing no impact injury. (*Neurosurgery* 1989; 24: 536). But see Gardner, Br J Ophth, 2003.

Subdural hematomas in infants. (letter) Rekate HL. *J Neuros* 1985; 62: 216-217. Arguing that Aoki & Masuzawa might have missed a number of SBS cases in their 1984 work, above. That accepting a parental history of a short fall in infants or toddlers with SDH + RH fails to consider the entity of SBS, and that a child abuse workup was not done in these cases. Answered by the authors with 1986 paper, below, where the authors essentially deny the existence of SBS, at least in Japan. Answered by Rekate et al. 1989 autopsy study of five shakens showing no impact injury. (*Neurosurgery* 1989; 24: 536).

Optic nerve sheath and retinal hemorrhages associated with the shaken baby syndrome. Lambert SR, Johnson TE, Hoyt CS. *Arch Ophth* 1986; 104: 1509-1512. Collected by Leestma (2005) as one of the articles presenting cases with a history of admitted shaking (total 54 cases in the literature). As to this article, Leestma describes the case as, "The baby had apparently been vomiting for 4 days prior to admission. The child's babysitter admitted shaking the child vigorously 4 days before admission. Examination showed ecchymoses over the sternum and other injuries... At autopsy papilledema was noted in one eye. The sternal injury was said to be consistent with 3-5 days' duration, as was the age of the subdural hematoma the child had."

Magnetic resonance imaging of intracranial injuries from child abuse. Alexander RC, Schor DP, Smith WL. *J Pediatr* 1986; 109: 975-979. Contains a case report abstracted by Leestma (2005) in his collection of all the cases with histories of admitted shaking, as follows: "This 3 ½ month-old boy had been irritable and 'stiff,' with shallow breathing. The father admitted 'patting him, shaking him, and giving mouth-to-mouth resuscitation.' the child had facial bruises, retinal hemorrhages, and bilateral subarachnoid and subdural hemorrhages but no fractures..."

Traumatic retinoschisis in battered babies. Greenwald MJ, Weiss A, Oesterle CS, and Friendly DS. Ophthalmology 1986; 93: 618-625. Paper presented at the American Academy of Ophthalmology in 1985. Presents five patients aged 2m to 9m who presented with RH and stupor. One had a skull fx, the others had no soft tissue or skull injury clinically. All were nonaccidental head trauma, basically SBS. He proposes traction on the retina as the mechanism of RH, giving a separation of tissue planes within the retina. "The pathophysiological mechanism of this syndrome may be quite unlike those of other hemorrhagic retinopathies." (By this they mean that this retinopathy is not caused by increased intracranial pressure or by vasculopathy, the commonly presumed mechanisms of other hemorrhagic retinopathies.) They report their followup observation of these patients over a period of years. See above under "RH -- In General" for further detail. Selected cases (confessed shaking cases) from this article are discussed by Leestma (2005). See above under "RH -- In General."

The shaken baby syndrome: a clinical, pathological, and biomechanical study. Duhaime A-C, Gennarelli TG, Thibault LE, Bruce DA, Margulies SS, and Wiser R. Journal of Neurosurgery 1987; 66: 409-414. See below under "Biomechanics."

Head trauma due to child abuse: serial CT in diagnosis and management. Sinal SH and Ball MR. Southern Medical J 1987 Dec; 80(12): 1505-1512. Retrospective study of 24 pts admitted for head trauma, all but one less than 1 y o. 17 were SBS (71%). 12 had serial CT done. 23 had followup more than 1 year in clinic, 3 are normal. 7 have seizure dso. 7 are severely disabled by either profound retardation, spastic quadriplegia, spastic diplegia, or cortical blindness. 5 are moderate disability by mod mental retard, mild cerebral palsy, or severe behavior dso. 5 have mild dsb. Siblings of two of the pts also died of child abuse. They describe interhemispheric SDH's, citing Zimmerman as the first to recognize the association. SAH was present in 23/24 on initial CT, including seven who had no SDH at that stage. Serial CT helpful in predicting outcome. The degree of atrophy predicted long term outcome.

Diffuse axonal injury in early infancy. Vowles GH, Scholtz CL, Cameron JM. J Clin Path 1987; 40: 185-189. Cited by Geddes et al., 2001, as being one of the few published studies of the neuropathology of DAI, along with Calder et al., 1984, Hadley et al., 1989, Shannon et al., 1998, and Gleckman et al., 1999.

The shaken baby syndrome. Brenner SL, Fischer H. J Neuros 1988; 66: 660-661. Cited by the NAME Ad Hoc Committee on SBS (2001) for the proposition that the Duhaime doll model and the Gennarelli adult primate model do not approximate the human infant.

Subdural hematoma is a rare complication of OI. Tokoro et al., Neuros 1988; 22: 595

Head injury in child abuse: evaluation with MR imaging. Sato Y, Yuh WT, Smith WL, Alexander RC, Kao SC, Ellerbroek CJ. Radiology 1989; 173: 653-657. Cited by the AAP (2001a) for the proposition that CT had some false-negatives on cerebral edema, that MRI was 50% more sensitive than CT on SDH's and on parenchymal injuries, but CT did not miss any surgically treatable lesions.

Shaken impact syndrome. Bruce DA, Zimmerman RA. Pediatr Ann 1989; 18: 482-489. Proposes that all shakens have impact injury, overt or occult. Considered by Ewing-Cobbs et al (1998) and Nashelsky (1995) to be the source of this hypothesis.

Neurologic manifestations of child abuse. Benzel EC, Hadden TA. Southern Medical Journal 1989; 82: 1347-1351. Leestma (2005) abstracts three cases from this article in his collection of all the published cases of admitted shaking, as follows: "In case 5, a 9 month-old male allegedly fell from a bed. From the manner in which the information was provided in the paper, it appears that the baby's mother admitted shaking the baby in an attempt to revive it, possibly after the baby aspirated. This child apparently had no external injury but did have bilateral chronic subdural hematomas with an acute somponent on one side. It is not noted if the child had retinal hemorrhages. The child recovered with good neurologic status. In case 12, the baby was a 3 year-old female who experienced a seizure while eating and was shaken by a parent apparently after the child aspirated. The child had bilateral chronic subdural hematomas and apparently intraparenchymal brain hemorrhage. No mention is made of retinal hemorrhages. The child had multiple

periosteal elevations in several long bones and burn on the flank. the child survived in a vegetative state. Inc ase 19, the baby was a 6 month-old male who apparently choked while eating and was shaken by a parent in an attempt to revive it after aspiration. The child had a subarachnoid hemorrhage and facial bruises. No mention was made of retinal hemorrhages. The child survived but with poor neurologic status...”

Radiologic contributions to the investigation and prosecution of cases of fatal infant abuse. Kleinman PK, Blackburne BD, Marks SC, et al. NE J Med 1989; 320: 507-511. Leestma (2005) abstracts two cases from this article in his collection of all the published cases of admitted shaking, as follows: “In case 1, a 4 month-old male was admitted to the hospital in respiratory arrest with cerebral edema, probably a subdural hematoma, retinal hemorrhages, and a healing tibial fracture. The baby died after 13 hours. There had been a previous admission for facial bruising. The father of the baby admitted that he held the baby in front of him and shook and squeezed him for 1 to 2 minutes. In case 2, a 5 month-old male was admitted in coma to the hospital after apparently suddenly stopping breathing. The father admitted shaking the baby in an attempt to revive him. The baby had retinal hemorrhages, interhemispheric subdural hematoma, intraventricular hemorrhage, and several healing skeletal fractures...”

The infant whiplash-shake injury syndrome: a clinical and pathologic study. Hadley MN, Sonntag VKH, Rekate HL, Murphy A. Neurosurgery 1989; 24: 536-540. Reporting five autopsy cases with no autopsy evidence of scalp contusion or fracture. See Aoki et al., *supra*, 1984, 1986. Reichert & Schmidt in their chapter in Lazoritz & Palusci’s book state that these authors observed “spinal cord injury at the cervicomedullary junction resulting in subdural or epidural hematoma formation. Spinal cord contusions were also identified in these five patients.” [Reichert KW and Schmidt M, Neurologic Sequelae of Shaken Baby Syndrome. In: Lazoritz S and Palusci VJ, eds., New York: The Haworth Maltreatment and Trauma Press, 2001, p. 81.] Geddes et al., 2001 mention this as being one of the few neuropathology studies of AHT, the others being Calder et al., 1984, Vowles et al., 1987, Shannon et al., 1998, and Gleckman et al., 1999. This article is discussed in some detail in Leestma’s (2005) collection of all the published cases of admitted shaking. Leestma refuses to categorize the cases as ones of admitted shaking, because the article does not give any source for the information that the patients were shaken, and leaves it unclear as to whether this information was obtained by history or was inferred by the clinicians. As Leestma states, “No specific incident data regarding shaking were provided, and there is doubt if shaking occurred... Very little data from the autopsies were provided, but it is said that none of the babies suffered a cranial impact. All of the infants had retinal hemorrhages, subdural hematomas, and presumed evidence of spinal injury.” He points out that these are the only reported shaken cases that had any documented neck injury. [The Geddes cases are not included in his review. –JKR] This paper is discussed in Leestma (2005), saying that it could not be determined where the authors got their information that the babies had been shaken, and thus that the fact of shaking may be just a clinical inference in these cases, no confession or other objective evidence being cited. This paper is cited by Hymel (2005) as one of the papers that support craniocervical apnea – secondary HIE as a common sequela of pure shaking.

Incidence of impact trauma with cranial injuries ascribed to shaking. Alexander R, Sato Y, Smith W, Bennett T. Am J Dis Child 1990; 144: 58-60. Reporting 24 allegedly shaken infants of which 12 had no clinical evidence of impact injury. Nine autopsied -- five showing no impact.

Serial abuse in children who are shaken. Alexander RC, Crabbe L, Sato Y, Smith W, Bennett T. Am J Dis Child 1990; 144: 58-60. See also "Neuroimaging" by Ewing-Cobb et al, 1998, which likewise emphasizes the prevalence of previous injury.

Late ophthalmic manifestations of the shaken baby syndrome. Journal of Pediatric Ophthalmology & Strabismus. 1990; 27(6): 299-303

Diagnostic imaging in infant abuse. Kleinman PK. AJR 1990; 155: 703-712. A literature review finding that the average age of shakens was six months.

Morphology and neurophysiology of focal axonal injury experimentally induced in the guinea pig optic nerve. Tomei G, Spagnoli D, Ducati A, Landi A, Villani R, Fumagalli G., Sala C, Gennarelli T. Acta

Neuropathologica 1990; 80: 506-513. Cited by Levin (2001) along with Smith, Andrus, 1998 as the only animal model work so far done on SBS.

Shaken baby syndrome. Spaide RF, Swengel RM, Scharre DW et al. Am Fam Phys 1990; 411: 1145-1153. Leestma (2005) includes a case report in this article in his collection of all the published cases of an admitted shaking history, as follows: "This child may have fallen 6 weeks prior to admission from her crib to the floor. Subdural effusions were found on admission. The mother admitted that she shook the baby after she cried all day long and after, finally having fallen asleep, she was awakened by noises made by siblings and a church bell ringing. The mother lost her temper and was enraged and grabbed the baby and shook her. [Had RH and possibly other injuries].

Vitreoretinal traction and perimacular folds in the eyes of deliberately traumatized children. Massicotte SJ, Folberg R, Torczynski E, Gilliland MGF, Luckenbach MW. Ophth 1991; 1124-1127. The authors reviewed the clinical and autopsy findings in three SBS victims who had perimacular folds. Two had impact trauma, one was shaken only. In each case, the vitreous had partially separated from the retina but remained attached to the internal limiting membrane at the apices of the folds and the vitreous base, implicating traction in the pathogenesis of these folds. Gives specimen photographs including two histologic photos, one showing the vitreous and internal limiting membrane attached to the crest of the fold and separated from the retina within the valley by a large subhyaloid hemorrhage. Also showing the vitreous remaining attached to the internal limiting membrane while the internal limiting membrane is pulled away from the retina. See also under "histology." A case from this article is abstracted by Leestma (2005) in his collection of all the published cases with an admitted history of shaking, as follows: "In case 2, the babysitter of the child said she had rolled off the couch and landed on her milk bottle or may have slipped while in the bathtub, hitting her head. [9 month old] The 12 year-old sitter later admitted to shaking the infant and throwing her to the floor. There were skull fractures and multiple injuries... There were optic nerve sheath hemorrhages; both eyes had perimacular folds and hemorrhages to the ora serrata in the eyes."

Battered child syndrome: cerebral ultrasound and CT findings after vigorous shaking. Zepp F, Bruhl K, Zimmer B, et al. Neuropediatrics 1991; 23: 188-191. Two cases from this article are abstracted by Leestma (2005) in his collection of all the published cases with an admitted history of shaking, as follows: "In case 1, the child had apparently been healthy until the day before admission, when she appeared sleepy and refused food; the next morning, the child could not be aroused. Ultrasound imaging indicated intracranial bleeding and a possible lesion in the medulla. [Also RH] The parents admitted they had vigorously shaken the [4 month-old] baby. In case 2, the parents admitted to repetitively having shaken the baby because of crying. There were chest injuries. No apparent subdural hematoma was noted, though the brain was edematous and there was increased intracranial pressure and bilateral retinal and vitreous hemorrhages."

Head injury in very young children: mechanisms, injury types, and ophthalmologic findings in 100 hospitalized patients younger than 2 years of age. Duhaime A-C, Alario AJ, Lewander WJ, Schut L, Sutton LN, Seidl TS, Nudelman S, Budenz D, Hertle R, Tsiaras W, Loporchio S. Pediatrics 1992 Aug; 90(2): 179-185. 100 consecutively admitted head injury patients under 24 months. 24 inflicted, 32 possibly inflicted, 44 accidents. All had ophth. RH were found in 10: 9 abuse and one fatal high-speed MVA. All 10 RH pts had SDH. Discusses translational versus rotational mechanisms. Short falls translational are not of great clinical consequence, even when they cause fx, unless an EDH occurs. Higher falls translational cause complex, depressed, or basal fractures or multiple fxx as well as focal brain contusions and SAH; unless the translational force is extreme, damage is predominantly focal and recovery of global neurologic function is usually rapid. In contrast, it has been shown experimentally and clinically that more significant diffuse brain injury results from the introduction of a significant **angular component** to the head's deceleration. Angular (rotational) deceleration leads to much more brain deformation and shear strain...and at progressively greater angular decelerations the phenomena of concussion, SDH, and DAI will occur. (This is taken from Genarelli's work on monkeys, but here explicitly transposed to humans.) SDH were uncommon in accidental injuries, occurring in only three children in MVA. In contrast, SDH and diffuse SAH are common in inflicted injury in very young children, occurring in 13 of 24 patients in this series. ... "The mechanism postulated is that of a child being held by the perpetrator who shakes, swings, or throws the child,

the head thus moving in an arc, stopping abruptly against a surface. Previous autopsy studies and biomech using infant models suggest that shaking alone does not generate sufficient deceleration forces to cause the SDH and brain injuries seen in these children (citing the 1987 biomechanical study.) The frequent radiologic or clinical findings of blunt impact in series of “shaking” injuries corroborate this conclusion, as does the rarity of an *unsolicited* history of shaking. [italics added --JKR] For these reasons, we now refer to this syndrome as the ‘**shaken impact syndrome**.’ The presence of fractures and bruises will be determined by the surface against which the rotating head decelerates.”

Gives Table 4 which lists the injury types that can be expected from various common mechanisms:

Fall under 4 ft	Concussion Linear fx EDH Ping-pong fx (if hit corner) ?Depressed fx
Fall over 4 ft	Above injuries Depressed fx Basilar fx Multiple fxx SAH Contusion ?SDH ?Stellate fx
MVA	Above injuries SDH DAI

“...[I]t is clear that RH can occur under a variety of circumstances, including vaginal delivery, spontaneous SAH, systemic HTN, intracranial HTN, thoracic or abdominal trauma, and in-hospital resuscitation. Whether superimposed hypoxia or ischemia with reflow exacerbates the finding remains unknown. Traumatic retinoschisis resulting from acceleration/deceleration forces applied to the eye has also been postulated as a mechanism for RH. The latter may be particularly relevant in very young children because of the more solid consistency of the vitreous body in the infant and the stronger adhesions at the vitreoretinal interface. Threshold values for the degree of deceleration required to result in RH have not been established. Since this study was completed we have seen three additional pts with well-witnessed accidental head injuries who had acute RH...”

Shaken Baby Syndrome: Inflicted Cerebral Trauma. American Academy of Pediatrics, Committee on Child Abuse & Neglect. Pediatrics 1993 Dec; 92(6): 872-875. Includes shake + impact, as long as shaking was a component.

When uncomplicated skull fxx are excluded, Billmire & Myers found that 95% of all serious intracranial injuries in infants are due to child abuse. Bruce & Zimmerman document that 80% of deaths from head trauma in infants and children younger than 2 years of age were the result of nonaccidental trauma. SBS symptoms are often subtle. The more severe cases often have delayed presentation. Unilateral or bilateral RH are present in 75% - 90%.

"SBS is characterized as much by what is obscure or subtle as by what is immediately clinically identifiable."--because symptoms may be subtle.

The pathophysiology of infant subdural hematoma Howard MA, Bell BA, and Uttley D. (British radiologists). Br J Neuros 1993; 7: 355-365. Has been cited to support the view that falls can cause fatal SDH. Also see Aoki, 1986; Greenes, 1998; Plunkett, 2001. Retrospective review of the charts and CT scans on 28 babies up to 18 mos over a 20-year period. All had findings consistent with an impact injury. "Our findings do not support shaking as the only cause of infant SDH formation and also suggest that non-accidental injury is a less common cause of SDH than it is believed to be." Cited by Wilkins, 1997, to the effect that some child pts are “at increased risk” for SDH caused by minor injury, and also that non-accidental injury is possibly overdiagnosed.” This article has been roundly condemned in the US literature.

Inflicted versus accidental head injury in critically injured children. Goldstein B, Kelly MM, Gruton D, Cox C. Critical Care Medicine 1993; 21: 1328-1332. Cited by Hymel et al., 2007 for the proposition that inflicted children do much worse than accidental children.

Permanently damaged: long-term followup of shaken babies. Fischer H and Allasio D. Pediatrics 1994; [[33(11)]]?: 696-698

Ruptured vascular malformation masquerading as battered/ shaken baby syndrome: a nearly tragic mistake. Weissgold DJ, Budenz DL, Hood I, Rorke LB. (Penn). Surv Ophth 1995 May-Jun; 39(6): 509-512. A seven week old infant with acute intracranial hemorrhage and ONSH discovered at autopsy. NP later showed an AVM.

Fatal intramuscular bleeding misdiagnosed as suspected nonaccidental injury. Wetzel RC, Slater AJ, Dover GJ. Pediatrics 1995; 95(5): 771-773. (have) Vitamin K deficiency due to exclusive breast-feeding following a home birth, had small subdurals, +RH, bruises of buttocks & legs, brain death. Admitted as SBS. See under RH -- other.

Crush injuries to the head in children. Duhaime A-C et al. Neurosurgery 1995; 37: 401

Abusive head trauma: the relationship of perpetrators to their victims. Starling S, Holden JR, Jenny C. Pediatrics 1995 Feb; 95: 259-262 See above under "Epidemiology" for summary of this article.

Role of apnea in nonaccidental head injury Johnson DL, Boal D, Baule R.. Pediatr Neurosurg 1995; 23: 305-310. Apnea plays a major role in the pathophysiology and ACCOUNTS FOR THE POOR OUTCOME IN THIS SUBGROUP. This conclusion is disputed in Duhaime's review article, pointing out that some of the ischemia is unilateral and that the chronic CT picture does not match that of near-drownings etc. See Hymel, 1995 for a brief summary of the papers supporting craniocervical apnea – HIE in SBS. This article predates those of Geddes, Whitwell by six years and anticipates their concept.

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.

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. But note that they studied the onset of "symptoms." (any symptoms, including post-traumatic sz. But the symptoms we are interested in are apnea and unconsciousness. Sz by themselves can be variably delayed. See under "Time of injury -- head.")

Long-term outcome in infants with shaking-impact syndrome. Duhaime A-C, Christian C, Moss E, Seidl T. Pediatric Neurosurgery 1996; 24: 292-298

Diffuse axonal injury: its mechanism in an assault case. Takeshi Imajo MD, Am J Forens Med Path 1996 Dec; 17(4): 324-326. This adult male was multiply kicked in the side of the head by assailant, survived 13 days. Had DAI with a microscopic lytic lesion in the corpus callosum. Dr. Imajo posits low-acceleration coronal rotational injury with freely moving head.

Shaken babies -- some have no impact injuries. Gilliland MGF, Folberg R. J F S 1996; 41: 114-116. Autopsy study of 80 fatal head injuries in children. Nine had no impact injury.

Radiographic Atlas of Child Abuse: a case studies approach. Harris VJ, Lorand MA, Fitzpatrick JJ, Soter DK. New York: Igaku-Shoin, 1996. ISBN 0-89640-258-4. Gives images from lots of shaken babies, reversal sign, skull fractures, SDH's, EDH, etc. See above under "Fractures -- In General" for discussion. A good book.

Ocular and cerebral trauma in non-accidental injury in infancy: underlying mechanisms and implications for pediatric practice. Green M et al. Br J Ophth 1996; 80: 282-287. Cited by Dr. Levin (2000a) for the proposition that "The overwhelming preponderance of medical opinion concludes that the association of characteristic brain injuries and retinal haemorrhages is diagnostic of SBS."

Disappearing subdural hematomas in children. Duhaime A-C, Christian C, Armonda R, Hunter J, Hertle R. Pediatric Neurosurgery 1996; 25: 116-122

Commentary on controversies in shaken baby syndrome and on Gilliland MGF and Folberg R: Shaken babies: some have no impact injuries. Taff M, Boglioli L, and DeFelice J. JFS 1996; 41: 729-730

Techniques for developing child dummy protection reference values. Klinich KD, Saul R, Auguste G, Backaitis S, Kleinberger M. Report by the child injury protection team, NHTSA, October, 1996. Cited in Cory and Jones, 2003, as postulating a 50% concussion probability at only **4,000 sec(-2)** peak angular acceleration for a pediatric auto vs. pedestrian model.

Evidence from proton magnetic resonance spectroscopy for a metabolic cascade of neuronal damage in shaken baby syndrome. Haseler LJ, Arcinue E, Danielsen ER, et al. Pediatrics 1997; 99: 4-14. Leestma abstracts a case from this article in his 2005 collection of all the published cases of admitted shaking, as follows: "In case 1, the child was said to have suffered a seizure after a 3-foot fall from a table, but one parent was reported to have witnessed the other parent severely shaking the infant. The child apparently was initially conscious but then went on to arrest. An MRI showed extradural and subdural collections, both of which eventually resolved without treatment. [Had cerebral edema, no RH]."

Shaken adult syndrome. Derrick J. Pounder. Am J Forens Med P 1997; 18: 321-324. This oft-cited case report of the 30 year old Palestinian political prisoner interrogated for three days who died of a large SDH *with mass effect*. No head impact at autopsy or by history. No neck injuries. No confession or judicial finding of shaking. Had severe chest bruising, no head bruising. Had a space-taking unilateral SDH with midline shift and brain stem herniation. Had retinal hemorrhages. Had brain swelling. Supposedly had DAI. (The mass effect notation is significant. Note Dr. Alexander's and Dr. Kirschner's and Dr. Spivack's responses to Plunkett's 2001 article on fatalities from witnessed falls, in which they point out that Dr. Plunkett's toddlers all died of mass-effect hematomas, rather than TAI; thus distinguishing those cases (and this one?) from SBS.) This case is cited and relied on by Mary Case and colleagues in the NAME position paper on AHT, 2001. Important to note Jenniann Geddes' and Helen Whitworth's scholarly criticism of the diagnosis of DAI made by Pounder and Kirschner in this case, and distinguishing the case on the ground of a space-taking lesion. Am J Forens Med P for September 2003, below. For another alleged case of shaken adult, see case report by Carrigan et al., 2000, below, a no-good report.

The whiplash shaken infant syndrome: has Caffey's syndrome changed or have we changed his syndrome? Lazoritz SL, Baldwin S, Kini N. Child Abuse & Neglect 1997; 21(10): 1009-1014. Caffey's original complex of findings diagnostic of the syndrome was "intracranial and intraocular hemorrhages in the absence of signs of external trauma or skull fracture, associated traction lesions of the long bones and no history of trauma." [Caffey's point was that "direct trauma" was not necessary to produce brain damage or metaphyseal fractures. JKR] Points out that the subsequent publications of Bruce & Zimmerman (1989) and Mehl (1990) have included head-impact cases within the syndrome. Also points out that long-bone fractures are not a feature of the modern cases, while rib fractures are. Presents a retrospective review of 71 nonaccidental subdural hematoma patients under 3 years of age admitted to Children's Hospital of Wisconsin 1990-1995 and compares them to the 27 cases originally collected from various sources by Caffey. Breaks out the comparison into various categories:

Fractures

In Caffey's material all 27 had long bone fractures (either CML or SPNBF).

In our cases, we found 43 nonskull fractures in 23 patients as follows:

Fresh rib	23%
Healing rib	18%
Tibia	16%
Radius	9%
Ulna	9%
Femur	7%
Humerus	11%

History

In Caffey's material usually there was no history of trauma. [In some there was a history of shaking; Lazoritz et al do not mention this. JKR]

In our 71 patients the histories were as follows:

No trauma	34%
Fell more than 3 feet	7%
Fell less than 3 feet	18%
Shaken	13%
Hit on head	13%
Dropped	10%
Thrown	3%
Hit on body	1%
Unk	1%

Perpetrators

In Caffey's material had

Nurse	55%	15 cases
Mother	15%	4 cases
Both parents	7%	2 cases
Sibling	4%	1 case
Unk	15%	5 cases

In our material had

Father	33%
Boyfriend	20%
Mother	7%
Other	42%

Discussion says Caffey obviously recognized that direct trauma (battering) was the usual cause of brain damage and fractures in abused infants. His purpose was to suggest that sometimes there was another cause, which might not be recognized or reported. "It appears that we ["we" being the recent authors] have, indeed, changed Caffey's syndrome and broadened it to include all forms of abusive head injury, including those caused by shaking alone, those caused by impact, and those caused by both. It is our opinion that ... it should be acknowledged that Shaken Infant Syndrome is a syndrome of severe head injury to infants caused by either shaking alone or by shaking plus impact." 1013. (Compare the formulation of Duhaime et al. (1998) that the term "remains a useful diagnostic paradigm," even though its original content correlates poorly with modern findings.) Comment that the change in perpetrators from the nurse and mother to the boyfriend and father reflects the disintegration of the family.

Outcome after severe non-accidental head injury. Haviland J, Ross-Russell RI. Arch Dis Child 1997; 77: 504-507. Reviewed records of 15 shaken babies and a control grp of 10 acc head inj who survived long term 3 mos to 3 yrs f/u. 12 (80%) had RH (11 bilat, 1 unilat). 8 (53%) had ev of external head trauma (either bruises or skull fx). 13 had SDH, 9 had suspicious soft tissue inj, 6 had extracr fxx. Outcomes: 2

d, 9 major neurol, 3 mod seq, 1 nl. As compared to the control group: 1 d, 1 severe handic, 1 mild, 6 normal. Cited by Hymel et al., 2007 for the proposition that inflicted children do worse than accidentals.

Cerebrovascular response in infants and young children following severe traumatic brain injury: a preliminary report. Adelson PD, Clyde B, Kochanek PM, Wisniewski SR, Marion DW, Yonas H. *Pediatric Neurosurgery* 1997; 26: 200-207. (have) Retrospectively reviewed the cerebral blood flow data and CO₂ vasoreactivity data on 30 severe TBI children under 8 admitted with GCS under 8. Age range 1 month to 8 years, average age 2.2 years (a much younger average age than the previous studies and thus more oriented to AHT, which is unique to the youngest age group). Labelled as a "preliminary study" because of short followup (mostly 48 hours). Poor outcome was seen in all ch under 1 year. It was previously known, albeit on the basis of a very small number of (published) patients, that the youngest children tend to present with poor CBF and not to improve. This is key to their poor outcome. (Historically and in this study, children under 4 have worse outcomes in TBI.) They review the previous literature on CBF in children (four previous papers). These had average ages of 7, --, 14, and 8. These included few children under 2. Concludes that for infants and young children, CBF under 20 ml/100g/min at any time always indicated a poor outcome. Over 55 ml/100g/min, usually seen as a secondary increase after some treatment, was assoc w more favorable outcome. CO₂Vr was markedly impaired in infants and y ch w a poor outcome. Includes some comments on the mechanism of malignant swelling. Notes the "seminal" work of Marmarou et al. (1996) indicating cytotoxic edema and not vascular engorgement. And indeed, in this present work, higher CBF was associated with a good outcome. Seeming (to me) to cast doubt on the view that vascular engorgement is the mechanism of swelling in this pop.

Head injury -- abuse or accident? Wilkins B. *Arch Dis Child* 1997; 76: 393-396. See under "Fall vs. Inflicted. Cited by Dr. Levin (2000a) for the proposition that "[T]he overwhelming preponderance of medical opinion concludes that the association of characteristic brain injuries and retinal haemorrhages is diagnostic of SBS."

Chapter 11, Forensic Neuropathology, by Jan E. Leestma, in Julio H. Garcia, ed., *Neuropathology: The Diagnostic Approach*. St. Louis: C.V.Mosby, 1997. pp.475-527. (have) Be careful: Geddes et al. (2001) point out that much of what is said in this book pertains to older children, not infants, particularly with regard to subdurals, which are characterized in a way typical of space-taking lesions seen in older children, (the subdurals in infants being thin and non-space-taking) and epidurals, which are almost never seen in AHT.

Shaken baby syndrome: report on four pairs of twins. Becker JC, Liersch R, Tautz C, et al. *Child Abuse & Neglect* 1998; 22: 931-937. Leestma (2005) includes three cases from this article in his collection of all the published cases with an admitted history of shaking, as follows: "In case 1, the child was reported to have been crying inconsolably all day, with frantic sucking actions. In the evening, the father found the baby not breathing and slapped him on the back and tried mouth-to-mouth resuscitation. He later admitted ethat he had 'shaken the baby too much' during the incessant crying of the baby. No external signs of trauma were noted. Extensive intra- and pre-retinal bleeding was noted in both eyes. In case 2, the baby had several episodes of vomiting and making 'strange noises.' The mother admitteed to shaking the child because she thought he was suffocating. She said she held the bayb by the chest and shook him. the child became cyanotic but respopnded to mouth-tomouth resuscitation but then had respiratory failure again. Apparently both parents had shaken the child. There were no external sings of trauma. The child had a leg fracture and other injuries. In case 4, the parents of the child were worried because the child did not awaken. The mother then shook the baby several times. The child then awakened but then lost consciousness and had a seizure. The mother said that she could not imagine that her shaking was that violent and denied intent to harm the child. [Had SAH and ICH]."

Nonaccidental head injury in infants -- the "shaken-baby syndrome." Duhaime AC, Christian CW, Rorke LB and Zimmerman RA. *NE J Med* 1998 Jun 18; 338(25): 1822-1829. (review article). See Letters, next below. "Several patterns of clinical and radiographic findings allow a definitive diagnosis [of the shaken-impact syndrome]. These include

a history of trivial or no trauma,

acute subdural hemorrhage, and
unexplained extracranial bony injuries or
clearly inflicted soft-tissue injuries; [or]

a definite history of no possibility of trauma with
clear physical or radiologic evidence of head impact with subdural hemorrhage."

The "shaken-baby syndrome." (letter). VJM DiMaio. NE J Med 1998 Oct; 339(18): 1329. "If one has proof of impact, why hypothesize that the child was shaken? There are no lesions to prove the child was shaken. You cannot base such a judgment on self-serving statements by the person who inflicted the injuries. Adding the word 'impact' to the term 'shaken-baby syndrome' does not prove the existence of the entity or justify the retention of this term. Let us simply drop both the term and the concept of the shaken baby and face the fact that the injuries are due to the impact of being slammed, swung, or thrown against a hard surface...." See "authors' reply," next below.

Authors' reply "Although the terminology and mechanisms may be debated, the entity that has been known as the shaken-baby syndrome clearly exists as a useful diagnostic paradigm. Many clinicians, pathologists, and child advocates are convinced that shaking is a part of this syndrome because of their collective experience with histories and confessions in which shaking was described...."

The shaken baby syndrome presenting as ALTE. (letter). Altman RL, Kutscher ML and Brand DA. NE J Med 1998 Oct; 339(18): 1329-1330. Because SBS and ALTE can have similar presentations clinically, they performed ophthalmoscopy on 75 infants presenting with ALTE. They found RH in 4 and finally diagnosed SBS in 5. Duhaime et al. reply that "The possibility of child abuse needs to be included in the differential diagnosis for all infants with altered mental status, subtle neurologic signs, and unexplained apnea or vomiting." In this regard, see also the article on missed AHT by Carol Jenny et al., listed below under "Unclassifiable" articles and discussed in the AAP Technical Report of 2001. See also under "Abdominal trauma" for the difficulty of recognizing abdominal trauma in preverbal children.

Anatomy of the shaken baby syndrome. Lancon JA, Haines DE, Parent AD. (U Miss) Anat Rec 1998 Feb; 253(1): 13-18. Mostly a recital of received doctrine, it does state that "the most severe brain injuries result from the addition of a forceful impact of the infant's or child's head against a firm surface. But then it goes on to emphasize the weak-neck-heavy-head doctrine, which doesn't match up with this statement.

Second-impact syndrome. Robert C. Cantu. Clin Sports Med 1998; 17: 37-44. Review art. 5 boxers who suffered severe head blows with altered cons, then boxed again hours to 2 days later and collapsed. Half had small SDH's. He also discusses another entity, the malignant brain edema syndrome, in which there is late collapse but no second impact. Rev in Child Ab Quarterly July 1998. Comment by Cindy Christian at the Second National Shaken Baby Conf 1998: SIS is extremely rare. She was only able to dig up seven reported cases. It requires certain clinical criteria: (1) the initial injury was clinically treated, (2) the interval was continuously symptomatic, (3) the second injury was minor and witnessed, (4) the second injury was immediately followed by abrupt fatal deterioration. Kent Hymel reviewed this literature under TADDS at the San Diego Conference in 1999.

Shaken baby syndrome/shaken impact syndrome. Robert M. Reece and Robert H. Kirschner, National Information, Support and Referral Service on Shaken Baby Syndrome, Summer 1998, pp 4-5. Estimates of the incidence range from 600 to 1400 per year in the US. Age from nb to 4 yrs; "The vast majority of SBS/SIS occur before the infant's first birthday and the average age of the victim is between 3 and 8 months of age. The time of shaking varies, usually ranging from 5 seconds to 15 or 20 seconds. The number of shakes ranges between 2 to 4 per second. ... The infant stops crying and stops breathing."

"The usual trigger for shaking a baby is inconsolable crying in the infant. Frustrated by attempts to console the baby, the perpetrator loses control and grabs the infant, either by the chest, under the arms, or by the arms, and violently shakes the baby."

Shaking and the sudden deceleration of the head at the time of impact does several things:

1. The bridging veins tear
2. The brain strikes the skull
3. Axons can be broken, shearing
4. Lack of oxygen during shaking
5. Damaged nerve cells release chemicals.

"The combined effect is massive traumatic destruction of the brain tissue, leading to immediate brain swelling and causing enormous increases in the pressure within the skull. This swelling compounds the problem, since swelling causes compression of the blood vessels and decreases the oxygen supply to the brain. "IT IS THESE INJURIES TO THE BRAIN, NOT THE BLEEDING UNDER THE DURA OR THE ARACHNOID MEMBRANES, THAT CAUSE THE SIGNS, SYMPTOMS, AND COURSE OF SHAKEN BABY/ SHAKEN IMPACT SYNDROME." (emphasis in original)

"RH seen in other conditions are usually closer to the surface, so-called preretinal hemorrhages, and resolve quickly. RH are not seen as the result of CPR or accidental short falls."

"A number of other lesions are variable findings in SBS/ SIS. These include skull fractures resulting from the impact when the infant is thrown against a hard or soft surface; fractures of the posterior arcs of the ribs...; fractures of the clavicles; and fractures of the long bones."

Symptoms vary depending on the length of shaking and whether thrown against surface. From irritability lethargy to convulsions vomit coma to death. "All of these symptoms are caused by generalized brain swelling secondary to trauma with these symptoms beginning immediately after the shaking and reaching their peak within 4-6 hours."

Editorial, Lancet 1998 Aug 1; 352: 335. Reviewing the NEJM Duhaime article and discussing nomenclature.

Neuroimaging, physical, and developmental findings after inflicted and noninflicted traumatic brain injury in young children. Ewing-Cobbs L, Kramer L, Prasad M, Canales DN, Louis PT, Fletcher JM, Vollero H, Landry SH, Cheung K. Pediatrics 1998 Aug; 102(2): 300-307. A prospective longitudinal hospitalization study of 20 inflicted and 20 noninflicted brain injury children aged 0 to 6 years. Followup time averaged 1.3 months. Conclusions: characteristic features of INFLECTED brain injury patients were (1) preexisting brain injury, (2) SDH, (3) seizures, (4) RH, (5) mental deficiency. 45% of the pts with inflicted injury had preexisting cerebral atrophy or chronic SDH. RH was seen in 14 inflicted and 0 noninflicted, BUT ophthalmic examination was not performed in some of the noninflicted group. Reported injury mechanisms from Table 2:

	Inflicted	Noninflicted
Dropped	5	0
Fell under 4 ft	5	2
Fell over 4 ft	0	3
MVA	0	12
Hit by moving object	0	3
No history	10	0

Following acute CT or MRI findings from Table 3:

	Inflicted	Noninflicted
SDH	16	9
EDH	0	4
ICH	1	6
Shear	0	4
Soft-tissue swelling cranium*		
Present	11	16
Absent	9	4
Edema/infarcts	5	6
Atrophy	7	0
Skull fx		
Linear	3	2

Comminuted		0		1
Diastatic		2		4
Depressed	3		3	
Multiple		1		5
Basal		0		1

Bodily injuries from Table 4:

Bruises		
Face	11	14
Torso	6	2
Skeletal fractures		
Ribs	3	1
Clavicle	1	0
Scapula	1	0
Forearm	1	1
Tib fib	4	0
Femur	2	1
C-spine	0	3
Face	0	3

* The authors relate this finding to the shaking-impact controversy. They state that the finding of soft tissue swelling over the cranium in 55% of the inflicted children “provides some support for the ‘shaking-impact’ mechanism of injury proposed by Bruce and Zimmerman. (citing Bruce & Zimmerman, 1989.) However, the remaining child did not show overt signs of assault involving the cranium. Because accurate histories of the assault are difficult or impossible to obtain, it is unclear whether child with no overt signs of assault involving the cranium received contact trauma to the head. There was no association between skull fracture and/or soft tissue swelling and either parenchymal involvement or retinal hemorrhage.” (In other words, a significant number of RH-SBS-type patients did not have scalp/skull impact findings on CT.)

Early neurobehavioral outcomes were good in only 20% of the inflicted versus 55% of the noninflicted group. The authors conclude that the grim outcome data for AHT support the need for early identification of these patients for counselling and patient-management purposes. They urge early ophthalmologic evaluation for this purpose.

“Our findings differ from those of Billmire and Myers, who...found that 95% of children with serious intracranial injury or hemorrhage had inflicted injuries.” “Skull fractures, soft tissue swelling..., skeletal fractures, and bruises/lac occurred with comparable frequency in both groups.” “Subdural and subarachnoid hemorrhages, which occurred in both groups, did not independently indicate the presence or absence of assault. However, in noninflicted TBI, SDH were most common in MVA and were not associated with either falls or crush injuries.” (emphasis added --JKR. See Duhaime et al., 100 children, 1992; Hymel, 1998.)

This paper was selected by Dr Reece as one of the top ten SBS papers of the past ten years at the 2006 National Shaken Baby Conference. In his review, Dr Reece brought out the following additional information from the article:

All the patients were moderate to severe TBI.

Alleged perpetrators: 30% biological fathers, 5% biological mothers, 10% other relatives, 15% boyfriend or girlfriend, 10% babysitters, 30% unknown.

Fractures: the inflicted were mostly ribs & extremities, the accidentals were mostly facial.

I am going to leave out other data Dr Reece brought out.

Abusive head trauma? A biomechanics-based approach. Kent P. Hymel, Faris A. Bandak, Michael D. Partington, Ken R. Winston. *Child Maltreatment* 1998 May; 3(2): 116-127. See under “Head injury -- fall vs. inflicted.” These authors import results from the transportation industry and military studies of head-injury mechanics. This world is divided into contact and noncontact injuries. As far as noncontact injuries or injuries that are partly noncontact: large subdurals (i.e. not confined to the area under an impact site or fracture) are caused by anteroposterior angular acceleration that is sudden and short. DAI is caused by slower angular acceleration in the coronal plane. DAI leads to immediate LOC.

Social and behavioral effects of traumatic brain injury in children. Andrews TK, Rose FD, Johnson DA. Brain Injury 1998; 12: 133-138

Glutaric Aciduria Type 1. Evaluation of abusive head trauma and GA1. Research presentation at the Second National SBS Conference, 1998. Steve Goodman MD and Andrew Sirotnak MD. 9/14/1998. (see below)

Intracranial aneurysms in infants and children. Allison JW, Davis PC, Sato Y et al. Pediatr Radiol 1998; 28: 223-229. The experience of four teaching hospitals over 25 years. 21 cases. Such aneurysms as a cause of intracranial bleeding are very rare and easily ruled in by imaging. Confusion with child abuse is therefore unlikely. See 00-7228.

Serial radiography in the infant shaken impact syndrome. Dias MS, Backstrom J, Falk M, Li V. Pediatric Neurosurgery 1998 Aug; 29(2): 77-85. One "chronic" subdural collection was absent on the first scan but appeared on a second scan performed 17 hours later.. These findings challenge some of the current dogma about the timing of radiographic changes following abuse... [This paper was criticized by scholars for lack of definiteness on the actual time of injury. -JKR]

Mail survey on SBS demographics 1998, by the Support and Referral Service on SBS of the Child Abuse Prevention Center. Published in NISRS/SBS Newsletter for Autumn 1998. Dawn Fitzpatrick mailed survey to all the child fatality review teams in the US in 1998. Responses from thirty states. 337 deaths. In 247 cases the relationship of the perpetrator to the victim was discovered:

62%	parents
20%	boyfriends
14%	unrelated caregiver
9%	stepparents
1%	grandparents

Also gives pie charts for age and sex of both victims and perpetrators:

Victims	57%	male	avg victim age	8.9 mo
Perps	70%	male	avg perp age	24 yr

Compare Starling et al., 1995.

Shaken baby syndrome: report on four pairs of twins. Becker C, Liersch R, Tautz C, et al. (Germany). Child Abuse & Neglect 1998; 22: 931-937. Four pairs of twins who were shaken. Most shakers said that inconsolable crying was the precipitating event. Three of the sets of twins had been born prematurely and had been hospitalized for weeks after birth. The authors suggest that impaired bonding was a risk factor for shaking.

Subdural hematomas in children under 2 years: accidental or inflicted? A ten year experience. Tzioumi D and Oates RK (Australia). Child Abuse & Neglect 1998; 22: 1105-1112. Saw 38 children. 21 nonaccidental, 15 accidental, 2 natural disease – nontraumatic etiology. Ophthalmoscopy was done on all 21 of the nonaccidental and on 5 of the accidental: of these, 16 (84%) of the nonaccidental had RH, none of the accidental. RH were bilateral in 7, unilateral in 6, and absent in two shakers. Patients were divided into "shaking" and "impact" groups depending on skull fractures or a history of impact. Compare the British radiologists Howard, Bell & Utley, 1993 on the prevalence of accidental SDH in infants. Compare Aoki's 1984 Japanese-American exchange under "Head injury -- fall vs. inflicted."

Non-accidental head injury in infants: the "shaken baby syndrome" revisited. Edward E. Conway, jr. Pediatric Annual 1998; 27: 677-690. Review article. Dr. Reece says it covers the subject well.

Axonal injury -- a diagnostic tool in forensic neuropathology? Oehmichen M, Meissner C, Schmidt V et al. FSI 1998; 95: 67-83. (See under "Head injury -- fall vs. inflicted.") This is not a child abuse paper. Also note that AI is NOT the same thing as DAI. They did H&E and beta-amyloid precursor protein (APP) staining on 252 mostly adult brains which suffered penetrating and nonpenetrating trauma or non-trauma with or without shock/hypoxia/brain death. The study did not demonstrate any difference between direct-blow and accel-decel injured brains. All trauma had 75% incidence of AI in the pons by APP staining. A

minimum of 3 hours survival was needed to demonstrate by APP in the pons. Finding of localized AI in the pons was nonspecific.

Axonal injury and the neuropathology of shaken baby syndrome. (Berlin) Shannon P, Smith CR, Deck J, Ang LC, Ho M, Becker L. Acta Neuropathologica (Berlin) 1998 Jun; 95(6): 625-631. 14 autopsies of shaken babies who had no skull fracture. All cases had beta-APP+ axons, but so did 6 out of 7 children dying of nontraumatic HIE. But only in SBS was it present in the **cervical spinal cord** and cervical nerve roots. "These findings corroborate suggestions that flexion-extension injury about the cervical spinal column may be important in the pathogenesis of SBS. This article is one of the only two articles that Geddes, Whitwell & Graham (2001) say have looked at BAPP in SBS (the other being Johnson, Boal & Baule, 1995). Geddes et al. cite these two articles as giving suggestive findings re the cervical BAPP, Johnson Boal Baule positing apnea as a result.

See also 1999 article in German by JF Geddes et al., below, concerning caudal medulla/ cervical cord AI. Hymel (2005) cites this paper as one of the papers, along with Hadley Sonntag and Johnson Boal Baule, that support craniocervical apnea – HIE as a common sequela of SBS.

Cerebral complications of nonaccidental head injury in childhood. Gilles EE and Nelson MD Jr. Pediatr Neurol 1998 Aug; 19(2): 119-128. Retrospective study of the radiology in 14 children. All patients suffered cerebral infarction. (Cerebral hypoattenuation). There were two patterns of infarction: focal and diffuse. The focal was the older children (mean age 19 months). The diffuse was the younger ones (mean age 5 months). All had a poor prognosis. The most common infarctions were subjacent to an SDH, in the posterior cerebral artery, the callosomarginal branch of the ACA, and border zone infarctions. No pts had a lucid interval.

Age-dependent material properties of the porcine cerebrum: effect on pediatric inertial head injury criteria. Kirk L. Thibault and Susan S. Margulies. Journal of Biomechanics 1998; 31: 1119-1126. See below under "Biomechanics."

Bruising in non-accidental head injured children: a retrospective study of the prevalence, distribution and pathological associations in 24 cases. Atwal GS, Ruttu GN, Carter N Green MA. FSI 1998 Sep; 96(2-3): 215-230. The bruising was mainly on the face, followed by the forehead and buttocks. Limb, chest, and abdominal bruising were uncommon. 29% had no fresh bruises. RH present in 96%. Why gripping does not cause bruises (of the chest or limbs).

Shaken babies (letter). Kleinman PK. Lancet 1998 Sep; 352(9130): 815-816.

Neuronal cytoskeletal changes are an early consequence of repetitive head injury. Geddes JF, Vowles GH, Nicoll JA, Revesz T. Acta Neuropathologica (Berlin) 1999 Aug; 98(2): 171-178. Dementia pugilistica. Examined five brain specimens. Found neurofibrillary tangles in the cortex of all 5, mostly grouped around blood vessels. No beta APP.

Shaken baby syndrome and the death of Matthew Eappen: a forensic pathologist's response. (letter) John Plunkett. Am J Forens Med Path 1999; 20(1): 17-21. Reviewed by Dr. Kirschner in Child Abuse Quarterly 2000 Jan; 7(1): 4-5. This article is too long and addresses too many complex issues to quote here. This letter is mostly a discussion of the "shaking alone" controversy; not clear what that has to do with Matthew Eappen, who had a skull fracture. Elsewhere Dr. Plunkett has commented that shaking may cause serious injury, but by the biomechanical data it cannot possibly cause SDH. He does not comment on whether it can cause DAI or apnea or brain infarction, or on the etiology of associated injuries such as cortical blindness and RH. It also delves into the controversy over whether a low fall can cause a SDH and also into the issue of rebleeds. Also comments on lucid interval. (Note that discussion of the "shaking alone" controversy is irrelevant to the Matthew Eappen case, since he had a skull fracture.) As Dr. Kirschner notes, whether a child's injuries resulted from shaking alone, or shaking with impact, (or just impact) is not significant; the point is that the injuries are inflicted. See also Cyril Wecht's *Letter to the editor*, Am J Forens Med Pathol 1999; 20: 301-302, arguing that the existence of SBS "must be seriously questioned," and that many of these injuries could be due to household falls. And Dr. Kirschner's further reply in Child Abuse Quarterly 2000 Apr; vii(2): 5-6, saying that "We need not rehash here the evidence

that shaking alone can and does produce fatal injuries, and that ... there are highly characteristic diagnostic signs of inflicted head trauma... There is also extensive clinical literature supporting the lack of serious injury or death associated with falls in the home, and the few articles that support such claims are so methodologically challenged that they cannot be taken at face value.” See further letter by Steve Cohle, Anthony Foster, Sandra L. Cottingham *Am J Forens Med Path* 2000 Jun; 21: 198-200, saying the consensus view of shaking alone is based on the concordance of clinical findings with confessions in a large number of cases.

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

Lucid interval	no
Rebleeds	nonpathologic
Impact	not necessary

Optic disc neovascularization following severe retinoschisis due to shaken baby syndrome. *Arch Ophthalmol* 1999 Jun; 117(6): 838-839.

Diffuse axonal injury in infants with nonaccidental craniocerebral trauma: enhanced detection by beta-amyloid precursor protein immunohistochemical staining. Gleckman AM, Bell MD, Evans RJ, Smith TW. *Arch path Lab Med* 1999 Feb; 123(2): 146-151. Used paraffin blocks from infants with 7 SBS, 3 other blunt head trauma, and 7 controls (1 cerebral edema, 1 HIE, 5 normals.) Compared BAPP axonal staining with H&E for axonal swellings. Found:

	<u>BAPP+</u>	<u>H&E+</u>
SBS	5/7	3/7
BHT	2/3	1/3
CTRL	0/7	0/7

Says that the H&E swellings were rare and difficult to find. Strongly recommends use of BAPP for forensic cases because of its greater sensitivity. Does not comment on specificity. Geddes et al., 2001, cite this article as being, along with Calder et al., 1984, Vowles et al., 1987, Hadley et al., 1989, and Shannon et al., 1998, among the few neuropathology studies of AHT. (They don't mention Oehmichen et al., 1998).

A 12-year ophthalmologic experience with the shaken baby syndrome at a regional children's hospital. Kivlin JD. *Trans Am Ophthalm Soc* 1999; 97: 545-581. Prospective series of 116 ch admitted 1987-1998. 84% had RH. “No fundus finding is pathognomonic for SBS.” “When RH are found in young children, the likelihood that abuse occurred is very high.” Leestma abstracts a case from this article in his 2005 collection of all the published cases with an admitted shaking history, as follows: “This 29 month-old female was admitted in coma, with extensive bruising of the head, face, limbs, and trunk. The chest showed ‘loop-shaped’ marks that were said to be pathognomonic for abuse. The wrists showed evidence of restraint injuries and many old burn marks. The baby's grandmother admitted to shaking the baby after being enraged at her resistance to toilet training and for swearing at her. The child apparently appeared dazed after the shaking and then fell down some stairs. The brain allegedly showed laceration of the corpus callosum... The eyes at autopsy showed evidence of retinal scarring and hemosiderin.”

Hyponatremic seizures in infancy: association with physical child abuse. Krugman SD, Zorc JS, Walker AR. *Pediatric Emergency Care* 2000; 16: 432-434. A child with RH and cerebral edema but no intracranial blood, thought to have been shaken.

Reduction of corpus callosum growth after severe traumatic brain injury (DAI) in children. Levin HS et al. *Neurology* 2000 Feb; 54: 647-653. Serial followup MRI study at 3 and 36 months post-injury of 53 children with TBI. The average age of the patients was ten (10) years. The injuries were caused by automobile accidents and falls. They found that there was reduction in or reduced growth of the corpus callosum.

Shaken baby syndrome: retrospective eye findings in 110 shaken babies. Kivlin JD, Simons KB, Lazoritz S, Ruttum MS. Ophthalmology 2000 Jul; 107(7): 1246-1254. A retrospective ophthalmologic study of 123 children admitted for AHT, of whom 90% (111) had fundusoscopic evaluations, of which 95 had dilated ophthalmoscopy. This study was primarily oriented to studying the ocular findings. SBS was defined as children under 3 who experienced SDH from abuse with characteristic bone injuries and the absence of a history of compatible accidental trauma. They excluded several pts who had small localized SDH's adjacent to a skull fx. RH were seen in 83% of patients who were examined funduscopically. Unilateral in 15% of these. The hemorrhages varied in type and location. "Dome-shaped hemorrhages under the internal limiting membrane were not common. They were recognized clinically most often in the macular area, but were found in any area of the retina on histopathologic examination, as reported previously (citing Greenwald, The shaken baby syndrome, 1990, and Riffenburgh & Sathyav, 1991.) Circular retinal folds were rare. Evidence of vitreous traction on the retina at the apex of circular retinal folds and at the edge of dome-shaped hemorrhages was found, as reported previously." (citing Massicotte, 199; Duhaime, 100 patients, 1992; Green, 1996; Mills, 1998.) "Unfortunately, there are no clinical ocular findings that are pathognomonic for SBS. Findings such as circular retinal folds and **dome-shaped hemorrhages**, originally thought to be most characteristic of abuse, **have been shown to occur in other conditions.** (citing Bacon, 1978; Kirschner & Stein, 1985; McLellan, 1986; Goetting & Sowa, 1990; Weedn CPR 1990; Keithahn, 1993; Morris, Kuhn, Witherspoon, 1994; Duhaime, 1996; Kramer, 1993; Clark spinal cord AVM, 1995. [Be careful: several of these refs, namely Kirschner & Stein, Weedn, and Clark, do not concern extensive RH. Goetting & Sowa contains inadequate data. -JKR.] States that two theories of the pathogenesis of RH in SBS continue to be debated: (1) Venous obstruction from sudden increases in ICP with or without chest pressure; (2) vitreoretinal traction. Does not take a position as between these theories. From the abstract: "According to the literature, when RH are found in young children, the likelihood that abuse has occurred is very high. Nonophthalmologists' difficulty in detecting RH may be an important limiting factor in identifying shaken babies so they can be protected from further abuse."

Dr Reece listed this as one of the top ten papers of the past ten years at the 2006 National Shaken Baby Conference. Dr Reece's presentation brought out a number of interesting findings that I did not list in my summary above. For example, 28% of these patients had been seen earlier, and in 1/3 of those, the diagnosis was missed. See above under "RH" for further of Dr Reece's presentation.

The annual incidence of AHT in Britain is 1:4000 live births. Barlow KM and Minns RA (Edinburgh), Lancet 2000 Nov 4; 356: 1571-2, quoted in Reuters Medical News on Medscape. 24.6 cases per 100,000 infants in a year and a half. Not necessarily fatal. Covered only age under one year.

Domestic violence: the shaken adult syndrome. Carrigan TD, Walker E, Barnes S. J Accid Emerg Med 2000 Mar; 17(2): 138-139. (Leeds) (have) Case report: 34 year old female with facial beating, arm grip marks, small L temporal SDH, RH. She had an occipital scalp hematoma and a right forehead abrasion. She said she had fallen down stairs, but later disclosed domestic abuse. Also had back and buttock contusions. No skull fx. She was initially obtunded but recovered full cognitive function within a matter of hours. Diagnosed with postconcussion syndrome. CT showed a small left temporal SDH with adjacent oedema. The RH are described as follows: "both retinal and preretinal haemorrhages in the right eye and a retinal haemorrhage on the left. Both maculae were affected by the haemorrhage (fig 1.)" There was markedly reduced visual acuity, and the patient was considered for a vitrectomy on the right. Fundus photographs of both eyes are given. (I only have a photocopy.) These appear to show a cluster of fairly large blot hemorrhages over the temporal vascular arcade OD with possibly a preretinal hemorrhage about 1.5 DD in size right over the macula. My copy is so dark that I can't tell, but I think that the hemorrhages are limited to the posterior pole. In the left eye, there is a small cluster of dot-blot RH in the posterior pole and what looks like papilledema or raised disk margins. Note by JKR: This is NOT a case of shaken adult syndrome. There was clearcut evidence of impact to the back of the head as well as direct eye trauma (Pt had periorbital bruises, epistaxis, and swollen nasal bridge.) Certainly it is abusive head trauma. The origin of the RH is problematic. The authors theorize that the RH might be related to chest or neck compression

Is race or ethnicity a predictive factor in shaken baby syndrome? Sinal S, Petree AR, Hermann-Giddens M, et al. Child Abuse & Neglect 2000; 24: 1241-1246. No. 68% of perpetrators are male.

The shaking trauma in infants: kinetic chains. Saternus KS, Kernback-Wighton G, Oehmichen M. FSI 2000; 109: 203-213. According to Hymel (2005), these authors “described autopsy evidence of unique injuries they felt were due to shaking, including injuries of the shoulder, the brachial plexus, and the muscles of the scapula; hemorrhages at the insertions of the sternocleidomastoid and interspinous muscles, and disruptions of the intervertebral discs of the neck.”

Traumatic axonal injury: practical issues for diagnosis in medicolegal cases. Geddes JF, Whitwell HL, Graham DI. Neuropathology and Applied Neurobiology 2000; 26: 105-116.

Summary: Argues that BAPP studies have shown that axonal damage is very common in anoxic and brain-swelling/ herniation settings, so that the finding of DAI is not necessarily traumatic. (This finding is replicated by Oehmichen et al., 1998. –JKR) To establish it as traumatic, you have to do detailed geographic sampling and correlate with ischemic lesions. If you diagnose that it is traumatic in origin, you should specify that by employing the term “traumatic DAI” as opposed to “hypoxic DAI.” Specifically look at the caudal medulla, the upper cervical cord, and the cervical nerve roots, because these areas may be specific for TAI as opposed to hypoxic or nonspecific/nontraumatic DAI in infants.

Reviews the history of DAI beginning with Strich (1956.) As initially recognized, it was always traumatic and characterized persons or animals who became immediately unconscious with head trauma, survived, and had prolonged coma in the absence of a focal lesion. (Adams, 1982) Early experimental work (Gennarelli, Thibault, 1982) showed that it was due to rotational or angular acceleration, “which explained why it was most usually associated with ... road traffic accidents or falls from a height.” There then followed nonprimate animal trauma models (rat, pig, guinea pig optic nerve stretch model) which showed that stretching or other mechanical deformation of axons is the mechanism. These experimenters used graded deformation of axons and observed the ultrastructural and biochemical results. Mild deformation caused transitory increase in the permeability of the axolemma with resulting transitory changes in transmembrane ions. (This is the basis for concussion.) Moderate deformation caused fluid entry into the cell, with cell swelling, but axoskeletal integrity could still be restored. With severe deformation, intracellular calcium ion accumulation led to compaction of neurofilaments and microtubules and collapse of the cytoskeleton, which is followed by impaired axonal transport, which is followed by accumulation of proteins and organelles at the damage point, causing the retraction ball. Adams et al., (1984, 1985, 1989, 1991), who described DAI in humans, discovered that it is an evolving lesion. “Primary axotomy very rarely occurs at the time of injury.” Axonal damage, whether traumatic or nontraumatic, both evolves over a period of hours, AND has reversible and irreversible forms. Silver stains were unable to demonstrate this, but immune stains can demonstrate these forms. Silver-stainable balls will not show up for 12-18h after the injury, but immune accumulations will show up in 2-3h. You have to take blocks from many areas in order to demonstrate the extent of the lesion and have any chance of shedding light on its biomechanics. Recommend at least 8 blocks:

1. [Anterior] CC and parasagittal frontal WM
2. Splenium
3. [BG] with posterior limb of internal capsule
4. Cerebellar hemisphere
5. Midb
6. Pons with peduncle
7. Med
8. Upper cervical cord

Axonal injury has a predilection for the posterior CC, so always get that (splenium.) Stay as far as possible from obvious foci of infarction or hemorrhage, since these cause their own secondary axonal damage. Use BAPP for 2-3h out to about a month. Use GFAP if looking for an astrocytic reaction. Use iron as needed. In short survival cases (hours to a few days), H&E and silver stains will underdiagnose axonal injury. Need BAPP in this setting.

BAPP displays impaired fast axonal transport; hence it is not specific for trauma. It shows up in multiple sclerosis and Binswanger-type conditions, and in anoxic injury. This last is most important. To some extent, ischemic AI can be distinguished from traumatic AI; ischemic AI by BAPP tends to occur in geographic patterns along the borders of [infarcts.] Also the gross topography of the areas may relate to the

territories of vessels compressed. But unfortunately the areas most commonly infarcted in the swollen brain happen to be the same areas most commonly affected by TAI (gives diagram indicating (a) parasagittal WM, (b) CC, (c) IC, (d) rostral brain stem, as being BAPP-positive in the case of a herniating focal lesion *and* TAI.) If there has been days of survival, ischemic injury can be disting from TAI by characteristic histologic features of ischemia.

There is a problem in distinguishing hypoxic DAI from traumatic DAI. That is the overlapping of the geographic locations of the expected lesions. It is also that there is the lack of a database. There are very small numbers of cases with published neuropathology. Only two NP studies of AHT have been published. (Shannon, 1998; Gleckman, 1999.) Shannon found BAPP widely dispersed in the brains of both anoxic encephalopathy infants and AHT infants, but only in AHT infants did they find AI in the high cervical cord and cervical nerve roots. Gleckman's SBS cases with +BAPP are probably just anoxic. But we have noted caudal medulla corticospinal-tract BAPP atypical for anoxic, which "may well represent the effects of local stretch at the craniocervical junction..." Commenting on the lack of cases, the authors conclude that "The lack of correlation between well-documented histories and NP findings means that in the interpretation of assault cases at least, a diagnosis of 'TAI' or 'DAI' is likely to be of limited use for medicolegal purposes."

Time of injury in infants: (contribution of BAPP staining) In one small series of adult brains, BAPP staining began at 2h and evolved... But "apart from demonstrating damaged axons much earlier than [conventional stains,] BAPP staining does not provide a more accurate means of dating injury." Also: "It is important to realize that TAI in infants has not been studied at all, and its evolution may well be different from that in older children and adults."

Biomechanics in adults: In traffics and falls from a height, you have acceleration-deceleration. Cannot completely exclude the poss of diffuse TAI from falls from one's own height in adult settings. Minor or localized TAI is certainly possible. If TAI occurs, look for it in the brain stem, where accel-decel wd be expected to cause.

Biomechanics in infants: One study by Shannon (1998) found BAPP in the high cervical cord and its nerve roots only in the NAI group and not in the hypoxic-ischemic group. Then there is Gleckman's report (1999) found BAPP in 5/7 SBS, but most of them were swollen and we have found that hypoxic BAPP is much more common than clearly traumatic BAPP in NAI. **But, in the caudal brain stem**, may be specific for trauma, since ischemic is not expected there.

There is a critical letter to the editor, Neuropathol Appl Neurobiol 2000 Oct; 26(5): 491, and I have the reply.

Betty Spivack comments in *Child Abuse Quarterly* for April 2001 that this art is an "authoritative review." Further, that, "The tantalizing implication of **low brainstem and high cervical spinal cord axonal injury** which might be directly linked to shearing at the craniocervical junction from shaking may be a cornerstone in understanding the frequency of apnea in this condition. [This] site of injury corresponds to the site of spinal epidural hematoma, subdural hematoma, and cord contusion reported by Hadley (Neurosurg 1989; 24: 536-540.)"

See also Geddes & Whitwell's 2001 pair of papers on TAI in AHT, below.

Topography and severity of axonal injury in human spinal cord trauma using beta amyloid precursor protein. Spine 2000; 25: 1227. In adult material.

Prognostic indicators for vision and mortality in shaken baby syndrome. McCabe CF, Donahue SP. Arch Ophth 2000 Mar; 118(3): 373-377. Retrospective review of the charts of 30 consecutive cases aged 1 – 39 mos (x= 9 mos). 93% had prer and 100% had intrar, 10% had vitr. All pts with nonreactive pupils on presentation died. 6/7 with midline shift died. Nearly all RH had resolved by 4 mos. Ventilator requirement predicted poor visual outcome.

Evaluation of infants with subdural hematoma who lack external evidence of abuse. Morris MW, Smith S, Cressman J, Ancheta J. Pediatrics 2000 Mar; 105 (3 Pt 1): 549-553. (have) Retrospectively reviewed all children referred for SCAN evaluation during 1997 who had any form of intracranial hemorrhage. There were 19 investigations bc of SDH.

8 obvious child abuse (RH, bruises, fractures)

- 2 small subdurals under linear fractures
- 9 SDH with no RH and no other ev of abuse

Studied nine cases, ages 11 days to 15 months. Of these nine, 8 ended up being diagnosed as inflicted cerebral trauma based on the SCAN investigation, one as “possible inflicted injury in a high-risk setting.” SCAN investigation uncovered the circumstances of trauma. “Infants with subdural hematoma but no other findings of abuse present a difficult challenge to child protection workers.”

Maturation-dependent response of the piglet brain to scaled cortical impact. Duhaime A-C, Margulies S, Durham SR et al. J Neurosurg 2000; 93: 455-462. See below under “Biomechanics.”

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 vs. inflicted.” Reports 18 cases of fatal pediatric short-fall head injuries culled from US government databases. Discusses biomechanics of shaking. Experimentally, impact duration longer than 5 msec will not cause SDH unless the angular acceleration is above 1.75×10 to the fifth radians per second squared. (citing Gennarelli & Thibault, 1982.) With an approx three inch radius of acceleration from the mid-neck to the midbrain (which is considered to be the center of mass of the infant head in the Duhaime model) this leads to a required minimum tangential (linear) acceleration of 17,500 m/sec². “A human cannot produce this level of acceleration by impulse (shake) loading.” SDH much more likely to result from impact on a hard surface. “A ‘high-strain’ impact typical for a fall is more likely to cause subdural hemorrhage than a ‘low-strain’ impact...”

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. The formulation is that SBS exists but you can never prove it in any individual case. If there are no impact injuries, that does not rule out impact. If there are impact injuries, that does not rule out shaking. [This formulation is clearly an attempt to move us beyond the sterile and essentially pointless debate over shaking, by claiming that we can have it both ways: they are probably all impacts, but shaking is an acceptable diagnosis, at least until biomechanical evidence comes in that finally refutes it. –JKR] It all boils down to rotational injury. Rotational injury causes DAI, and RH, SDH, and SAH are markers for DAI. DAI in this setting usually cannot be morphologically observed because these babies do not live long enough and because retraction balls are difficult to see in infants due to their very small axons. Contusion tears are rarely seen in infants. The scientific basis for believing that SBS exists is: (a) Rotational injury (magnitude not stated) is known to cause shearing injury. (b) The shearing threshold for infant axons is unknown, but “no present evidence rules out” the possibility of its being within the range of accelerations attainable by shaking. (c) Shearing injury has been demonstrated in infants who by history were shaken and who by autopsy had no morphologic evidence of impact. Abusive head injury commonly occurs in response to prolonged crying. To stop them from crying. The arms and legs may also be violently flung about, causing metaphyseal fractures to the long bones. Many (victims) do not have any external injuries. Ribs are the most common bones injured. Mortality of AHT is 7% to 30%. Child as old as 4 or 5 years can be AHT. Goes into the heavy head, weak neck, thin pliant skull, flat skull base, wide shallow subarachnoid space, unmyelinated WM, small axonal size. Translational and rotational forces. Translational movement of the brain is quite benign (citing Ommaya, 1968; Gennarelli, 1982.) “The trivial falls that children sustain in falling from furniture and even down stairs primarily involve translational forces. Rotational injury of the brain results in DAI. (Citing Ommaya Gennarelli, 1974; Duhaime on 100 pts, 1992; Hanigan on tin ear syndrome, 1987; Margulies, Thibault on DAI, 1989; Brenner, J Neurosurg, 1988.) Criticizes the Duhaime model of a doll with a rubber neck, and Gennarelli’s adult primate model, citing Brenner, 1988. Adduces confessions and witness statements of shaking, and autopsies with no impact site, but qualifies these by saying that “this experience must be received with some caution.” [Confessions are unreliable and witnesses usually are close relatives.] Impacts may not be reflected on the scalp if the striking surface is padded or if it is broad and firm. A child’s scalp is very elastic and stretches on impact. (And the skull readily deforms if it is a young infant. –JKR) Not all impacts are registered as hemorrhages in the galea. In the vast majority of cases, it is not possible to definitely characterize children’s head injuries as being caused by either pure impact or pure shaking because the pathologic changes in the brain are identical... If there are focal injuries, an impact can be assumed, but coexistent shaking cannot be excluded. In the absence of signs of

an impact, however, shaking alone should not be presumed bc there may well have been an impact that cannot be identified.” (citing Gilliland, 1996.) Both shaking and impact produce the **markers for DAI**. So they are saying that shaking can produce DAI, but shaking cannot be pathologically established as a fact in any case. Goes on to describe the marker lesions. As to SDH, it is probably always present in infant DAI (implying because of its lower biomechanical threshold) but is not always seen at autopsy bc interhemispheric blood may “not be seen” at autopsy. The correlation of autopsy SDH with CT SDH is not 1:1 bc CT can also miss thin collections. As to SAH, it is almost always present, apt to be patchy near the midline, very small. If there is SDH, there is SAH, bc the bridging veins arise in the SAS and are invested by arachnoid mater as they cross the SDS.

As to RH, “Currently their pathogenesis is not precisely understood. Their presence highly correlates with rotational head injury...” Makes an interesting statement: “The RH seen in abusive head injuries are similar to those that are frequently observed in full-term neonates after vaginal delivery.” More severe intraocular injury correlates with more severe head trauma, citing Green et al. (Br.), 1996. (But note that Green’s article speaks in terms of the anatomic *planes* of intraocular injury, beginning with subhyaloid, then intraretinal, then subretinal, then VH and choroidal, not in terms of the amount or extent of intraretinal hem or the type of RH as to schisis versus nonschisis.) Cites Levin, 1990, for the proposition that RH associated with ICP are peripapillary and associated with papilledema.

As to ONSH, it is nonspecific, strongly correlates with the presence of SDH, “although there is not necessarily a direct connection between the subdural compartment of the orbital sheath and the subdural compartment of the intracranial cavity.” (Citing Green, et al., 1996.) ONSH can be seen in non-trauma cases (citing Greenwald, 1986.)

As to DAI, retraction balls may be visible after 18 to 24 hours, very difficult to see in young children bc of the small size of the axonal processes. BAPP as early as 2 hours. Petechiae are very seldom seen in young chil bc the vessels stretch and do not break. Contusion tears are rare. Watch out for artifacts misinterpreted as contusion tears. Important statement: “It is not usually possible to morphologically establish the existence of DAI in young chil by demonstrating the classic pathologic changes of retraction bulbs, tissue tears, or intraparenchymal hemorrhages, although these findings may be demonstrated on occasion. (citing Vowles, 1987; Mary Case’s ASCP Check Sample, 1997.) Many of these children die too soon after injury for these pathologic changes to be established. For this reason, it is important to appreciate the **markers** of shearing injury to indentify these cases as DAI.” (emphasis added.) Mary Case has made this statement before (SBS Conf 1998, San Diego 2000.)

Discusses time of injury. Immediate decrease in LOC. LOC is caused by axonal injury in the deep gray matter and rostral brainstem, not by anoxia or ICP. Respiratory compromise, however, may be delayed. It may be due to caudal brainstem damage, but the poss of delay is not explained here. Repetitive injury mentioned, which is survived.

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.

As to fall versus abuse, gives a carefully worded discussion of this “area of concern.” “The trivial falls that children sustain in falling from furniture and even down stairs primarily involve translational forces. Rotational injury of the brain results in DAI. (Citing Ommaya Gennarelli, 1974; Duhaime on 100 pts, 1992; Hanigan on tin ear syndrome, 1987; Margulies, Thibault on DAI, 1989; Brenner, J Neurosurg, 1988.) “Fatal accidental shearing or diffuse brain injuries require such extremes of rotational force that they occur only in obvious incidents such as motor vehicle accidents. ... falling from considerable heights (greater than 10 feet)...* Isolated SDH/SAH (apparently meaning without a fracture?) in fatal accidental head injuries in young children is less than 2%, compared with 98% in AHT. Trivial falls “are associated with primarily translational forces and not with the rotational forces necessary to develop tearing of bridging veins...or other shearing injury.” Does not comment on alleged falls that are non-trivial, e.g. out of a high chair, with an adult who falls, on a furniture that falls, off a bunk bed, onto a very hard surface, during horseplay, etc.

* (Note the judicial rejection of this analogy in *People v. Martinez*. Note also that they are placing the energy required for shearing in a very high energy regime, but still claiming that such energies can be developed from manual shaking -- but not from any form of household fall. But what are the measured energies involved in these events? –JKR)

I am informed that this position statement expired by its terms in 2003. --JKR

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. <http://www.aap.org/policy/t0039.html> Randy Alexander, Kent Hymel, Robert Block, Carole Jenny and others. "SBS is a serious and clearly definable form of child abuse. It results from extreme rotational cranial acceleration induced by violent shaking or shaking/impact." "Shaking by itself may cause serious or fatal injuries (citing Alexander, Sato et al, 1990; Gilliland & Folberg, some have no impact inj, 1996.) In many instances, there may be other forms of head trauma, including impact injuries. Thus, the term shaken/slam syndrome (or shaken-impact syndrome) may more accurately reflect the age-range of the victims and the mechanisms of injury seen." "The constellation of these injuries does not occur with short falls." Shaking is "so violent that individuals observing it would recognize it as dangerous and likely to kill the child." Ages up to 5. Ev of previous cranial injuries is found in 33-40% (citing Alexander et al., Serial abuse, 1990; Ewing-Cobb, 1998). Subtle symptoms include poor feeding, vom, lethargy, irritability over days or weeks, often attributed by pediatricians to viral illness or colic. These symptoms will be obviously abnormal even to unsophisticated parents. 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..." In 75% to 90% of cases RH are present. MRI is 50% more sensitive for subdural blood than CT, but in Sato's study CT did not miss any surgically treatable injuries. (Sato et al., 1989) SDH may be minimal over the convexities and most prominent in the interhemispheric fissure. There may be only SAH and no SDH. DAI is common (citing the Section on Radiology, 2000) "however, for technical reasons, it is often not possible to demonstrate this pathologically or radiologically in individual cases." Common late neuropathological findings are cerebral atrophy or infarction from hypoxic damage and chronic extra-axial fluid collections, cerebral atrophy, and cystic encephalomalacia. "The diagnostic entity of 'benign subdural effusions' should be viewed with caution, because multidisciplinary evaluations in previously described cases were lacking." (citing Aoki, 1984) The mortality of SBS is about 25%. Survivors have cortical blindness, spasticity, seizure dso, microcephaly, chronic subdural fluid collection, enlarging ventricles, cerebral atrophy, encephalomalacia, porencephaly, also learning, motor, or behavioral problems often thought to be of unknown cause.

Mechanisms of brain injury in infantile child abuse (editorial). Shannon P, Becjer L. Lancet 2001 Sept. 1; 358: 686-687. Literature review.

Release of biochemical markers of damage to neuronal and glial brain tissue is associated with short and long term neuropsychological outcome after traumatic brain injury. Herrmann M, Curio N, Jost S, Grubich C, Ebert AD, Fork ML et al. J Neurol Neuros Psychiatr 2001; 70(1): 95-100. Get CSF NSE or S-100. Increases rapidly if injury is present. Crosses the brain-CSF barrier rapidly. Still experimental.

Neuropathology of inflicted head injury in children I. Patterns of brain damage. Geddes JF, Hackshaw AK, Vowles GH, Nichols CD, Whitwell HL. Brain 2001 Jul; 124 (7): 1290-1298. Studied 53 AHT brains and spinal cords including spinal nerve roots using gross pathology, conventional histology, and BAPP. Had 37 infants and 16 older children aged 1 year to 8 years. They studied various types of lesions, including thin-film SDH, space-taking SDH, cord EDH, and three forms of axonal injury:

vascular-ischemic axonal injury
focal traumatic axonal injury (TAI)
diffuse (traumatic) axonal injury (DAI)

Vascular-ischemic axonal injury was BAPP+ axons distributed in "a geographic pattern of white-matter immunoreactivity, which was usually widespread and related to vessels." (p. 1294) and was found in the presence of global hypoxic-ischemic neuronal change seen on H&E. Also "Focal geographic BAPP expression, commonly seen in the diencephalon and brainstem, was taken to be outlining areas of incipient ischaemia resulting from brain swelling" (citing their earlier work, 2000). This was considered to be due to brain swelling/ICP.

Focal TAI “was identified by finding BAPP-immunoreactive axons or bulbs, scattered or in groups in hemispheric white matter, corpus callosum and internal capsule.” This particular pattern was found in 11 victims -- five infants and six older children.

DAI was identified when there was cerebral TAI plus brainstem involvement (other than hypoxic-ischemic type brainstem involvement.) DAI “is defined as widespread traumatic axonal damage occurring throughout the centrum semiovale, particularly parasagittal white matter, corpus callosum, internal capsule and cerebellar peduncles in the rostral brainstem (Adams et al., 1989).” This particular pattern was found in only three of the 53 victims and the authors found it to be “rare.”

A signal finding here was that of TAI **limited to the lower brainstem or cervical nerve roots**. This particular finding was made in 11 cases -- all infants. And the authors point out that this pattern was not rare. Hence it may be telling us something about mechanism in AHT. On p. 1297 the authors say that this findings is “similar to that reported in adults with nondisruptive cervical cord damage due to a hyperextension neck injury” (citing Lindenberg & Freytag, 1969, and Geddes et al., 2000). “Injury at this point, presumably caused by stretch to the neuraxis caused by cervical hyperextension, might provide an explanation for the frequent occurrence of apnoea at presentation.” (referring us to Geddes II for further.)

Reviews the literature of the neuropathology of fatal head injury in the pediatric age group:

Lindenberg & Freytag, 1969, finding contusion tears.
 Calder et al., 1984, finding contusion tears.
 Hadley et al., 1989, finding damage to the craniocervical junction.
 Hart et al., 1996, correlated postmortem imaging with autopsy findings.
 Vowles et al., 1987, suggested DAI was a feature of AHT.
 Shannon et al., 1998, suggested DAI was a feature of AHT.
 Gleckman et al., 1999, suggested DAI was a feature of AHT.

Leestma, 1988, merely reiterated the features of adult trauma.
 Brown & Minns, 1993, merely reiterated the features of adult trauma.
 David, 1999, merely reiterated the features of adult trauma.

Says that these articles paid little attention to confounding factors such as brain swelling and global hypoxia. We, on the other hand, “took great care to ... attempt to distinguish between axonal damage caused by trauma and axonal damage secondary to hypoxia-ischemia, raised ICP, and/or brain shift.” (p. 1297).

The mechanism of thin-film SDH in infants may be different from that of SDH in adults, because its morphology is different.

The authors admit (p. 1297) that their criteria for inclusion of cases as definite AHT were not as tight as they would like. For example, 12 cases were defined as AHT because there was a judicial conviction of child abuse. The authors note that a judicial conviction does not necessarily establish AHT as a medical fact, particularly since in these cases there was no extracranial trauma. But what are you going to do? The other articles in the literature on AHT suffer from the same defect. [See the critical discussion of this issue by Donoho in “Evidence-based medicine, 2003, below.]

71% of their victims had RH. No cases of RH without subdural bleeding.

The following from review by Betty in *Child Abuse Quarterly* for fall 2001: 53 children who died of AHT. 71% RH. 63% apnea at presentation, 36% skull fx, 19% rib or clavicle fx, 11% long bone fx. 72% had SDH, mostly thin and with no mass effect in infants (Betty has emphasized this in the past.) 77% had hypoxic neuronal injury, 48% had at least some TAI. 30% had cervical EDH, and these had TAI of the brain stem, cervical cord, and nerve roots. Suggest that this **cervical/stem injury may be the source of**

immediate apnea, and apnea in turn the source of diffuse retraction balls in the cerebrum in a vascular-related (ischemic, not traumatic) pattern.

In their Reply to Punt, 2004, Geddes et al. remark that since this paper was published, they have learned from experience “to be more cautious” about using confessions as a criterion for recognizing abuse. See under “Head Injury -- Fall vs. Inflicted,” below.

This article is attacked by Punt et al. and defended by its authors in *Pediatric Rehabilitation*, 2004. See below.

These articles are also attacked as “junk science” by Dr Jerold F. Lucey, editor in chief of *Pediatrics*. See Letter, *Pediatrics* 2004; 113: 433. But defended on other grounds by Pat Lantz in letter, “Glass houses,” above under “RH -- in general.”

Neuropathology of inflicted head injury in children II. Microscopic brain injury in infants. Geddes J, Vowles GH, Hackshaw AK, Nickols CD, Scott IS, Whitwell HL. *Brain* 2001 Jul; 124(7): 1299-1306. Compared AHT with SIDS. Saying that it has not yet been established that traumatic DAI (TAI) occurs in victims of AHT. [Compare a similar comment in passing by Duhaime et al. in their 2003 biomechanical study, below.] Here compared the brains of 37 AHT victims aged 9 months or under with the brains of 14 infants aged 12 months or under who died of various natural causes including SIDS (7), respiratory infection (5), perinatal asphyxia (1), and gastroenteritis (1). Found that of the AHT brains, 29 had neuronal changes of hypoxia. 25 had BAPP+ in axons (this is throughout the axons in a lengthwise staining, not retraction balls), including 11 who were found dead (no hospitalization). Five brains had minimal TAI affecting only the corpus callosum or central white matter. Two had DAI widespread enough to be considered DAI. In 13 cases the axonal pathology “appeared to be largely vascular in nature, associated with brain swelling and increased ICP.” (p. 1300) In seven other cases, BAPP+ retraction balls were found only in the caudal pons and medulla, localized to the corticospinal tracts (long tracts). In three cases retraction balls were found in the cervical spinal cord or nerve roots. Among the 14 controls, there was never any BAPP positivity or retraction balls in the spinal cord or nerve roots, and only two in the brain, located in central white matter. These were both cases that had cerebral edema and increased ICP (the gastroenteritis case and the perinatal asphyxia case).

The authors review the literature on microscopic examination of the brain in AHT, finding five studies (Lindenberg & Freytag, 1970), Calder et al., 1984, Vowles et al., 1987, Shannon et al., 1998, Gleckman et al. 1999).

Cited by Kemp et al., 2003, for the proposition that “it may not be necessary to shake an infant very violently to produce stretch injury to its neuraxis.”

These articles are discussed in letters, Retinal hemorrhages in infant head injury. Clark BJ, Adams GG, Luthert PJ. *Brain* 2002 Mar; 125 (3): 677-678 (letter), and Neuropathology of inflicted head injury in children. Parulekar MV, Elston JS. *Brain* 2002 Mar; 125(3): 676-677 (letter), and Reply. Jennian Geddes and Helen Whitwell. *Brain* 2002 Mar; 125(3): 678. (See under RH.) Parulekar et al. argue that craniocervical-induced apnea might account for the diffuse brain changes but would not account for the RH, which are almost certainly caused by vitreoretinal traction. But Clark et al argue that abruptly increasing ICP could cause the RH and therefore would be consistent with Geddes’ work. Geddes replies that since experts in the same specialty disagree so sharply about the pathogenesis of RH, that proves that nobody knows the pathogenesis of RH. Another critical comment is Punt J, Bonshek RE, Jaspan T et al., The unified hypothesis of Geddes et al. is not supported by the data. *Pediatric Rehabilitation* 2004; 7: 173-184. But see Geddes et al’s *Reply* to that criticism, *Pediatric Rehabilitation* 2004; 7: 261-265. See also next below.

Silenced Angels: The Medical, Legal, and Social Aspects of Shaken Baby Syndrome. James R. Peinkofer, ACSW/LCSW. Greenwood Publishing Group, Inc., 2002? ISBN 0-86569-313-7. An advocacy-oriented work dealing fairly comprehensively with all areas including “myths” such as CPR and vaccine.

Shaken Baby Syndrome: A Multidisciplinary Approach. Lazoritz S and Palusci V, eds. Binghamton: Haworth Press, 2001. ISBN 0-78901351-7.

Forensic Pathology, Second Edition. DiMaio VJ and DiMaio D. Boca Raton: CRC Press, 2001, pp 358-363. Argues that all SBS cases are cases of inflicted head impact trauma. 1. Impact often leaves no scalp mark or skull fracture, quoting Bernard Knight's textbook. 2. Many cases where the history is that of shaking have indisputable clinical or autopsy evidence of impact. 3. Where are the neck or cervical cord injuries that would logically be expected? 4. The biomechanics doesn't work. 5. The reported no-impact cases were all from early series where for the most part there were no autopsies done. 6. Caffey's article is deeply flawed. [On this last point, DiMaio & DiMaio barely scratch the surface. -JKR]

Manifestations of shaken baby syndrome. Kivlin JD. Curr Op Ophth 2001 Jun; 12(3): 158-163. RH in SBS vary in type and location. No particular type is pathognomonic for the condition. RH are not needed to make the dx of SBS. A review article. "The dx of abuse shd be made by someone particularly trained in this area, who can put together the entire picture of inadequate or changing history, fractures of various ages...SDH, and RH."

The spectrum of postmortem ocular findings in victims of shaken baby syndrome. Marshall DH, Brownstein S, Dorey MW., Addison DJ, Carpenter B. (Ottawa) Can J Ophth 2001 Dec; 36(7): 377-383. Retrospective review of the autopsy material on six patients aged 1 to 34 months from 1971 to 1995. From the abstract: "...ranged from a focal globular hem at the posterior pole to extensive intraocular hem involving the entire retina with perimacular folds." All had ONSH. All had intracranial hemorrhage, 2 had skull fx, 3 had rib fx, 4 had high spinal cord hemorrhage.

The cause of infant and toddler subdural hemorrhage: a prospective study. Feldman KW, Bethel R, Shugerman RP, Grossman DC, Grady MS, Ellenbogen RG. Pediatrics 2001 Sep; 108(3): 636-646. Prospective study of children under 36 months who had SDH, excluding known hemorrhagic disease and other known predisposing factors for bleeding. Did full workup including retinal exams, social workup.

Total admitted with SDH and meeting the study criteria	66
Abuse ruled in	39
Abuse ruled out	15
Undetermined cause of SDH	12

Mean age of abused was 8.7 mos, of nonabused was 19.1 mos. The usual Hx for abused was a minor fall or no hx of any injury. The universal Hx for nonabused was either MVA or well-documented trauma. Only the abused and the UNDET had chronic SDH (17/39 ab and 8/12 UNDET). RH in 28/39 abused (72%). RH in only 1 of 3 accidentals who had eye exams, and this one RH was recognized as being the type of RH that is associated with increased ICP. Conclusion: nearly 1/5 of SDH resulted from unintentional trauma. Cf Chamnanvanakij, 2002 on neonatal SDH's, below; Rogers, 1998 under "Birth Injury," above.

Shaken baby syndrome and hypothermia. (letter) Allasio H, Fischer H. Child Ab Negl 2001 Nov; 25(11): 1413-1414.

Animal models for shaken baby syndrome: ethical testing yields valuable insight to the nature of the eye and brain injuries in SBS cases. Alex V. Levin, MD, MHSc, FAAP, FAAO, FRCSC. SBS Quarterly, Winter 2001. <http://www.dontshake.com> (have) Earlier whiplash experimental work on animals had to do with head injury related to motor vehicle accidents or jet pilot military maneuvers. (citing Ommaya, Faas, Yarnell, 1968). More recently rats have been shaken to cause DAI, but no eye studies were done. (citing Smith, Andrus et al., 1998). A stretch model of guinea pig optic nerve has been published. (citing Tomei, Spagnoli et al., 1990). Dr Randell Alexander has suggested that the woodpecker offers a natural model for protective mechanisms. Dr Levin has dissected woodpeckers. Finds that (a) the eyeball cannot move within the orbit, and (b) the sclera is reinforced by bone. Since woodpeckers with a fixed and rigid eyeball do not get retinal damage from deceleration, and human babies with a movable and soft eyeball do, we may infer that the movable and soft eyeball increases vulnerability to retinal injury from deceleration.

Also the woodpecker vitreous is not adherent to the posterior retina. But woodpeckers do not have retinal blood vessels(!), so they cannot possibly serve as a model for RH. Suggests (a) repeating the rat shake experiment with eye dissection, (b) getting natural or experimental shaking on larger animals. Dogs shake ground hogs to death.

People v. Manjit Kaur Basuta, D034429, Cal App 4th (2001). (have) Reversing conviction of a San Diego day care lady for assault on a child with force likely to produce great bodily injury or death under PC 273(a) and (b) by shaking a 13 month old male weighing 34 pounds? (this wd be an average two-year-old) who died with a large SDH and bilat unspecified RH plus old SDH. No mention in the opinion as to whether the autopsy showed evidence of impact or not. It appears the prosecution's theory was shaking. Defense theory of a fall from his own height and rebleed. Prosecution T by Randy Alexander and Dr. Daniel Davis (FP). Defense T by Dr. Janice Carter-Lourensz and Dr. Plunkett. Conviction reversed on the following evidentiary grounds:

1. Trial court allowed ev that there was an earlier SDH, but *excluded* T that child's *mother* had previously shaken the child on grounds of confusion and waste of time under Ev Code Sec. 352. (Prosecution conceded that it was relevant under 210.) Held, abuse of discretion because T of the mother's earlier shaking wd hv allowed the jury to consider that it cd hv bn the mother, rather than defendant, who caused the earlier SDH. Under the defense rebleed theory, if defendant did not cause the earlier SDH, and rebleed of this due to a fall caused the death, then the jury could find that defendant was not responsible for the death. Exclusion of the T prevented the jury from considering this factual issue and thereby prevented the defense from presenting its theory of the case. (The court also argues that this T cd hv bn used by the defense to attack the credibility of a prosec w, the defendant's Guatemalan housemaid.) The court concludes that defendant was prejudiced by this exclusion.
2. A prosecution w police officer at the conclusion of the prosecution's case mentioned the fact that the Guatemalan housemaid had taken a polygraph (not its results).
3. Certain scientific evidentiary issues regarding SBS. (The court characterizes this discussion as *obiter dictum*.)
 - a. Whether medical opinions regarding SBS need to be pre-evaluated under *Kelly-Frye* (*P. v. Kelly*, 17 Cal3d 24 (1976) and *Daubert*. Defense contended that the lack of such pre-evaluation deprived d of DP.
 - b. Exclusion of T of a defense biomechanics expert that shaking cd not cause fatal SDH but a short fall could.

The court states that the prosecution's *medical* T regarding SBS and SDH was "inductive or epidemiological, ... based on clinical experience and the study of actual cases." The defense *biomechanical* T, on the other hand, was based on experiments and biophysical theory. Plunkett's T that shaking cd not cause SDH based on biomechanical data from the automobile industry and Duhaime et al., and Plunkett's T that short falls can cause fatal SDH and serious brain injury based on biomechanical research and government statistics. Same T from Dr Carter-Lourensz. Defense biomechanician Dr. Jones, if called, wd hv T that (a) shaking alone cd not cause SDH, (b) short fall onto a hard surface cd cause SDH, (c) there are generally accepted values for the force required to cause SDH. [The court notes that "No other ev or T was offered to corroborate the conclusion that such accepted value existed."] (d) He conducted an *experiment* to determine whether a short fall to a hard surface by a 34 pound 29 inch tall "infant" could cause SDH, and found that it cd; his experiment replicated an earlier published study also relied on by Dr. Plunkett. Prosecution argued in 402 hearing that these experimental models did not replicate the child's actual head and neck, and the opinion did not satisfy *Kelly-Frye*. The trial ct excluded Dr. Jones's experiment based on *Kelly* that it was "too theoretical to comply with generally accepted standards of scientific reliability." The trial ct excluded Dr. Jones's general opinion that shaking cd not cause, based on the prosecution's objection that Jones was not a medical doctor.

The appellate court first states that in California ev law the treatment of "scientific ev" is different from the treatment of "scientific expert *opinion*" under the learning of *P. v. Stoll*, 49 Cal3d 1136 (1989). This learning is that ev of a scientific "experiment, data, or

technique” must meet a “general acceptance” test as to its reliability, but expert scientific *opinion* T need not. The reason is that juries tend to assign infallibility to the results of machines which read out objective technical data which a witness “merely relays” to the jury. On the other hand, opinion T is not ascribed this level of infallibility by juries, because “it is understood [by jurors] that human beings make mistakes.” Also, experiments and technical measuring devices, unlike human experts, may be “new,” and not yet generally accepted by scientists as reliable. This is the rationale for the more conservative evidentiary standard for technical tests, experiments, and measuring devices as distinguished from opinions. Tests must satisfy a *Kelly* hearing; opinions need not. Thus the *Kelly* rule has never been applied to medical testimony; medical determination of the causative mechanism of injuries is not a “new technique” for purposes of *Kelly-Frye*. Defendant attacks this dichotomy as arbitrary and a denial of due process. Either the *Kelly* test should be applied to medical expert opinion as well as experimental tests, or it should be removed from both. The appellate court does not accept this argument, because the California Supreme Court has not (yet) accepted it. The court further declines to hold that the medical experts’ use of experiential, epidemiological, and “anecdotal” data is unscientific as a matter of law. So the court rules against defendant’s due process contention on the evidentiary treatment of biomechanics experimental results. As to the admissibility of Dr Jones’s biomechanical opinion about SBS and falls, the appellate court concludes that the trial court abused its discretion in excluding this opinion T by Dr Jones (as distinguished from his experimental results.) Nonphysician experts on medical topics are accepted. Appellant also argued that since SBS is medically controversial, medical opinion about it lacks general acceptance for *Kelly* purposes. The court mentions this but does not rule on it.

This case may go up to the California Supreme Court.

The opinion was provided by Dr. John Plunkett via NAME-L.

People v. Stephen Martinez, 2001 WL 1630083 (Colo. App. 2001).

The death of a four month old female who became unconscious in the presence of mother’s boyfriend. Child was the product of a previous relationship, left in the care of defendant boyfriend. Boyfriend first said child just started choking. Later he said he shook the baby, and while he was shaking her, her head accidentally hit the crib. Autopsy showed an occipital skull fracture and apparently another skull fracture (the Denver *Paste* story mentions two skull fractures), “localized subdural and subarachnoid hemorrhages” (per headnote), ONSH, RH. D was convicted of first degree murder, requiring the mental state of intent to cause serious injury or death. The issue at trial was defendant’s mental state. Whether the degree of force he used was such as to allow the jury to infer intent to cause serious injury. He said he did just minimal shaking. Prosecution pediatrician was allowed to testify over objection that the amount of force was equal to a fall from a multiple story building. Defense objected on relevancy grounds and on Rule 403 prejudice-outweighs-probative-value grounds. Prosecution in final argument based its intentionality theory on this degree-of-force testimony. *Held*: the testimony was *irrelevant*, because it dealt with only the *maximum* degree of force that could cause a SDH, not the minimum degree of force that could cause one. The issue of defendant’s state of mind dealt with what could have been the *minimum* degree of force used by defendant. Hence, to use the court’s words, “the evidence presented through the pediatrician did not shed any light on this issue.” And it was clearly not harmless error.

This case is on appeal by the State. A story in the Denver *Paste* by Howard Pankratz quotes a former child abuse prosecutor to the effect that this decision is “devastating,” apparently because prosecutors have relied on this degree-of-force multistory building testimony for years in AHT cases. And quotes a defense attorney to the effect that this type of T is misleading because it substitutes maximum possible force for minimum possible force, and T has to be precisely tailored. Mr Pankratz quotes another prosecutor to the effect that minimum-possible-force T wd lead to speculation, because the minimum force to cause an SDH could be quite low, consistent with a low-height fall, even though we know that is a mere theoretical possibility and could not possibly be the real cause of these deaths. It is not consistent with reasonable

medical certainty, and defense experts should not be allowed to testify outside the range of reasonable medical certainty.

Intracranial hemorrhage in children younger than 3 years: prediction of intent. Wells RG, Vetter C, Laud P. Arch Pediatr Adol Med 2002 Mar; 156(3): 252-257. From the abstract: "To determine whether certain CT imaging patterns ... help predict intentional compared with unintentional injuries." Retrospective case series over a 10 year period. 293 children under 3 with ICH. They somehow knew after the fact (presumably from final clinical or medicolegal evaluation) whether the injuries were or were not intentional. They found that they could achieve a SPEC of 83% (with a 95% confidence interval 74% to 89%) by using a combination of four CT variables:

- convexity SDH
- interhemispheric
- hygroma
- absence of a skull fracture

Diffusion-weighted magnetic resonance imaging in shaken baby syndrome. Biousse V, Suh DY, Newman NJ, Davis PC, Mapstone T, Lambert SR. (Emory Univ). Am J Ophth 2002 Feb; 233(2): 249-255. Selected 26 children aged 6 weeks to 24 months (mean age 7 months) with acute SDH and either suspected SBS or confirmed SBS. 18 had confirmed SBS. Of these, 17 had RH. Of the total 26 pts, all had SDH, 10 had occult fractures, 18 had RH (17 confirmed shakens and one not confirmed). One confirmed shaken had no RH. All confirmed shakens had abnormal DWI. The DWI showed larger lesions than conventional MRI. DWI can detect RH independently. Found that DWI showed suggestive of diffuse or posterior cerebral **ischemia**. "DWMRI characteristics suggested cerebral ischemia, which appears to play a major role in SBS. ...In all patients with confirmed SBS, DWIMRI... suggested diffuse or posterior cerebral ischemia, in addition to subdural hematomas, in the pathogenesis of this disorder." Compare Geddes, 2001, above.

The spectrum of postmortem ocular findings in victims of shaken baby syndrome. (letter) Brownstein S, Dorey MW. Can J Ophth 2002 Feb; 37(1): 4. Discussing Marshall et al., Can J Ophth 2001 Dec; 36(7): 377-383, above.

Neuropathology of inflicted head injury in children. (letter) Parulekar MV, Elston JS. Brain 2002 Mar; 125(Pt 3): 676-677.

Ophthalmology of shaken baby syndrome. Levin AV. Neurosurg Clin N Amer 2002 Apr; 13(2): 201-211. From the abstract: Numerous pre-retinal, intraretinal, and subretinal hemorrhages extending out to the edges of the retina and/or splitting of the retina (traumatic retinoschisis) seem to be particularly indicative of shaking, with a very narrow differential diagnosis. Shaking itself appears to be a key element in creating hemorrhagic retinopathy.

Traumatic axonal injury after closed head injury in the neonatal pig. Raghupathi R, Margulies S. J Neurotrauma 2002; 19: 843-853. See below under "Biomechanics."

Subdural hematoma in term infants. Chamnanvanakij S, Rollins N, Perlman JM. (Dallas) Pediatr Neurol 2002; 26: 301-304. From the summary by Penny Grant in the *Quarterly*: Reviewed CT scans done on 26 term neonates who had some symptom such as duskiess, apnea, full fontanelle, etc. This was out of 50,000 term deliveries (.05%). Most of them were apparently not attributed to birth trauma but were idiopathic. All were less than 3mm thick. Some were infratentorial. It was suggested that all were associated with brain parenchymal injury, whether visible or not, because the symptoms were not explained by the small SDH's. Compare Ken Feldman, 2001 and Rogers, 1998 and Loh, 2002, below.

Acute subdural hematoma in infancy. Loh JK, Lin CL, Kwan AL, Hwong SL. (Kaoshung) Surg Neurol 2002 Sep-Oct; 58(3-4): 218-224. From the abstract: "Acute SDH in infants is distinct from that occurring in older children or adults because of differences in mechanism, injury thresholds, and the frequency with which the question of nonaccidental injury is encountered." They reviewed retrospectively the records and

films of 21 cases of acute SDH in infants aged 6 days to 12 months. The most common cause of the injury was shaken baby syndrome. The most common clinical presentations were seizure, retinal hemorrhage, and consciousness disturbance. The 8 patients with large hematomas were evacuated; the 13 smaller hematomas were managed conservatively. Of the 8 who were evacuated acutely, none developed chronic SDH. Of the 13 who were managed conservatively, 11 went on to develop chronic SDH's which had to be evacuated by burr hole. Of the chronics, 62% made a good recovery, 19% had moderate disability, 14% had severe disability, and 1 (5%) died. Does not comment on whether brain atrophy was observed in either group. "Conclusion: infantile acute SDH if treated conservatively or neglected, is an important cause of infantile chronic SDH." Seems to be saying that even the small hematomas in this setting are not benign, because they lead to symptomatic chronic SDH with resulting morbidity. [By JKR: Interesting that SBS was "the most common cause" but not the only cause. By JKR: these neurosurgeons are assuming that the hematoma, not parenchymal injury, is the cause of the morbidity. But they do make their point that acute SDH seems to be the definite precursor of chronic SDH. I wonder what Mary Case would make of that.]

Retinal haemorrhages in infant head injury. Clark BJ, Adams GG, Luthert PJ. Brain 2002 Mar; 125(Pt 3): 677-678.

The spectrum of postmortem ocular findings in victims of shaken baby syndrome. Can J Ophth 2001 Dec; 36(7): 377-383. See letter, Can J Ophth 2002 feb; 37(1): 4.

[A postmortem on "pure" subdural hemorrhages in infants and toddlers.] Maxeiner H. Klin Paediatr 2002; 214: 30-36. Cited by Morad et al. (2004) for the proposition that Maxeiner "described a series of 17 children who died of abuse. He noted that children, who presented dead or in a deep coma with acute brain edema, typically did not have significant subdural hemorrhage despite multiple ruptures of bridging veins at autopsy." See Morad et al. (below) for a complete review of cases of SBS without intracranial hemorrhage, of which there are as of the end of 2004 16 reported cases. Morad et al. postulate that rapidly developing cerebral edema compresses the subdural and subarachnoid spaces so that blood cannot get into them, and tamponades the broken bridging veins.

Unexplained SDH in young children: is it always child abuse? Fung ELLW et al. Pediatrics International 2002; 44: 37-42. (Hong Kong). From the summary by Randy Alexander in *Quarterly*: Retrospective study of 9 children aged 0-24 months (all but two were under 12 months). Seven had multidisciplinary child abuse conferences and eight had ophthalmology consultations. Seven had normal bone surveys, two did not have bone surveys. Found 4 abuse, 5 nonabuse.

The criteria for nonabuse were apparently: no other injuries, good social circumstances, non-changing history, and negative multidisciplinary review.

The nonabuse cases had:

Case 1: 1 mo w fever, L frontoparietal acute SDH plus chronic L SDH, small L RH, developed spastic quadripl and severe devel delay.

Case 2: 14 mo fell from chair: occ scalp hematoma, SDH, EDH, devel RUE paraparesis and dev delay.

Case 3: 10 m fell from standing position: Extensive bilat RH, acute SDH, recovered.

Case 4: 2m fever LOC bilat SDH, devel spastic quadripl

Case 5: 24m no hx of trauma: + RH NOS, acute SDH, spastic quadripl

"We believe that, despite a magnitude of opinion to the contrary, the issue of whether 'trivial' head trauma can cause SDH and RH remains unresolved." Argue that the prevailing view that unexplained subdural hematoma and retinal hemorrhages are pathognomonic for child abuse is "a self-fulfilling prophecy." (For such a definition of SBS see Morad et al., 2004, below.) Randy Alexander criticizes this article on the

ground that the severe brain injury could not have happened from the minor histories given. The brain injury, not the hematoma. He says the authors should not have focused on the hematoma, but should have asked themselves how such severe parenchymal injury could result from no trauma or an own-height fall. Both sides accuse each other of circular reasoning. The “self-fulfilling prophecy” comment in this article is cited by Professor C.A.B. Clemetson of Tulane in one of his articles arguing that an SBS picture could be caused by infantile scurvy (Barlow’s disease); see above under “Differential diagnosis -- specific disease entities” and “Vaccine SBS.”

Massive subdural haematomas in Menkes disease simulating shaken baby syndrome. Nassogne M-C, Sharrard M, Hertz-Pannier L, et al. Child’s Nerv Sys 2002; 18: 729-731. Case report of a 9 week old male with seizures, abnormal CT scan. No RH. Found several months later to have developed severe disability and large extraaxial fluid collections. Reported in the Quarterly for April 2003, where Dr Reece comments that it is a straightforward case of Menkes disease as he has ever seen. SBS was never in the differential.

Pediatric spine and spinal cord injury after inflicted trauma. Ghatan S, Ellenbogen RG. (Seattle) Neuros Clin N Amer 2002 Apr; 13(2): 227-233. Rare sequelae of intentional trauma. Easily overlooked. Probably underreported. But the upper cervical spine and brain stem “may significantly contribute to the major morbidity, mortality, and neuropathology in shaken infants.” These structures are highly vulnerable to shaking injury on theoretical grounds, and outcomes with cognitive delay due to hypoxic damage (global brain injury) could have occurred secondary to brainstem and high cervical cord injury. See also under “Cervical Spine,” above.

Radiology of nonaccidental trauma. Rustamzadeh E, Truwit CL, Lam CH. Neuros Clin N Amer 2002 Apr; 13(2): 183-199. Reviewing Duhaime’s retrospective study of outcomes, infants who were unresponsive at presentation or who required intubation at presentation mostly remained vegetative or severely impaired at followup. In those with acute seizures, 60% were severely impaired while [about half] had moderately severe to good outcomes. Those under 6 months generally had severe disability. CT findings of diffuse hypodensity (black brain) or loss of gray-white differentiation led to severe disability on followup. Focal areas of hypodensity were 50-50 as between good outcome and severe disability outcome.

Correlation between retinal abnormalities and intracranial abnormalities in the shaken baby syndrome. Morad Y, Kim YM, Armstrong DC, Huyer D, Mian M, Levin AV. Am J Oph 2002; 134: 354-359. Retrospective chart review of 75 SBS cases that had dilated indirect ophthalmoscopy and CT/MRI in a major referral center. Found:

Evidence of impact	40%
No evidence of impact	60%
SDH	93%
SAH	16%
EDH	1%
Cerebral edema	44%
Infarction	12%
Intraparenchymal blood	8%
Papilledema	5%
Ev of increased ICP*	39%
Confluent RH	82%
Traumatic retinoschisis	32%

* Ev of incr ICP consisting of one or more of: papilledema, widespread bilateral cerebral edema, unilateral edema with midline shift, large intracranial hemorrhage with radiographic compression of brain.

No correlation between laterality of the intracranial injury and laterality of the RH. No correlation between evidence of impact and RH. No correlation between increased ICP and RH.

Ocular injuries in shaken baby syndrome. Tsao K, Kazlas M, Weiter JJ. Int Ophth Clin 2002 Summer; 42(3): 145-155.

A flask full of jelly: the first in vitro model of concussive head injury -- 1830. Feinsod M. Neurosurgery 2002; 50: 386-391. See below under "Biomechanics."

Biomechanics and neuropathology of adult and paediatric head injury. Ommaya AK, Goldsmith W, Thibault L. Br J Neuros 2002 Jun; 16(3): 220-242. See below under "Biomechanics."

Can the initial history predict whether a child with a head injury has been abused? Hettler J and Greenes DS. Pediatrics 2003 Mar; 111(3): 602-607. PVP and SPEC to predict whether "definite abuse" or "not definite."

Retrospective review of 163 patients 0-3 admitted with traumatic intracranial injury. They excluded all natural causes such as AVM. 49 of the admissions (30%) were definite abuse.

<u>thing</u>	<u>SPEC</u>	<u>PVP</u>
no history of trauma	.97	.92
persistent neurol abn at discharge w hx of no or mild trauma	1.00	1.00
CPR offered as cause	1.00	
changing history	1.00	
RH		
moderate or high-specificity fractures		
high-specificity skin marks		
delayed presentation	low	low

Abusive head trauma in Maine infants: medical, child protective and law enforcement analysis. Ricci L, Giantris A, Merriam P, Hodge S, Doyle T. Child Abuse & Neglect 2003; 27: 271-283. This was a retrospective chart review using ICD-9 codes to identify head trauma admissions in the 0-24 month age group in two centers in Maine. Identified 95 child admissions for head trauma, of which 20 hospitalizations involving 19 children were selected as likely abuse-related using the following criteria: intracranial trauma plus one or more of the following: confessed or witnessed assault, inconsistent history, suspicious bruises, suspicious fractures, extensive retinal hemorrhages. Determination of the inconsistency of the history was made subjectively by author Ricci "using a model similar to Duhaime et al. (1992)."

The presenting complaint was a minor accident (e.g. fall less than 4 feet) for 12 of the children (60%). Eight injuries were claimed to have been witnessed, but none was witnessed by more than one adult. There was a history of prior injury in 30%, a history of prior symptoms that in retrospect were suggestive of abuse in 45%, and a history of prior medical evaluation for signs and symptoms possibly abuse-related in 65%. (Gives what the symptoms were.) At initial hospital presentation, the symptoms and findings were

coma	9	(45%)
apnea	6	(30%)
tense fontanelle or enlarged HC	11	(55%)
bruising specific for inflicted	12	(60%)
ev of prior injuries	15	(75%)
RH	19	(95%)
skull fracture	9	(45%)
rib fractures	3	(15%)
metaphyseal fractures	2	(10%)
long bone fractures	2	(10%)
SDH	19	(100%)
cerebral edema	10	(53%)
parenchymal injury	6	(32%)

Two diagnoses were missed by the hospital at the time. One because the family presented well, and the other because the providers believed the history of an accidental injury.

In their discussion, the authors comment on the following topics: 1. The frequency of AHT compared to all head trauma in infants and children (21%). 2. The forms of presentation of AHT. 3. AHT being missed by providers. 4. Bruising. 5. Evidence of prior injuries. 6. LP's. 7. Risk factors. 8. Identifying the perpetrator. I will summarize their comments.

1. AHT was 21% of all admissions for head trauma in this age group (age 2 weeks to 17 months).

Dr Reece's review in the *Quarterly* for July 2003 says had 19 cases of AHT out of 95 infants and young ch adm for head trauma (21%). Of the 19 AHT cases, the history in 60% was a minor event such as a short fall. No fall was w by more than one adult.

hx of previous injury		30%
hx of previous injury or findings of previous injury		75%
hx of previous sx suspicious for abuse at the time		45%
hx of previous sx suspicious for abuse in retrospect		65%
irritability	100%	
vom	22%	
lethargy	44%	
inflicted bruises		60%
skull fx		45%
rib fxx		16%
long bone fxx		10%
SDH		100%
fatal outcome		16%
parental substance abuse		53%
domestic vi		42%
criminal record		42%
difficult child		26%
persistent crying		21%

In 93% of the cases where a perpetrator was identified by law enforcement, that individual was alone with the victim at the time sx first developed 93% of the time. Three defendants were acquitted. Law enforcement identified conflicting medical opinions as an obstacle to successful prosecution in 15% of the cases.

The article reports on p. 276 that "In 4 of the 19 cases, the perpetrator *confessed* to injuring the child. One child was shaken because of apparent jealousy, one was shaken because of crying, one was shaken because of a toileting accident, and one child was slammed down in anger." [emphasis added -- JKR]

Nonophthalmologist accuracy in diagnosing retinal hemorrhages in the shaken baby syndrome. Morad Y, Kim YM, Mian M, Huyer D, Capra L, Levin AV. J Pediatr 2003; 142: 431-434

Shaken baby syndrome in Canada: clinical characteristics and outcomes of hospital cases. King WJ, MacKay M, Sirnick A. Can Med Assoc J 2003; 168: 155-159. From Dr Block's review in the *Quarterly*: Reviewed 364 cases over ten years. Ages 7 days to almost 5 years, median age 4 mos. SDH 79% by CT, 87% by MRI. SAH 32% by CT, 23% by MRI. RH 76%. Cervical spine 4%. Male perps 70%. Often presented with lethargy, symptoms easily missed, nonspecific. 19% fatal outcome, only 18% made a complete recovery.

Canadian Joint Statement on SBS. Located on the Health Canada web site. http://www.hc-sc.gc.ca/dca-dea/publications/jointstatement_web_e.html The primary author seems to be Marcelina Mian. The web site states that it was last updated 9-2-02.

Definition: “Shaken Baby Syndrome is a collection of findings, all of which may not be present in any individual child with the condition. Injuries that characterize Shaken Baby Syndrome are intracranial hemorrhage; retinal haemorrhage; and fractures of the ribs and at the ends of the long bones. Impact trauma may produce additional injuries such as bruises, lacerations or other fractures. Shaken Baby Syndrome is a condition that occurs when an infant or young child is shaken violently, usually by a parent or caregiver. Some experts believe that impact trauma to the head is a necessary component of the mechanism of injury. Signs of impact may or may not be visible because the impact which produces sudden deceleration of the head may be against a soft object such as a mattress.”

What is the incidence? Currently there is no definitive answer to the question of how many babies are affected by SBS in Canada. The incidence of SBS may be severely underestimated due to missed diagnosis and underreporting.

Which children are most at risk? SBS can occur at any age but occurs most frequently in infants less than one year of age. A baby's demands, especially crying, can become the trigger for a frustrated parent or caregiver to shake a child. Infants are particularly susceptible because of their relatively large heads, heavy brains, and weak neck muscles and because they are shaken by people who are much larger and stronger than they are.

How Forceful a shaking causes injury? The severity of the shaking force required to produce injury is such that it cannot occur in any normal activity such as play, the motions of daily living, or a resuscitation attempt. The act of shaking that results in injury to the child is so violent that untrained observers would immediately recognize it as dangerous.

How is the brain injured? Violent shaking has its most serious effect on the infant's head, causing it to whip backward and forward and to undergo rotational forces. The shaking causes the shearing of blood vessels around the brain, leading to a subdural haematoma (a haemorrhage around the brain). The brain itself may be injured as it smashes against the skull during shaking. Nerve cells in the shaken brain may be damaged or destroyed. As a consequence of these injuries, brain swelling and a lack of blood and oxygen may result, producing further damage. The resulting brain dysfunction can be manifested in a number of ways.

What are the signs and symptoms of the injury? Infants who have been shaken may have symptoms ranging from irritability or lethargy and vomiting, to seizures or unconsciousness with interrupted breathing or death. Babies with mild shaking have symptoms similar to a viral illness. Caregivers and even physicians who are not aware of what has happened to the baby may not detect the head injury, or rib and long bone fractures, and may attribute the baby's fussiness to a more benign cause such as the 'flu.' The more serious the child's neurological injury, the more severe the symptoms and the shorter the period of time between the shaking and the appearance of symptoms. From the time of the shaking these children do not look or act as usual -- they may not eat or sleep or play normally.

Babies who are shaken may be brought to medical attention by a caregiver who offers no history of injury, a vague account of events, or an explanation that is not consistent with the physical findings. Unless the physician is aware of the possibility of abuse and knowledgeable about the signs of SBS, the cause of these children's symptoms can be missed.

Giving a lengthy list of references.

Retinal and subdural haemorrhages: Aoki revisited. Gardner HB. Br J Ophth 2003 Jul; 87(7): 919-920.

Evidence-based medicine and shaken baby syndrome. Part I: literature review, 1966-1998. Mark Donohoe, MD (New South Wales). Am J Forens Med P 2003 Sep; 24(3): 239-242. Using the heuristic categories of EBM, this author makes the point that in most of the literature during this historical period the reasoning was circular: they used the complex SDH + RH as a selection criterion for babies having possible infliction, and then, since there usually was no other clinical evidence, “found” that all the babies with SDH + RH were inflicted. As he puts it, “Many of the authors repeated the logical flaw that if RH and

SDH are nearly always seen in SBS, the presence of RH and SDH ‘prove’ that a baby was shaken intentionally.” (p. 241), and “Many studies ... make the obvious logical error of selecting cases by the presence of the very clinical findings and test results they seek to validate as diagnostic. Not surprisingly, such studies tend to find their own case selection criteria pathognomonic of SBS.” (p. 239) Most of the studies had no controls, which “would normally not occur” in science. Evidence-based medicine uses “quality of evidence” ratings to reach a QER or “quality of evidence rating” for each study. These QER ratings are as follows:

I: consistent evidence obtained from more than 2 independent randomized and controlled studies or population-based epidemiologic studies

II: consistent evidence from 2 randomized controlled studies, a single multicenter randomized controlled study, or a population-based epidemiologic study

III-1: consistent evidence from 2 or more well-designed and controlled studies

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

III-3: evidence from a single case study or a single cohort study

III-4: conflicting evidence from 2 or more well-designed and controlled studies

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

Under this schema, the author has reviewed 54 articles from the period 1966-1998, ending with Jayawant’s “important study.” He assigned each article to one of four categories:

randomized controlled trials*	1
case series	26
case reports	12
other (reviews, opinions, etc.)	15

Results: Of the case series, 21 had no control groups and 3 had inappropriate control groups. Only four case series had selection criteria based on confession or conviction; the others had selection criteria either unstated or by suspicion only. Inappropriate control groups consisted of head impact trauma patients, not healthy patients or patients with unrelated illnesses.

Overall, “There was no evidence on the subject of SBS that exceeded QER III-2 by the end of 1998... The majority of the evidence achieved only a level of QER IV... [T]he data available in the medical literature by the end of 1998 were inadequate to support any standard case definitions, or any standards for diagnostic assessment.” (p. 241)

Comment by JKR: The EBM schema is designed for clinical trials. By definition, clinical trials are impossible in child abuse medicine.** So what is the point of applying the EBM schema to this field? Obviously, the result of applying inappropriate heuristic criteria to a field to which they are inapplicable is going to be low EBM scores for that field, but what does this mean? Well, the whole effort boils down to the author’s very apt observation that I quoted at the beginning, that most of the studies in SBS are guilty of circular reasoning. The whole business about “evidence-based medicine” is really just a rhetorical vehicle for making that point. [See the comment on vague inclusion criteria in Geddes et al., article I, 2001. – JKR].

* The single randomized clinical trial was a trial of electroretinography that didn’t work.

** The author acknowledges that “controlled” studies of shaking are not possible, citing “clear data deficiencies arising from difficulties in performing experiments. It is clearly unethical to intentionally shake infants to induce trauma, and there is an obvious problem with studies and reports that rely on either

indirect or disputed evidence of the occurrence, severity, or type of trauma.” (p. 239) The author deals with this problem in the course of reviewing the case-series articles, by saying that since most of them had either no control groups or inappropriate control groups, they would normally not even be included in a database for evidence-based medicine, but he includes them anyway (as QER III-2) because there aren’t any other articles. But he fails to consider the implications of this for the applicability of the whole EBM schema to SBS or other child abuse research. If the author had reviewed the literature on burns or fractures or sexual abuse, he would have encountered exactly the same difficulty of lack of controls and vague selection criteria. The result would again be low QER scores. Would he be forced to conclude that there is “no evidence” for abusive burns, fractures, and sexual abuse?

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. From the abstract: “A histological review of dura mater [slides] taken from a postmortem series of 50 paediatric cases aged up to 5 months revealed fresh bleeding in the dura in 36/50, the bleeding ranging from small perivascular haemorrhages to extensive haemorrhage which had ruptured onto the surface of the dura. Severe hypoxia had been documented clinically in 27 of the 36 cases. In a similar review of three infants presenting with classical ‘shaken baby syndrome,’ intradural haemorrhage was also found, in addition to subdural bleeding... 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. Hypoxia with brain swelling would also account for retinal haemorrhages, and so provide a unified hypothesis...” Commented on in Dural haemorrhage in nontraumatic infant deaths: does it explain the bleeding in “shaken baby syndrome?” A response. Smith C, Bell JE, Keeling JW, Ridsden RA. *Neuropathol Appl Neurobiol* 2003 Aug; 29(4): 411-412, saying that this “microscopic subdural haemorrhage” is just a well-known autopsy artifact. Author’s reply, pp. 412-413. I have commented in detail on this article in connection with my published review of a successor article by Geddes & Whitwell published in FSI for 2004. See below. Mary Case also disagrees with this view as to the possible anoxic etiology of SDH, SAH, and RH. See her response to the article by Lucy Rorke in *Inflicted Childhood Neurotrauma* (AAP, 2003), below under “Shaken.” --JKR

Shaken adult syndrome revisited. (letter) Geddes JF and Whitwell HL. *Am J Forens Med P* 2003 Sep; 24(3): 310-311. This Palestinian prisoner with chest bruising, no head bruising, extensive unilateral SDH with midline shift, DAI, and retinal hemorrhages, was considered by Pounder and by Kirschner as having been shaken, even though there was no confession of shaking. The victim survived three days. We have re-evaluated this conclusion in the light of our current knowledge about DAI (citing Geddes, Hackshaw, Vowles et al., *Brain* 2001). Work in the 1990’s has shown that a degree of TAI may be found in virtually all head injuries. Also, that there is a spectrum of TAI as to its extent -- ranging from DAI as the most severe and extensive form, widespread throughout the brain, the clinical features of which are attributable principally to the involvement of the brain stem, out to only scattered hemispheric axonal damage whose sequelae are unknown and may be clinically undetectable. Because of this distribution spectrum, the only way a diagnosis of DAI can be made is by wide sampling along with immunochemistry. Silver stains are no good. In Pounder’s case, we have had the neuropathology report translated from the Hebrew. We find that the neuropathologists took 40 brain sections but did not identify where they came from. Apparently no special stains were used. “Shrinkage beads” are described “here and there.” No attempt was made to distinguish between secondary ischemic damage and primary traumatic damage. There is no record of whether the posterior fossa structures were involved, “a prerequisite for DAI.” Result: DAI is not diagnosable from this report. Also pointing out that a mass-effect subdural such as this man had is unusual in child abuse. Overall result: “[This case], although of great interest, is not relevant to so-called shaken baby syndrome.” See Kemp et al., 2003, next below, for further commentary by Dr Geddes on the same score.

Apnoea and brain swelling in non-accidental head injury. Kemp AM, Stoodley N, Cobley C, Coles L, Kemp KW. *Arch Dis Chil* 2003; 88: 472-476. (UK) Uses hospital records (no autopsies) from 65 patients under two with inflicted SDH (age range 19 days to 23 months). of whom 34% had clinically documented apnea. As far as brain swelling,

apnea	48% had brain swelling documented
no apnea	19% had brain swelling documented

[By JKR: These data indicate that apnea independently contributes to brain swelling, which seems obvious anyway.]

The authors go on to ask whether these data shed light on the question, “What degree of trauma is associated with non-accidental head injury?” They note that 85% of the patients had extracranial injuries, mostly fractures. They conclude that the data are consistent with, albeit not probative of, the proposition that since apnea independently contributes to brain swelling, the only degree of trauma that is needed to cause brain swelling is the degree of trauma needed to cause apnea, and that may not be very much. They quote Geddes II (2001, above) to the effect that “It may not be necessary to shake an infant very violently to produce stretch injury to its neuraxis.”

The article is followed by a commentary by Dr Geddes to the effect that it is HIE, not mechanical brain trauma, that causes the brain impairment and death in SBS cases, and further, that “The biomechanical evidence that shaking can produce subdural and retinal hemorrhage has recently been shown to be dubious.” The SDH is merely an indicator that there has been some degree of trauma to the head. (See also her commentary, above, on the “shaken adult” case).

Dr Block comments on Dr Geddes’ commentary in the October 2003 issue of the *Quarterly*. He calls Dr Geddes’ comments “troubling,” because neither this article nor Dr Geddes’ own work shows that mechanical brain trauma is not the underlying cause or part of the cause of the neurological outcome.

[Paraphrased by JKR]

Child abuse: radiologic-pathologic correlation. Lonergan GJ, Baker AM, Morey MK, Boos SC. From the Archives of the AFIP. RadioGraphics 2003. (See also under “Fractures -- in general,” and “Abdominal radiology.”) This monograph has a good short summary of the SBS controversy as of 2003. “Recently, the rotational acceleration hypothesis itself has been questioned.” Because (1) rarely is neck injury found, although cervical cord/ cervical nerve root injury has been found (citing Shannon, Smith, Deck et al., 1998; Geddes I, 2001; Geddes II, 2001). (2) Brain pathology reveals hypoxic-ischemic injury, not trauma, and this might result from brain stem or basal ganglia effects on cardiorespiratory function. “Unfortunately, this theory fails to address the additional findings of SDH and retinal hemorrhage.” Further discussion: The term “shaken baby syndrome” has come to be loosely applied. “Impact injuries are not strictly consistent with the diagnosis of shaken baby syndrome... Additional injuries not explained within the shaking hypothesis must be explained by additional or alternate diagnoses. It is important that abusive injury be recognized as abuse independent of the biomechanical controversies.”

Anthropomorphic simulations of falls, shakes, and inflicted impacts in infants. Prange MT, Coats B, Duhaime A-C, Margulies SS. J Neurosurg 2003 Jul; 99(1): 143-150. See below under “Biomechanics.”

Can shaking alone cause fatal brain injury? A biomechanical assessment of the Duhaime shaken baby syndrome model. Cory CZ, Jones MD. (Cardiff School of Engineering). Med Sci Law 2003 Oct; 43(4): 317-333. See below under “Biomechanics.”

Neuropathology of inflicted childhood neurotrauma. Rorke LB. In: Reece & Nicholson, eds., *Inflicted Childhood Neurotrauma: Proceedings of a conference...* AAP, 2003. First, she states that while subdural hematomas are well documented, more subtle neurological findings are often missed because of inadequate autopsy technique or because “the pathologist is uninformed or inexperienced.” Says that following a strict autopsy protocol is necessary to avoid this. She offers the Philadelphia autopsy protocol here, as an appendix. Next, she comments point by point on certain types of autopsy findings in inflicted childhood neurotrauma:

1. **External injury is commonly absent or subtle.** Citing Gilles & Nelson, cerebral complications (1998); Munger et al., ocular & associated (1993); and Duhaime et al., long-term outcome (1996). But external lesions “are sometimes found.”

2. Scalp contusions: reflection of the scalp often uncovers them, usually parietooccipital.
3. Skull fractures occur in 25-40%. Usually parietooccipital.
4. Intracranial hemorrhage:
 - a. Epidurals of the head are rare. As to the spinal cord, it is common in the rostral cervical cord in whiplash cases, sometimes lower down. But cautions that spinal epidural by itself should not be interpreted as evidence of abuse.
 - b. Subdural occurs almost universally, although Case et al. in the NAME Position Paper (2001) and Geddes I (2001) report it in as few as 70%. Hematomas may vary in age if repetitive trauma.
 - c. Subarachnoid is reported by the Position Paper to be almost universal in fatal cases, usually parasagittal in location. "Rarely, complications consequent to coagulation defects or impaired cerebral circulation leading to venous thrombosis following trauma may lead to prominent SAH."
 - d. Parenchymal/ intraventricular hemorrhage. Isolated intracerebral hemorrhage should raise suspicion of a natural process such as an AVM or a coagulopathy.
5. Contusions and lacerations of the brain she classifies into "superficial," "deep," and "brainstem."
 - a. Superficial contusions are not generally found in pure or primarily shaken infants. But they can be found in the **olfactory nerve** and the **gyrus rectus**, where they are easily missed. (Citing the SBS review in NEJM (1998) and her own chapter in Ludwig & Kornberg (1992).
 - b. Deep contusions and lacerations are due to shearing forces and include:
 - gliding contusions
 - DAI (centrum ovale and corpus callosum)
 - ependymal tears
 - tears of the great vein of Galen

Now as to these,

1. Gliding contusions are usually in the frontal lobe white matter. Have no bleeding grossly, but may have a little microscopically. Citing Lindenberg & Freytag.
2. DAI is not often obvious grossly. It requires H&E and silver stains and/or BAPP. Its pathogenesis is a matter of dispute because of the difficulty of distinguishing it from ischemia. Citing Geddes II and Graham & Gennarelli in Greenfield's 6th edition, 1997. Identifying callosal lesions may be difficult if the infant has been maintained on a respirator for more than 24 hours. The lesions are usually in the splenium. Look microscopically for [retraction balls], red cell collections, or inflammation.
3. Ependymal tears, usually in the lateral ventricle, allow egress of necrotic brain into the CSF, as seen microscopically. [Are these distinguishable from artifacts of respirator brain? --JKR]
4. Tears of the great vein of Galen are rare but should be considered when there is a large accumulation of blood in the pineal-tectal region.
- c. Contusions and tears of the brainstem. Most common at the pons and locus ceruleus, which is significant because major brainstem pathways of cardiorespiratory control go through this area. Also found in the medulla and cervical cord.
6. Spinal cord examination is very important because if traumatized it could account both for the "malignant brain edema" of Bruce et al. (1981) and the finding of severe brain dysfunction in pure shakings

given that “Duhaime and colleagues are probably correct that lethal trauma to the brain itself does not result from shaking alone.” (p. 170). The “malignant brain edema” of Bruce and the brain pathology of pure shaking both may result from acute anoxic encephalopathy due to cervical cord injury. “Systematic careful study of the spinal cord, roots, and surrounding tissue often discloses hemorrhages within nerve roots and ganglia and epidural hemorrhages, but less often primary damage to the cord.” Also watch out for vertebral artery tears if you find subarachnoid blood in the medulla and cisterna magna.

7. Anoxic encephalopathy and infarctions. (The radiographic counterpart is the “black brain.”). Very common (77% in Geddes II). Deterioration of the tissue due to respirator brain makes it very difficult to diagnose infarctions (or DAI) microscopically the longer the child has been on the ventilator.

8. Chronic CNS lesions. These children come with a history of impairment.

Summary: The neuropathological diagnosis of inflicted childhood neurotrauma can be made with a reasonable degree of medical certainty when there is a characteristic pattern of findings seen in the context of the clinical history. Namely,

1. Clinical features

- a. An apparently well child who presented with sudden cardiorespiratory and neurologic decompensation
- b. Few if any external lesions of the head, face, or neck
- c. SDH seen on radiology with or without black brain
- d. RH

2. Pathological features

- a. SDH
- b. Cerebral swelling
- c. Contusions or lacerations of the olfactory bulbs, tracts, or gyrus rectus
- d. Partial or complete transection of the corpus callosum
- e. Rostral spinal injury -- soft tissue hemorrhage, nerve root avulsion, with or without cord injury
- f. Acute neuronal necrosis
- g. DAI
- h. RH
- g. ONSH

Gives the appendix protocol for doing the head/brain/spine/cord dissection and microscopy. The sections are those listed in Rationale and technique for examination of nervous system in suspected infant victims of abuse. Judkins AR, Hood IG, Mirchandani HG, Rorke LB. Am J Forens Med P 2004 Mar; 25(1): 29-32. [See above under “Autopsy technique.”]

Response by Vernon Armbrustmacher, MD. He asks the question, **What is the fatal lesion in AHT?** The SDH is small. The SAH is small. What is the fatal lesion of the brain? The DAI lesions (small parenchymal hemorrhages) typically seen in adults “are rarely observed” in infants who die of SBS. (p. 183) Using BAPP, “some studies” (Vowles et al., 1987; Shannon et al., 1998; Gleckman et al., 1999) have suggested that TAI is present in these infants. But then there is Geddes. So the BAPP studies are “in conflict.” [Geddes herself is simultaneously pushing this point, more strongly. See Geddes JF and Whitwell HL, Inflicted head injury in infants. FSI 2004; 146: 83-88, discussed elsewhere herein. –JKR] So if it maybe isn’t DAI, what is it that kills or maims these infants? He likes the Geddes hypothesis of brainstem/ cord trauma leading to apnea. [But what if the baby was not shaken? And what about the geographic distribution of the brain lesions seen in nonfatal cases? --JKR] He also points out that no one knows the amount of force needed to cause TAI.

Response by Mary Case. Agrees that the diagnosis and frequency of TAI in SBS is unknown and needs research. This would also bear on the time-of-injury issue, since DAI is instantaneous unconsciousness, but cervical apnea might not be. Case dismisses the argument of Geddes that SDH, SAH, and severe RH could be caused purely by anoxia. These are definitely traumatic.

Neuroimaging of intraparenchymal lesions predicts outcome in SBS. Bonnier C, Nassogne MC, Saint-Martin C, Mesples B, Kadhim H, Sebire G. (Brussels). Pediatrics 2003 Oct; 112(4): 808-814. Long term outcome. Retrospective chart review of 23 pts w AHT followed up for a mean of 6 years. Found that 61% had severe disability, 35% had moderate difficulties, 1 was normal. Poor developmental outcome was significantly associated with

low initial GCS
severe RH
skull fracture
cranial growth deceleration
intraparenchymal lesions within the first 3 months

The intraparenchymal lesions in question were contusions, infarcts, and other white-matter lesions, all of which were found to lead to long-term atrophy.

Late outcome following central nervous system injury in child abuse. Perez-Arjona E, Dujovny M, DelProposto Z et al. (Wayne State). Childs Nervous System 2003; 19(2): 69-81. This is a literature review. Found that AHT carries a significantly worse prognosis than accidental head trauma. Late followup CT and MRI scans showed cerebral atrophy in 100% and cerebral ischemia in 50%. Also persistent emotional, cognitive, and social problems.

Characteristics of inflicted TBI and noninflicted TBI. Keenan Pediatrics 2004; 114: 633-639. According to Dr Reece's review of the top ten SBS articles of the past ten years presented at the 2006 national SBS conference, this was a prospective study of 152 cases in children under 2. The inclusion criterion was the presence of intracranial injury by CT or MRI. The criterion for dx of iTBI was one of the following: (a) confession, (b) CPS determination, (c) coroner's determination. Had 152 included cases: 80 iTBI, 70 non-iTBI, 2 undet. The mortality of iTBI was 26%. The iTBI were younger. As to the history given, there was no history of trauma in 2/3 of the iTBI group, while zero of the non-iTBI group had no history of trauma. 53% of the non-iTBI group had a documented MVA. A history of a fall was about equally frequent in both groups. As to the presenting complaint, you had

	iTBI	non-iTBI
respiratory changes	32%	0
unresponsiveness	23%	0
seizures	12%	0

As to the clinical findings, you had

	iTBI	non-iTBI
skull fracture	18%	59%
RH	76%	8%
SDH	94%	61%
SAH	11%	23%
EDH	1%	18%
rib fractures	28%	
injuries other than head & ribs	38%	
no external ev of injury	35%	
old injuries	35%	

Dr Reece commented that these data are similar to past data.

Bilateral fourth nerve palsy occurring after shaking injury in infancy. Cackett P, Fleck B, Mulvihill A. JAAPOS 2004; 8: 280-281. (U.K.) A 13 week old male alleged to have fallen. SDH, SAH diffuse RH, and a metaphyseal fracture of the distal tibia. At followup 14 months later he had a fourth nerve palsy.

Inflicted head injury in infants. Geddes JF and Whitwell HL. Forensic Science International 2004 ; 146: 83-88. These authors have previously published a pair of much-discussed articles in *Brain* (2001) proposing that the axonal injury in the cerebra of shaken babies could be purely hypoxic in origin, while those in the caudal medulla and cervical cord were suggestive of hyperextension injury, leading to a hypothesis of traumatic apnea as the initiating event of all the cerebral pathology in AHT or at least in shaken babies. But how could subdural hemorrhage and retinal hemorrhage come from pure hypoxia? Don't these apparently traumatic lesions contradict a hypothesis of purely hypoxic etiology? And what about diffuse axonal injury, a clearly traumatic condition reported to be common in AHT? In the present paper, which is actually a summary of an earlier article in *Neuropathology and Applied Neurobiology* (29: 14-22, 2003), the authors attempt to answer these objections.

The authors postulate that both subdural hemorrhage and retinal hemorrhages could be artifacts of hypoxia, not necessarily the result of trauma. Their support for this hypothesis is a review of British literature on the autopsy pathology of the dura mater in infants, and their own prospective series of 50 autopsies of fetuses and infants who did not have head trauma, on whom they performed histologic study of sections of the cranial dura mater. The autopsy material ranged from 18 week stillborns to 5 month old infants, all of whom died of causes not involving any trauma. In this material, the authors found histologic evidence of subdural hemorrhage in a maximum of 11. (The actual number is unclear; the authors state that the slides showed blood on the extradural surface in 11 cases, and that "in some cases this was identifiably the subdural surface.") Since this hemorrhage was not detectable grossly in any of the cases, the authors term it "microscopic subdural hemorrhage." They found histologic evidence of intradural hemorrhage (hemorrhage within the dural tissue itself) in 36 cases. The authors' review of the British literature shows that intradural hemorrhage in fetal material has been noted by pathologists as far back as 1930. The authors postulate hypoxia as the cause of these histologic findings, and cite Volpe's *Neurology of the Newborn* for the proposition that hypoxia can cause subdural hemorrhage. The authors' *piece de resistance* is an autopsy photograph of the cranial cavity of a 26 week stillborn who died of abruptio placentae. The photograph shows areas of discoloration of the dura mater at the vertex and along the lambdoid suture, which was identified histologically as intradural hemorrhage with microscopic subdural hemorrhage. The authors postulate that the hemorrhage was caused by intrauterine hypoxia. Thus, from a histologic finding of intradural hemorrhage in 36/50 atraumatic infants and "microscopic subdural hemorrhage" in up to 11/50 infants, the authors postulate that hypoxia could be the cause of subdural hemorrhage found in infants with known or suspected head trauma. As to how hypoxia could cause subdural hemorrhage, the authors suggest that it could be breakthrough bleeding from intradural hemorrhage, with vascular and hemic sequelae of hypoxia such as endothelial growth factor playing a role. They note that cerebral hypoxia is an almost constant concomitant of AHT, as is traumatic apnea.

The authors' review of the literature on the neuropathology of abusive head trauma finds that there is little scientific foundation for the currently conventional view that diffuse axonal injury (DAI) is the characteristic brain injury in this population. The authors dismiss published work postulating DAI in these infants (including the 2001 NAME Position Paper) as being based on "a single limited study" by Vowles et al. (1987), which was performed before the advent of beta-amyloid precursor protein staining and is therefore dismissed as obsolete. The same goes for all earlier work, including Lindenberg & Freytag (1969), Hadley et al. (1989), Brown & Minns (1993), Shannon et al. (1998), and David (1999). With DAI gotten out of the way by this line of argument, and subdural hemorrhage gotten out of the way by their present work on hypoxia, the authors find themselves free to propose that none of the intracranial pathology commonly found in abusive head trauma is diagnostic of trauma, let alone inflicted trauma.

That leaves retinal hemorrhages. The authors argue that these, too, could be an artifact of hypoxia, combined with increased intracranial pressure. This is an argument that has been made before, and the authors repeat it here.

Comment by JKR:

Intradural hemorrhage along the dural sinuses is an artifact of labor and has been well known to perinatal pathologists for decades. (See the published comments of Keeling et al. in *Neuropathology and Applied Neurobiology* 2003; 29: 411-413 for further discussion of this point. See also Keeling J.W., Ed., *Fetal and Neonatal Pathology* (1987), p. 492). The more strenuous the labor and the longer the intrapartum survival, the more prominent this intradural hemorrhage will be. Close examination of the autopsy photograph presented by the authors shows the presence of a cephalhematoma on the external surface of the skull, a sign of precipitous labor which explains the demonstrated intradural hemorrhage.

The next problem is the authors' postulate that intradural hemorrhage could be caused by asphyxia because they found it in 79% of the infants who were asphyxiated. The problem with that is that they also found it in 50% of the infants who were not asphyxiated.

The next problem is that little word "microscopic" placed before the words "subdural hemorrhage." The authors found microscopic subdural hemorrhage in something less than 11 out of their 50 infants. The problem with "microscopic subdural hemorrhage" is that small amounts of blood seen microscopically on histologic slides of the dural surface, particularly near the dural sinuses, can easily result from perimortem leakage or postmortem sedimentation. Such sediments are well known to autopsy pathologists and are an artifact of no pathologic significance. Macroscopic subdural hemorrhage is a different story; it is a highly significant pathologic finding. If the authors had been able to demonstrate even a few cases of macroscopic subdural hemorrhage in their fifty atraumatic infants, they would have produced a truly significant report. As it is, they are merely jacking up a commonplace laboratory artifact into a purportedly revolutionary research discovery.

The fact that the authors' main point can be dismissed should not, however, lead us to dismiss some of the ancillary arguments that are made in this wide-ranging discussion of the pathology of the shaken baby syndrome. I would like to single out two points made by the authors that score hits and point to critical issues in child abuse pathology:

1. The fact that traumatic DAI (or TAI) is seldom found in abusive head trauma. The NAME position paper to the contrary notwithstanding, this is a fact. It needs explaining.
2. The fact that retinal hemorrhages, even very severe ones typical of those seen in abusive head trauma, can be caused by things other than trauma. This is a fact. No one accepts the authors' view that severe RH can be caused purely by anoxia; if it were, it would be seen in near-drownings, near-SIDS, and other anoxic situations. But it does not require trauma. This needs explaining.

Note by JKR: The above article summary is substantially as published in the *Quarterly* for March 2005, and is copyrighted by the *Quarterly*.

Perimacular retinal folds from childhood head trauma. Lantz PF, Sinal SH, Stanton CA, Weaver RG Jr. *BMJ* 2004 Mar; 328: 754-756. See under "RH -- traumatic retinoschisis," above. Case report of a 14 month old toddler who had a big-screen TV fall on top of him, had a complex skull fracture with bilateral perimacular folds. There was no shaking. Abuse was carefully ruled out. This is the first published report of traumatic retinoschisis in a non-SBS setting. There will be others. Includes discussion by Henry Krous of the diagnostic criteria for SBS and controversial nature of some aspects of SBS. This seminal article opened the first serious debate about the validity of the vitreoretinal traction theory of the pathogenesis of RH in SBS advanced by Masicotte et al. and Greenwald et al. The controversy continues. As I have said elsewhere, I think it is only a matter of time before Lantz's abrupt-ICP theory of the pathogenesis is proven right. --JKR

Inflicted childhood neurotrauma (shaken baby syndrome): ophthalmic findings. Forbes BJ, Christian CW, Judkins AR, Kryston K. *Journal of Pediatric Ophthalmology & Strabismus* 2004; 41: 80-88. Literature review aimed at ophthalmologists.

Knight's Forensic Pathology, 3d ed., 2004. New York: Oxford University Press, 2004. Chapter 22, Fatal Child Abuse. Gives an objective summary of the literature on shaking, allowing that shaking can possibly cause SDH, but impact is probably necessary for brain injury, on biomechanical grounds (high strain rate versus low strain rate) plus the finding of Duhaime et al., 1987 that all of 13 alleged shaking fatalities had autopsy evidence of impact, even though none was evident externally, and Howard Bell & Utley, 1993, that 28 instances of infant SDH all had historical or pathological evidence of impact even though three had a history of shaking only. (p. 470) (Knight accepts this much-criticized study, in which several histories of own-height falls and couch falls were accepted as genuine even with skull fractures and fatal SDH. Brian Holmgren, JD states in the summer 2006 issue of the *Quarterly* (p. 16) that Howard Bell & Utley's article "has been ridiculed in the professional literature.")

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. A retrospective multicenter review of 453 cases of AHT in which there were 81 confessions. Study used to examine (a) reliability of confessions, and (b) time of injury. According to the abstract and Dr. Reese's review in the summer 2004 issue of the *Quarterly*,

81 confessions

69 with enough detail to define the historical mechanism of injury

pure shaking	32
pure impact	20
both	17

clinical findings

pure shakings	32
skull or scalp injury	4 (12%)
pure impacts	20
skull or scalp injury	12 (60%)

As to time of injury, in 57 cases the perpetrators described the time of infliction. Of these, 52 (91%) said the baby became immediately symptomatic. In the other 5 cases, the time of onset of symptoms was less clear, but it was always within 24 hours. The authors note that in all cases, the perpetrators' description of the symptoms was consistent with the symptoms that were clinically observed on presentation.

Authors' conclusions:

1. Shaking alone can produce the clinical pattern of AHT (intracranial bleeding, brain injury, retinal hemorrhage). As the authors put it, "Most perpetrators admitted shaking without impact. These data, combined with the relative lack of scalp and skull injury, suggest that shaking alone can produce the symptoms seen in children with inflicted TBI."
2. The symptoms of inflicted head injury in children are immediate.

See Dr Reece's editorial comment in the *Quarterly* for Dec 2005 on Leestma, Case analysis of brain-injured admittedly shaken infants: 54 cases, 1969-2001, where Dr Reece points out that Leestma deliberately omitted this article and several others that document confession shaking cases published subsequent to 2001. This paper by Starling et al. is discussed by Hymel in his short editorial review of the articles pro and con shaking injury, 2005, below. Hymel says that Starling et al. found that 55 out of 81 perpetrators admitted shaking the victim, and 32 of these admitted *only* to shaking. Of those 32, 29 had

SDH (91%), 27 had RH (84%), 2 had skull fracture (6%), and 2 others had scalp swelling. Obviously then, (at least to me --JKR) four of those pure-shaking perpetrators were lying, or their confessions “were incomplete” (Hymel). The final clinical outcomes on discharge as to pure shaking confession cases and impact confession cases were not distinguishable. Hymel does not comment on the significance of this article, but quotes its findings in some detail. At the national SBS conference 2006 Dr Reece included this article in his list of the top ten SBS articles of the past ten years.

Retinal hemorrhages in an 8-year-old child: an uncommon presentation of abusive injury. Mierisch RF, Frasier LD, Braddock SR, Giangiacomo J, Berkenbosch JW. *Pediatric Emergency Care* 2004 Feb; 20(2): 118-120. Case report of **an 8 year old boy** with intracranial hemorrhage and severe cerebral edema/ICP. Police apparently obtained a confession of severe abusive injury including shaking. Conclusion: The diagnosis of SBS is not limited to babies, and the possibility of abusive shaking injury shd also be considered in older children...

The evidence base for shaken baby syndrome. Geddes JF, Plunkett J. (guest editorial) *BMJ* 2004 Mar; 328: 719-720. Relies on the articles of Pat Lantz (case report of nontraumatic perimacular folds in a 14 month old) and Mark Donohoe (Evidence-based medicine and SBS). “The conclusions of Lantz et al. and of Donohoe make disturbing reading, because they reveal major shortcomings in the literature relating to a field in which the opportunity for scientific experimentation and controlled trials does not exist, but in which much may rest on interpretation of the medical evidence.” Then going on to abuse other than SBS, saying that “The recent literature contains a number of publications that disprove traditional expert opinion in the field...” Giving as examples Plunkett’s 2001 *Am J Forens Med P art on fatal low-height falls (q.v.)* “What is the force necessary to injure an infant’s brain? Again, we do not know.”

Followed by an unsigned official editorial saying, *inter alia*, “Controversy surrounds the precise causation of the brain injury, the retinal and subdural haemorrhages, as well as the degree of force required and whether impact in addition to whiplash forces is needed.” Every individual case must be evaluated in detail. “In shaken baby syndrome, it is the combined triad of subdural and retinal haemorrhage with brain damage, as well as the characteristics of each of these components, that allow a reconstruction of the mechanism of injury, and assessment of the degree of force employed.” Goes on to discuss Geddes’ BAPP work as an indication that “The mechanism of brain damage is problematic.” See response letter from 106 doctors engineered by Dr Reece, below.

The evidence base for shaken baby syndrome. Response to editorial from 106 doctors. Reece RM. *BMJ* 2004 May; 328: 1316-1317. (1) Geddes & Plunkett’s initial assertion that doctors believe that the head injury from shaking “is invariably severe” is in conflict with the findings of Alexander, Sato, 1990; Ewing-Cobbs, 1998; Kemp, Stoodley, 2003; Jenny, Hymel, 1999, showing that 30-40% of shakens have evidence of having survived previous shakings. (2) As to RH, reiterates that RH are much more common in abuse, not that they are pathognomonic; no one claims that they are pathognomonic. (3) As to Donohoe’s article, its coverage is limited by the fact that Donohoe used only the keyword “shaken baby syndrome” in doing his literature search, and did not use related terms such as “inflicted head trauma,” etc. Also: low EBM scores are inevitable in the SBS field bc controlled trials are impossible. (4) As to short falls, Plunkett cites his own article, which has significant problems. Note that *contact* subdurals can certainly result from short falls. Short falls can also occasionally cause severe illness or death from a space-taking lesion, or occasionally from malignant cerebral edema. All of Plunkett’s fatal short-fall cases seem to fall into these categories. And even his study showed that death from short falls is exceedingly rare .02%. (5) As to biomechanics, the research is still being done. It is “premature” to conclude from it that shaking cannot cause infant brain injury. And note all those confessions.

Comment by JKR: This last item, on biomechanics, is interesting. Note that the 106 doctors did not claim that biomechanically concluding that shaking cannot cause (primary) brain injury is impossible. They only said it is “premature.” They seem to be admitting that the biomechanical research is pointing more and more in that direction, and that it’s only a matter of time.

Patterns of presentation of the shaken baby syndrome: Four types of inflicted brain injury predominate. (letter). Minns RA and Busuttil A. *BMJ* 2004 Mar; 328: 766. This letter is in response to Geddes & Plunkett’s “evidence base” article, above. These two Scottish physicians here report that they have collected a database of five years of Scottish cases of suspected non-accidental head injury diagnosed after

multiagency evaluation including uncoerced confessions and criminal convictions. Their database shows four typical presentations:

(a) Hyperacute encephalopathy (cervicomedullary syndrome). (6% of cases). Extreme whiplashing resulting in brainstem injury with respiratory arrest. Show by Geddes work in infants with a survival of less than one day to have localized axonal damage at the craniocervical junction, in the corticospinal trax, and in the cervical cord and its nerve roots. These are true whiplash cases. They are seldom seen by clinicians because they present deceased.

(b) Acute encephalopathy. (53% of cases). Depressed consciousness, seizures, increased ICP, apnea, hypotonia, anemia, shock, bilateral subdural haematomas, and widespread haemorrhagic retinopathy. Coexistent rib fractures, metaphyseal fractures, or other non-accidental injuries may be found. This is the classic shaken baby syndrome seen by clinicians and involves repetitive whiplash injury. May be called the shaken impact syndrome if evidence of impact is present. By MRI this type of brains shows ev of widespread shearing injury (diffuse cerebral hypodensities) with convexity and interhemispheric SDH which expand over the first week, white matter shearing with tears and petechiae at the gray-white junction and corpus callosum.

(c) Subacute nonencephalopathic presentation. (19% of cases). Less intense brain injury. Does not have the swelling, the diffuse cerebral hypodensities, or clinical encephalopathic features. "These children have various combinations of subdural and retinal haemorrhages, rib fractures, and other skeletal fractureess, bruising, etc.

(d) Chronic extracerebral presentation. (22% of cases). (Delayed presentation). Small infants with isolated SDH which is often chronic with a rapidly expanding head circumference with vomiting and signs of increasing ICP: vomiting, irritability, failure to thrive, hypotonia, seizures but with little encephalopathy. The head injury was not intense enough to rupture the large bridging veins or the brain, only the smaller and weaker bridging veins. Any RH that may have originally been present have disappeared by the time of presentation. In this situation, child abuse can only be established by ancillary features such as fractures, along with identifiable risk factors. The exact mechanism of injury may not be established.

In summary, they postulate a spectrum of intensity in inflicted infant head injury. "Infants can be traumatically injured in many ways and many instances are unwitnessed. Thus the generic term non-accidental head injury or inflicted traumatic brain injury should be used in preference to shaken baby syndrome, which implies a specific mechanism of injury. After the history, examination, and investigations have been considered the following concussions about the cause of brain injury can be reached:

- (1) characteristic of non-accidental trauma
- (2) consistent with non-accidental trauma
- (3) possibly due to non-accidental trauma
- (4) not due to non-accidental trauma.

Animal models of shaken baby syndrome: revisiting the pathophysiology of this devastating injury. Bonnier C, Mesple B, Gressens P. (Brussels) Pediatric Rehabilitation 2004 Jul-Sep; 7(3): 165-171. Shook mice. 75 % of survivors had focal brain lesions consisting of hemorrhagic or cystic lesions of the white matter, corpus callosum and cerebellum. All showed late white matter atrophy.

Knight's Forensic Pathology, Third Edition. Saukko P and Knight B. New York: Oxford University Press, 2004. Chapter 22, "Fatal Child Abuse." Discussion of the shaken controversy. Please see below under "Unclassifiable."

The 'unified hypothesis' of Geddes et al. is not supported by the data. Punt J, Bonshek RE, Jaspan T, McConachie NS, Punt N, Ratcliffe JM. Pediatric Rehabilitation 2004; 7: 173-184. (UK)

The first argument the authors make is to contradict a particular statement that is made in Geddes II, the statement that “Nobody really knows how babies are injured.” This statement, say Punt et al., “is erroneous.” Against this statement Punt et al. adduce the following four arguments [I have rearranged their presentation somewhat for clarity --JKR]: (1) The negative evidence that trivial head injuries are common in infants and almost always have a benign outcome. (2) Major accidental head trauma in this age group demonstrates that major forces must have appertained and the causes and mechanisms are readily apparent. (3) Confessions, which acknowledge severe force, and the fact that the clinical findings in unconfessed cases match those in the confessed cases, tending to confirm the accuracy of the confessions. (4) Geddes’ own data in Geddes II show that in every one of their 37 fatal inflicted head injury cases the trauma was significant and not trivial (along with numerous shaking and slamming confessions the article includes three drops and one fall. The three drops had rib fractures, skull fractures, leg fractures.); they present not a single case in which fatal injury happened from trivial or mild trauma. Geddes et al. have admitted this in open conferences. Punt goes on to use Geddes’ own data to point out that 15/37 babies had multiple fractures or abdominal injuries, 16/37 had skull fractures, and 27/37 had subscalp bruising.

Next the authors attack another specific statement made in Geddes II (actually a continuation of the same sentence as the above). This is the statement that “It may not be necessary to shake an infant very violently to produce stretch injury to its neuraxis.” Punt et al. say that this statement is based on Geddes’ finding in their 53 infants that very few had a demonstrable pattern of TAI. Punt points out that Geddes et al. themselves admit that diagnosing TAI in infants under 1 year is difficult (“was not always easy.”) Indeed, Punt et al. (who as Geddes will later argue are not neuropathologists) expand this point by noting that in later court testimony highly experienced neuropathologists including Geddes et al. themselves have proven themselves unable to distinguish between hypoxic and traumatic DAI. AND: since this distinction has proven to be so difficult, it becomes suspect that Geddes II not only purports to diagnose this distinction accurately in all but one case, but did so without any blind comparison of slides or outside review of the findings. In other words, on this highly subjective and difficult diagnostic point, Geddes et al ask us to take their word for it that they could distinguish TAI and that it was ruled out.

Next Punt et al attack the unified hypothesis. (a) Looking back at Geddes II, they note that of the 13 babies with neck injury, at most 85% had apnea. Not only that, but in the 24 infants who did NOT have neck injury, 83% had apnea -- essentially the same percentage as the neck-injured infants. So Geddes II fails to demonstrate that neck injury has anything to do with causing apnea. (b) Going to the “dural haemorrhage” article (2003, above), here are the problems with it: (1) Of its 50 cases, only one (the index case) had any grossly detectable subdural hemorrhage. (2) Only one case had any brain swelling, negating the authors’ hypothesis that SDH was caused by brain swelling. (3) Their purported results contradict those of Towbin in 600 babies (1970), who found that subdural, brainstem, and spinal cord hemorrhage was always caused by trauma, while subarachnoid and intraventricular hemorrhage were caused by hypoxia, and this is supported by overwhelming clinical, radiological, and scholarly experience. (4) If you look closely at the three cases they report of intradural hemorrhage due to hypoxia (their cases 24, 25, and 28), you find that all three of them were subjected to extreme violence, AND none of them had a neck injury. (5) Of their SDH cases, only 1/3 had evidence of increased ICP; so how can they conclude from this that increased ICP causes SDH? (6) The paper presents no data concerning RH, so how can it conclude that RH were caused by ICP alone. (7) AHT RH are rarely accompanied by any disc swelling. (8) Geddes uses increased brain weight as the only criterion for determining the presence of increased ICP, but this is a shaky method. (9) Their suggestion that systemic hypertension might cause or contribute to the SDH is not correct, because there is no systemic hypertension. (10) Neurosurgical experience shows that ruptured or avulsed bridging veins are commonly encountered at craniotomy in these patients. (11) Tasker has published extensive work on increased ICP findings in nontraumatic coma, and nowhere does he refer to any SDH. And note Rao et al., 1999, who studied 73 babies with AHT or nontraumatic cerebral edema, and found that all of the AHT’s had SDH and none of the nontraumatics had SDH. (12) If the unified hypothesis works, the authors should have found some infants who had the appropriate combination of findings to fit into the unified hypothesis, namely neck injury + raised ICP + thin SDH + RH + no ev of trauma. But they present exactly zero babies who have this complex of findings.

Now let’s go on to read Tim David’s review in the Autumn 2004 issue of the *Quarterly*. He says these authors argue that Geddes’ own data tend to refute her conclusion that pure anoxia due to cervical spine injury can cause the brain findings in SBS, and that trivial hyperextension can cause this cervical spine injury. Argues that 15 out of Geddes’ 37 babies had multiple fractures or abdominal injuries, refuting the

idea that they could have suffered their findings from trivial or accidental trauma. Only 8 out of 37 had no evidence of trauma. As to whether pure ICP could cause SDH and RH, , point out that cases of nontraumatic encephalopathy do not have SDH or RH. Punt et al. make the following attacks on Geddes:

1. Their method of distinguishing anoxic axonal injury from TAI is not reproducible.
2. Many (15/37) of the babies that had cervical spine injury in fact had autopsy evidence of major trauma, namely broken bones.
3. Most of the cases where Geddes asserts that the SDH and RH were caused by anoxic brain swelling did not have increased brain weight.
4. "Microscopic" subdural hemorrhage. Only one baby had gross SDH. Microscopic subdural hemorrhage is diagnostically insignificant.*
5. The authors did not study the eyes in any of their cases.

Geddes et al. were invited by the journal to reply to Punt's critique. *Pediatric Rehabilitation* 2004; 7: 261-265. Their reply is reviewed by Betty in the Spring 2005 issue of the *Quarterly*, and is also summarized herein next below.

* This same point is discussed by me in review of Geddes' article "Inflicted head injury in infants," *FSI* 2004; 146: 83-88, in the Spring 2005 issue of *Quarterly*. See above under "Shaken." See also Punt's later (2006) e-letter to BMJ at <http://adc.bmj.com/cgi/eletters/91/3/205>.

Invited Response: Violence is not necessary to produce subdural and retinal haemorrhage: a reply to Punt et al. *Pediatric Rehabilitation* 2004; 7: 261-265.

Shaken baby syndrome without intracranial hemorrhage on initial computed tomography. Morad Y, Avni I, Capra L, Case ME, Feldman K, Kodosi SR, Esernio-Jenssen D, Lukefahr JL, Levin AV. *Journal of AAPOS* 2004 Dec; 8(6): 521-527. (American Academy for Pediatric Ophthalmology and Strabismus). Working off of SIGCA-MD-L, they found eight (8) cases of SBS from around the world in which the initial CT scan failed to demonstrate any intracranial hemorrhage. Age range from 2 to 27 months. 5 died and were autopsied. 3 survived with neurological or visual deficits. The authors defined these cases as SBS based on the following case definition:*

the presence of at least two of the following criteria:

- (1) altered state of consciousness with or without seizure and an abnormal CT scan showing characteristic [hemorrhagic or nonhemorrhagic] findings of SBS
- (2) typical skeletal injury
- (3) retinal hemorrhage with or without macular schisis
- (4) history of child abuse that included shaking with or without blunt head trauma or an inadequate history to explain the observed injuries

All the babies had cerebral edema on initial CT. Autopsies showed SAH and SDH in cases 1 and 3. The other autopsied cases: case 4 showed hern with necrosis of the cerebellum and upper spinal cord. Case 7 showed HIE. Case 8 showed edema. Interestingly, no brain showed DAI or documented shearing injury by autopsy. Why did they not have SDH or SAH on initial CT? The authors posit the compression ("collapse") of the subdural space due to rapid onset of cerebral edema, with tamponade of the broken bridging veins. They attribute this same hypothesis to Maxeiner, who in one traffic case demonstrated multiple ruptures of the bridging veins without any hemorrhage, and attributed this to tamponade by cerebral edema. (Maxeiner, 1999). Or else inaccuracy of the CT compared to MRI. They review the literature on the frequency of SDH in SBS and on the accuracy of CT in SBS. They explicitly attribute the cerebral edema to "shearing injury" of the cerebrum.

I will now summarize their eight cases:

- Case 1 4 months. Father said the baby “just started shaking.” CT showed cerebral edema only; MRI showed a small SDH. OD showed commotio retinae limited to the posterior pole. OS showed diffuse commotio retinae plus a subhyaloid hemorrhage next to the optic nerve. No other injuries. Autopsy showed SAH. Father confessed to shaking. Lived five days on ventilator.

- Case 2 8 months. Baby sitter said she choked on object. CT showed cerebral edema with infarcts. Fundi showed multiple intraretinal hemorrhages in the periphery. Survived in PVS. No other injuries.

- Case 3 27 months. No history of trauma. Multiple bruises. CT showed HIE with edema progressing to hern. Fundi showed multilayer hemorrhages. Autopsy showed apparently scalp contusions, and a large convexity SDH, and ONSH.

- Case 4 3 months. Found unresponsive. CT showed extreme cerebral edema. Fundi showed retinoschisis. Autopsy negative for intracranial hemorrhage, positive for herniation. With necrosis of the cerebellum and upper spinal cord. Retinas showed schisis cavities.

- Case 5 1 year old. No history of trauma, found in status epilepticus. CT showed mild cerebral edema. Fundi showed multiple severe intraretinal and preretinal hemorrhages extending to the periphery, plus a perimacular fold OD. MRI showed a thin layer of blood either subarachnoid or subdural on day 5. Survived with deficits.

- Case 6 14 months. No history of trauma. CT showed a depressed right occipital skull fracture with no hemorrhage. Fundi showed multiple retinal hemorrhages extending to the periphery. Skeletal survey showed a healing fracture of the ulna and tibia; bone scan was positive for other fractures. Exam showed multiple bruises. Survived with severe neurological and visual deficits.

- Case 7 2 months. Found unresponsive by boyfriend. CT showed cerebral edema. Skeletal survey showed a posterior rib fracture and a tibial metaphyseal fracture. Fundi showed five retinal hemorrhages in the periphery of the left eye. Expired after three weeks. Autopsy showed HIE, no intracranial hemorrhage. Boyfriend confessed to shaking.

- Case 8 6 months. History of being shaken and thrown by his aunt. CT showed cerebral edema. Had a left humerus fracture and bruises of the face. Autopsy showed cerebral edema. Eye pathology showed RH.

Cases 6, 7, and 8 had skeletal fractures (37%). Cases 3, 6, and 8 had bruises. Only cases 3, 6, and 8 had any evidence of blunt force trauma to the head. Cases 1, 2, 4, and 5 had no trauma anywhere. Cases 1 and 8 had confessions (shaking in case 1, shaking and throwing in case 8.)

Their point is that you do not have to have intracranial hemorrhage in order to diagnose SBS. They cite the reported frequencies of SDH and SAH

93% and 16%	(Morad et al., 2002)
92%	(Kivlin et al., 2000)

The authors state, “SBS usually is characterized in part by intracranial hemorrhage. At presentation subdural hemorrhage is seen in 10-80% and subarachnoid hemorrhage in 10-72% of victims.” (citing Levin’s earlier review of retinal hemorrhages in volume 18 (2000)). They note previously published case reports of SBS cases without intracranial hemorrhage seen on imaging. Frank et al., 1985, Elner et al., 1990, Krugman et al., 2000, Green et al., 1996, Maxeiner, 2002. A total of 8 previously published cases. So now there is a total of 16 published cases. It appears that the point of the publication is to make

emergency physicians aware that SBS is still in the differential diagnosis even if there is no intracranial hemorrhage. It also supports the concept of Mary Case and the NAME Position Paper that the SDH is merely an index of the severity of the underlying brain injury (shearing injury) which is the basic lesion in AHT. Note that all these cases had at least moderately extensive RH.

[Comment by JKR: This conclusion is the exact opposite of the postulate of Geddes et al. (2004), who postulate that you can get SDH due to brain swelling with no trauma. These guys postulate that you can get *no* SDH due to brain swelling *with* trauma. Obviously we cannot have it both ways; either swelling causes SDH or it prevents SDH. But this “swelling” argument could explain why the SDH in most AHT cases is so thin.]

* Note that this definition of SBS has moved definitively away from Caffey’s original definition which required *both* fractures *and* SDH for the diagnosis. Cf. Lazoritz et al., The whiplash shaken infant syndrome: has Caffey's syndrome changed or have we changed his syndrome? (1997), where they mention the rarity of long bone fractures in present-day cases but dwell more on the evidence of skull impact which is common today and was unknown to Caffey.

The report of the Cannings Review Team, 2004. The Attorney General’s report to the House of Lords. The Solicitor General’s report to the House of Commons. Regarding Cannings-type cases (SIDS cases and SBS cases) From the report: Introduction.

“1. On 19th January this year (2004), on the day that the Court of Appeal published its judgment allowing the appeal of Angela Cannings against conviction, I [the Attorney General of the Realm] announced that I had established a review of all cases of convictions in the last 10 years of a parent for the unlawful killing of babies and infants under 2. At the same time I asked the Crown Prosecution Service to review the current cases involving an unexplained infant death.

[Discusses the potential unsafety of criminal convictions in SIDS cases, as to which see above under “SIDS VERSUS SUFFOCATION.”]

Goes on to discuss SBS cases:

42. “Shaken baby syndrome” cases. 97 cases. 9 were identified as Category 3 cases.

43. “The term ‘shaken baby syndrome’ has become very widely used throughout the world to describe a category of non-accidental injury believed to result from severe shaking of a baby or small infant, associated for some commentators with impact of the head. This term is open to objection; it is not so much a medical diagnosis as an explanation for an injury. There is not agreement on exactly what injury patterns do or do not fall into this category. There is a particular debate over the issue whether the injuries can be the result of shaking alone or whether shaking with impact is involved (although the view can be held that this may be a sterile debate as all shaking is likely to involve some impact 9chin on chest wall or back of head on spine)). As a result, other terms are also in use, including shaken impact syndrome whiplash shaking syndrome, and abusive head trauma.

44. However, the term “shaken baby syndrome” is one that has become familiar not only to health care and child protection professionals but also to the wider public. I will, therefore, use that term in this report for convenience...

45. SBS cases differ from SIDS cases...

46. [But there is the similarity that they are controversial among experts... I have been much assisted by Professor Tim David...]

47. Summarizes the orthodox view of SBS

48. Gives the Geddes & Whitwell argument as “another school of thought.” To wit, that “[T]he symptoms which have been taken as indicative of criminal behavior can in fact be caused by less trauma than had previously been thought, or more recently, by no trauma at all. A central feature of the hypothesis is the proposition that the subdural haematoma typical of the SBS diagnosis may be caused by lack of oxygen, and not just physical trauma, and that the lack of oxygen can itself be caused by less severe trauma to the brain stem, or, as I have noted, more recently, no trauma at all. (citing Geddes et al. Dural haemorrhages in non-traumatic infant deaths, 2003). The basis on which this is proposed is hotly disputed. For example, in a recent article by Punt et al., the authors mount a vigorous and detailed attack on the methodology, the logic, and the evidential base for the Geddes approach. Geddes responded with a robust defence in which the conclusion is maintained that a lack of oxygen can cause a subdural haemorrhage. I do not think it

unfair to say that the Geddes theory has not been proven. [See also the article review by JKR in the Spring 2005 issue of the *Child Abuse Quarterly*.]

49. “The [legal] issue which arises is whether this material, in the form of the views of Dr Geddes and others, casts doubt on convictions in SBS cases particularly where the verdict was obtained prior to the publication of Dr Geddes’ work in 2001.”

50. Reviews recent lower-court cases involving expert testimony on SBS degree of force.

51. It is clear that this difficult area will receive continued careful thought by the medical and scientific community in the future...

52 - 53. The Court of Appeal has granted leave to appeal several SBS cases, fatal and nonfatal. These will not be heard before summer 2005 at the earliest. So I am going to report the issue now.

54. This work will have to be provisional.

Now, as to the report of the CRT team, it is attached as Annex C.

The neurological outcome of non-accidental head injury. Barlow K, Thompson E, Johnson D, Minns RA. (Edinburgh) *Pediatric Rehabilitation* 2004; 7: 195-203. From Dr Reece’s review in the Winter 05 *Quarterly*. Followup of 25 AHT children for five years.

hemiparesis	4 patients
tetraplegia	2
ataxia	8
brainstem	5
oculomotor palsy	1
pseudobulbar palsy	1
other...	
cortical blindness	4
extraocular abn	7

Global outcome score

normal	8
mild deficits in mobility	2
moderate deficits	3
severe deficits	6
profound	4

Found that 36% required long-term nursing support. This paper is cited by Hymel et al., 2007 for the proposition that “Many survivors of inflicted head trauma during infancy later manifest developmental delays,, sensory deficits, feeding difficulties, recurrent seizures, intellectual deficits, motor impairments, attention deficits, and/or educational and behaviroal dysfunctions.”

Update on child maltreatment with a special focus on shaken baby syndrome. Newton AW, Vandeven AM. (Harvard) *Curr Opin Pediatr* 2005 Apr; 17(2): 246-251. (See also Update on childhood sexual abuse, in the same issue.) From the abstract: “There remain controversies in the diagnosis of shaken baby syndrome but several carefully done case series are clarifying our knowledge of the myriad presntations of this form of non-accidental head trauma.” As far as treatment and long-term sequelae the effectiveness of home visitation is coming into question.

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. In this chapter, Dr Starling uses the term “shaking” as her term for abusive head trauma. She summarizes the shaking debate as follows [with some emendations by me --JKR]:

Critics of Duhaime’s proposal that impact is necessary make the following arguments:

1. Duhaime’s use of primate data did not take account of immaturity features of the infant.

2. Duhaime's model only measured peak acceleration.
3. Duhaime's model did not address repetitive shaking.
4. "Models are only beginning to capture the characteristics of the immature brain and skull [to say nothing of neck; see Zimmerman's discussion in *Inflicted Childhood Neurotrauma* symposium, 2003, above under "Cervical spine and neck," discussing the papers of Hadley et al. and Geddes et al., who found trauma of the cervicomedullary junction. --JKR], and none have addressed the differences between a single blow or whiplash and multiple shakes, nor do models address the role of secondary injury in the neurological outcome." p. 41
5. Six of Duhaime's 13 infant autopsies did not show evidence of impact, and others, for example Gilliland, have shown cases with no impact injuries. Citing Hadley et al., 1989, Alexander, Sato et al., 1990, Gilliland & Folberg, 1996, and Pounder's shaken adult, 1997.
6. Dr Starling's point about secondary injury and its role in the neurological outcome deserves to be enumerated separately. Severe secondary injury seems to be much more a feature of abusive head trauma than of accidental head and neck trauma. Therefore this by itself becomes a diagnostic feature of AHT. (Obviously, severe secondary injury may be caused by delayed rescue, as in the case example given on p. 42 by Dr Starling). But more than that: severe secondary injury is a feature of shaking, that is, cases with a shaking history and cases without evidence of impact. -- JKR
7. The amount of force involved in shaking is not quantified. Starling quotes the AAP statement, 2001, that it is "so violent that individuals observing it would recognize that it is dangerous..."

Dr Starling gives a case example of a 3 month old male infant left in his father's care who presented by private auto with seizure activity and no history of trauma. The father called his wife at work and had her come home when the baby went into distress while watching TV, did not call 911. ER found respiratory distress and extreme lethargy, requiring immediate intubation. Bruising noted on left cheek and left ear, which the father attributed to baby sleeping with his cheek on his bottle. Bruises were not present when the mother left for work that morning. CT sh L hemisph and interh SDH, with effacement of the L lateral ventricle. Extensive bilateral RH with TR on L. Skeletal survey negative.

Goes on to discuss AHT in general, beginning with the clinical presentation. Shaking is thought to be either an anger response or a method of quieting the crying infant. If it works once, it may be used again. The presentation may be either catastrophic or subtle. In the subtle presentations, vomiting, irritability and sleepiness may be mistaken by physicians for viral illness. Therefore, the diagnosis is missed in 30% of cases (citing Carole Jenny et al., JAMA 1999). Mentions the utility of looking for xanthochromia in a spun sample of CSF, since bloody fluid may be mistaken for a traumatic tap. In the more overt cases, seizures are the presenting symptom in 30 – 70% of cases. In taking the history from the caregiver, it is important to elicit enough detail to generate a timeline. "Red flags for abuse include histories that vary over time or between caregivers, reported child behavior that may not be developmentally appropriate, or described events that do not account for the injuries seen on presentation." (p. 42) The correct diagnosis is often delayed by the absence of external injury. [emphasis added --JKR]

Discusses RH. 70 – 90% of cases. Unilateral in 14 – 20%. RH can be categorized as preretinal, intraretinal, or subretinal. Subretinal hems are distinguished by seeing the retinal vessels coursing over them. Preretinal hems are distinguished by seeing that they cover up the retinal vessels. Preretinal hems are also called subhyaloid hems. Intraretinal hems are described by appearance as flame, dot, or blot. As to the mechanism of RH, says that there are several theories. "The most likely explanations are the orbital and vitreous shaking theories."

- a. The orbital shaking theory proposes that the eye shakes forward and backward within the orbit, in which it is only loosely fixed, and this stretches the optic nerve, causing ONSH. This ONSH in turn causes RH. "It is unclear how this intradural hemorrhage translates to retinal hemorrhage, but it may be the

result of autonomic dysregulation or central vessel damage impairing blood return from the eyeball.” (citing Levin in *Recent Advances No. 18*, 2000).

b. The vitreous traction theory, which Dr Starling describes as more likely.

Discusses the differential diagnosis of RH. Birth hemorrhages affect up to 59% of normal newborns, with increased incidences in prolonged labor and difficult delivery. Even the deepest birth hemorrhages disappear in 14 to 21 days. The further differential diagnosis of RH includes sepsis, leukemia, meningococcal meningitis, coagulopathy, and vasculitis. But those hems are minor and generally limited to the posterior retina. Seizures do not cause RH. (citing Sandramouli et al., 1997 and Tyagi et al., 1998). Accidental trauma is in the differential because 2% of serious accidental head injury produces RH. Citing DiScala et al. (2000) who found RH in 0.6% of accident victims and 22.8% of abuse. As to CPR, there are a few case reports of CPR causing small posterior RH. As to Terson’s syndrome, says “Terson syndrome in children is very rare; when seen it usually is associated with shaking injuries.” (citing Levin, 2000). As to Purtscher’s syndrome, it is retinal exudates or white patches after severe compressive chest trauma; it is also very rare in children. Cites the case report of McEniery et al. (1991) on a child with inflicted chest compression who had no RH.

Associated injuries. The absence of external signs of abuse was the hallmark of SBS, noting that this was emphasized by Caffey in his original report. But nowadays, rib fractures, skull fractures, and bruising are found in 40 – 80% of cases. Careful attention must be paid to the skin of the face and head for bruises and abrasions. Also grab marks of the chest or any extremity. Also spanking. In Merten’s series, 70% of AHT children had skull fractures. 51% in Feldman’s series (compared to only 1% of children with accidental SDH). “The absence of associated injuries in AHT may make the syndrome more difficult to diagnose.” Carole Jenny finding that 31% of AHT infants had been missed during the initial visit.

Discusses imaging.

Intracranial injury. As to SDH, says that perhaps the most specific site for AHT is interhemispheric, citing Rao et al, 1999, and Zimmerman et al., 1979. Points out that AHT SDH is rarely surgical, but is a marker for the underlying brain injury. As to DAI, says that infant axons are more vulnerable to DAI because of their small size and lack of myelin. Severe DAI results in immediate unconsciousness. Less severe DAI results in immediate decrease in the level of consciousness. Gives an example of shearing injury seen on CT, showing up as a well-defined black gap in the frontal pole, and described as a cleftlike tear in the gray-white junction. It is also known as a gliding contusion. Shearing injury may be initially undetectable on imaging, because it occurs at the cellular level. Sometimes DAI is undetectable by pathology, either because of short survival or just being undetectable [often due to the autolytic condition of the brain status post prolonged ventilator therapy --JKR]. “The absence of pathological evidence of DAI is not proof that it did not occur.” (p. 47). In other words, the sensitivity of pathology is rather low for this lesion. (citing Alexander et al.’s chapter on AHT in Reece’s textbook). Mentions the directionality of DAI: lateral acceleration is much more likely to cause it. If there is DAI great enough to be visible on imaging studies, there was immediate loss of consciousness, with no lucid interval.

Secondary injuries. Secondary injury is the death of cells that were not injured during the original event. Examples include increased ICP, coagulopathy, and cerebral infarction. Cerebral infarction can be caused by three things:

shock
apnea
ICP

Traumatic apnea is caused by TAI of the medullary respiratory center.

Cerebral edema is often seen on the first CT scan, as early as 1 to 3 hours after injury. (Citing Mark Dias et al., 1998 and Wilman, Bank, time of injury, 1997). The “reversal sign” is severe edema causing the deep cerebral nuclei and cerebellum to appear whiter on CT than do the cerebral hemispheres. (p. 47)

Cervical injury. It has been reported (citing Hadley, Sonntag, 1989), but Ken Feldman found that it was rare and not worth looking for.

Time of injury. 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)

Perpetrators. Males are over 50%, usually fathers or boyfriends. Female babysitters are 14%. Nonfamilial caregivers rarely confess.

Differential diagnosis. There is no illness or entity that mimics all the findings of SBS, particularly the intracranial hemorrhage and extensive RH.

- automobile accident
- falls
- bleeding disorders (as to which, see F XIII deficiency art next below)
- metabolic disorders
- birth injury
- rare causes
 - ruptured subarachnoid cyst
 - venous sinus thrombosis
 - ruptured AVM
 - tumors
- unique theories of causation
 - rebleed
 - second impact
 - TADD

As to motor vehicle accidents and falls, cites DiScala's study and Reece's study showing that SDH was seen in only 10% of accident victims versus 50% of abused. "Although case reports of significant household accidents have been shown to cause posterior retinal hemorrhages (citing Cindy Christian et al., household falls, 1999), minor falls and household accidents do not cause the extensive retinal hemorrhages and diffuse brain injury seen in inflicted head trauma." (p. 50) As to falls, she gives a full-page table summarizing the literature on falls versus abuse (p. 51), with emphasis on those studying reliably observed falls. Conclusion: "[S]imple household falls as an explanation for serious head injury should be considered inadequate." As to bleeding disorders, discusses platelet dysfunctions such as VWD and TTP, which cause superficial bleeding into soft tissue, hemophilia which rarely causes ICH, HDN occurs in

1/200 to 1/400 infants; the diagnostic test is that the bleeding stops when vitamin K is given. In no case does a bleeding disorder explain the combination of RH and brain injury. Discusses traumatic coagulopathy commonly seen in children hospitalized with severe TBI of any cause. As to metabolic disorders, GA-1, it often has macrocephaly at birth. Then it has sudden onset usually at age 6-18 months with hypotonia, loss of head control, seizures, and dystonia. This is followed by progressive encephalopathy and developmental delay with a characteristic CT picture including frontotemporal atrophy. Levin found only eight cases in the world having RH, and seven of these were a single dot or blot. As to birth injury, it can cause stroke and ICH, but onset of symptoms usually is immediate at the time of birth. As to other rare causes of ICH, (see above list), Dr Starling states that "[T]hese entities are not associated with the type of RH seen in SBS." (p. 52) As to "unique theories of causation," you have the rebleed theory, a courtroom theory. Infant rebleeds are not symptomatic. Plus, repetitive accident is unlikely. Ewing-Cobbs et al., 1998 found that 9/20 abused children had previous SDH, while none of 20 accident children had any. Hymel et al. found that large nonacute collections were always the result of abuse, not occult accidents. As to the second impact syndrome, young athletes suffer a witnessed and documented head injury, remain active, and then collapse. How likely is this sequence in an infant, noting that the initial injury has to be witnessed and documented. As to TADD, these were focal contact injuries, almost all in older children. The focal primary injuries were later complicated by hypoxia or cerebral ischemia, leading to global effects. Contrast this with the diffuse injuries of shaken infants.

Case report: unusual presentation of factor XIII deficiency. Almeida A, Khair K, Hann I, Liesner R. (Great Ormond Street). Haemophilia 2005; 8: 703-705. Factor XIII stabilizes clots by crosslinking fibrin. Without it clots are easily dissolved. Deficiency causes severe bleeding tendency (hemophilia). There have been 200 known cases in the world. Autosomal recessive. The usual presentation is prolonged umbilical stump bleeding or prolonged bleeding at circumcision, plus easy bruisability and poor wound healing. Intracranial hemorrhage is seen in 30% of cases. Patients will have a normal PT and PTT and normal platelet counts. The authors present a two year old daughter of consanguineous parents who presented with drowsiness and irritability. Head CT showed an intracerebral hemorrhage. Normal clotting times and CBC. Operated, with a complicated postoperative course. Developed DVT which propagated into the IVC and right atrium. Started on heparin. After suffering a rebleed which was unexplained, a full hematologic study was undertaken, and it revealed a F XIII level of 3% of normal. A study of the patient's little brother showed a F XIII level of 5%, and he had a history of prolonged umbilical stump bleeding. The index case had a poor neurological outcome. No RH are mentioned. Treated with F XIII infusions, with no further bleedings.

R. v. Harris, [2005] EWCA Crim. 1980 (Supreme Court of Judicature, Court of Appeal (Criminal Division), before Gage, LJ, Gross, J, McFarlane, J. Evaluating the effect of Geddes' unified hypothesis on criminal prosecutions for SBS in light of *R. v. Cannings*. Found: "In our judgement, it follows that the unified hypothesis can no longer be regarded as a credible or alternative cause of the triad of injuries [in SBS]." Why? 1. Geddes herself emphasizes that it is only a hypothesis, not a fact. 2. A host of other experts have criticized it and it is very controversial. On the other hand, the court does not find the diagnostic specificity of the triad to be established as a medical certainty. All the evidence must be taken into account in each case. The courts will not attempt to resolve a scientific controversy. So it will be left to the jury to evaluate the expert opinions and circumstantial and testimonial evidence in each case. Comment by JKR: The Crown appears to have won this case, because under this ruling, as a matter of law, the defense will no longer be permitted to argue to juries that a non-traumatic etiology (the Geddes hypothesis) could account for the findings in a case where the "triad" is present AND competent experts have testified that the injuries were caused by abuse. The defense will have to argue some other etiology, such as fall, bleeding disorder, or something. So the defense has had a courtroom weapon stricken from their hands in the United Kingdom. The opinion also includes a canvassing of the issue of biomechanics and short falls in shaken. Plunkett testified, as did Thibault and a British bioengineer named Dr Bertocci. The court concludes that biomechanics is too uncertain at this stage to overcome "primary facts." "Dr Thibault's views are altogether too difficult to reconcile with evidence of primary fact in this case, for the conviction to be regarded as unsafe by reason of the biomechanical evidence." The court rules that "the notion of an accidental fall in this case...is simply fanciful." The court gives an extended discussion of all these issues, and resolves all of them in favor of the Crown.

Neuroradiological aspects of subdural haemorrhages. S Datta, N Stoodley, S Jayawant, S Renowden, A Kemp. Arch Dis Chil (UK) 2005; 90: 947-951. Retrospective review of the films from 74 children under 2 with SDH or subdural effusion. Review by two blinded radiologists. Found that MRI is necessary in most cases bc CT misses small SDH's.

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) down to 2001* that contain case reports where babies were documented to have been shaken, as opposed to some other history. Comes up with 41 cases exclusive of the 13 cases of Hadley, Sonntag, Rekate (1989); in Hadley's cases it is not clear where the shaking information came from, whether it was from the history or just inferred by the clinicians, so those cases are treated separately. Of the 41 historically shaken cases, there were 11 pure shakings, 12 shaken-plus-impact, and 18 in which impact could not be determined from the information provided. Here I will give the text of the review I contributed to the *Quarterly*:

Case analysis of brain-injured admittedly shaken infants: 54 cases, 1969-2001. Jan E. Leestma, MD. American Journal of Forensic Medicine and Pathology 2005; 26: 199-212.

This is a literature review. The author has extracted the individual case details from 23 articles in the published literature on SBS in order to isolate the clinical characteristics of babies who were documented to have been shaken. (That is, either the shaking was eyewitnessed or one of the caregivers admitted to the clinicians that he had shaken the patient.) The articles studied begin with that of Guthkelch (1971) and go through those of Mushin & Morgan (1971), Caffey (1974), Oliver (1975), Ellison et al. (1978), Bennet & French (1980), Carter & McCormick (1983), Benstead (1983), Frank et al. (1985), Eagan et al. (1985), Greenwald et al. (1986), Lambert et al. (1986), Alexander et al. (1986), Duhaime et al. (1987), Hadley, Sonntag et al. (1989), Benzel & Hadden (1989), Kleinman et al. (1989) Spaide et al. (1990), Massicotte et al. (1991), Zepp et al. (1992), Haseler et al. (1997), and Becker et al. (1998). The author tabulates the clinical details of these patients case by case with respect to age, sex, state of consciousness on admission, state of respiration on admission, vomiting, seizures, retinal hemorrhage, vitreous hemorrhage, retinal folds, retinal detachment, papilledema, and acute subdural hematoma -- details that are considered significant for forensic diagnosis. He also records other significant details of each case such as body bruises, fractures, chronic subdural hematomas, and past history of abuse. The purpose of this is to see what the literature tells us, not about what clinicians have inferred about the clinical effects of shaking, but what the shaken patients actually had. The author gives attention both to the clinical findings (such as retinal hemorrhage, bruises, etc.) and to the time-course of symptom development (relating to the instant-unconsciousness doctrine.)

The author finds in this literature forty-one (41) patients who had a documented history of being shaken. Of these, eleven (11) patients were pure shaking cases, with no evidence of head impact. Twelve (12) had a history of shaking but also had evidence of impact. That leaves eighteen (18) shaken patients in whom there is not enough published information to determine whether there was evidence of impact or not. Then there were thirteen (13) additional cases contained in the article of Hadley, Sonntag et al. (1989); these patients are stated by Hadley et al. to have been shaken, but Hadley's article does not tell where this information came from -- whether it was received from the history or inferred by the clinicians. This group of cases the present author separates out for separate consideration because of the vagueness of the source of information as to shaking; the author states, as to Hadley's cases, that "No specific incident data regarding shaking were provided, and there is doubt if shaking occurred."

Concerning the eleven (11) pure-shaking patients, the author finds that as a group they had the following characteristics: Their average age was 5.1 months (as opposed to 9.3 months for the shaken-with-impact patients). They were about equally male and female. Only three (3) of them were autopsied. As to the clinical details, what was reported broke out this way:

	Yes	No	Not reported
Conscious on admission	2	9	0
Normal respiration	4	5	0

Seizing	5	2	4
Immediate onset of symptoms	0	4	7
Acute subdural	6	4	1
Chronic subdural	1	3	7
Subarachnoid hemorrhage	6	2	3
Parenchymal hemorrhage	2	5	4
Spinal cord injury	0	0	11
Spinal EDH or SDH	0	0	11
Retinal hemorrhage	9	0	2
Vitreous hemorrhage	3	0	8
Retinal folds	0	0	11
Fresh rib fractures	0	10	1
Old rib fractures	1	7	3
Facial injuries	0	11	0
Body injuries	3	8	0
Extremity injuries	2	8	1
Past history of abuse	2	2	7
Fatal outcome	3	8	0
Survived with deficits	4	7	0
Survived intact	4	7	0

It is to be noted from a study of the author's tabular presentation of the clinical details from all these 54 cases that there are question marks ("??") under many or most of the data items for most of the cases. In other words, the clinical details we consider diagnostically significant were not included in the published case material for most of the shaken babies.

The grouping of 12 shaken-plus-impact patients had certain differences: they were older (9.3 months), more often male, more often apneic on presentation (66% versus 25% for the pure shakers), and had worse outcomes (50% mortality versus 27% for the pure shakers). The level of consciousness on admission was comparable (73% obtunded or comatose versus 80% of the pure shakers). The historical onset of symptoms was more rapid (50% immediate versus 0% immediate for the pure shakers). The only cases of retinal folds (2) were reported in the shaken-plus-impact grouping; these were cases of Massicotte (1992) and Kivlin (1999). (Greenwald's macular cysts or domelike lesions with their residual "circular cicatrix" are not taken to represent retinal folds for purposes of this article.) Two special findings by the author stand out from his comparison of pure shakers versus shaken-plus-impacts: (1) None of the pure shakers had retinal folds. (2) None of the pure shakers had neck injuries.

The author draws the following principal conclusions:

1. "[C]ase-based information on allegedly 'shaken' infants is often scanty or missing."
2. The specifics of what happened are rarely elicited and almost never published.
3. There may be only eleven (11) cases in over 30 years of published case reports that meet the criterion of "shaken-only" infants.
4. "With respect to retinal pathology, the number of valid cases is simply too small to permit conclusions to be made regarding any form of retinal pathology and the phenomenon of shaking forces."
5. "It is obvious that delays in the appearance of symptoms do occur and are more likely than the immediate appearance of symptoms following the shaking incident(s)."
6. "Given the nature of the case report literature it is impossible to determine with scientific rigor what role shaking might play in abusive head injury in the face of obvious impact injuries. It is also not possible from this case analysis to infer that any particular form of intracranial or intraocular pathology is causally

related to shaking; rather, it appears that most of the pathologies in allegedly shaken babies are due to impact injuries to the head and body, regardless of what came before.”

7. It is unclear which injuries might have been caused by “accidental physical forces or by underlying or secondary disease processes.”

Comments by JKR: 1. Dr. Leestma’s study and his final conclusion are consistent with the respected opinions of DiMaio (DiMaio & DiMaio, *Forensic Pathology*, 1993) and Knight (Saukko & Knight, *Knight’s Forensic Pathology*, 3d ed., 2004) that so-called shaken babies are really slammed or thrown babies. The present article is a scholarly contribution to that body of learning. But unfortunately Dr. Leestma throws in a hooker -- conclusion #7 above. He claims that these 23 articles permit the conclusion that many of the babies might have suffered some or all of their injuries from innocent events or preexisting disease. This is violently opposed to what DiMaio, Knight, and all of the authors of the 23 articles conclude. These authors are unanimous in concluding that the injuries seen were caused by abuse. (Otherwise, why would they have published the articles?) Not only that, but the idea that the devastating and sudden clinical presentation of these conditions with vague and inadequate histories comports with an innocent etiology flies in the face of logic and the unanimous opinion of clinicians in the field. I therefore urge special attention to this particular conclusion of Dr. Leestma, which occurs, almost as an aside, in the last paragraph of his “Discussion” section. 2. The author’s #6 conclusion is deeply dishonest; it stands medicine on its head. Why? Because it is the nature of child abuse that the history given is usually inadequate or vague. Clinicians are forced to use other data to infer what really happened because an adequate history is not available. It is intellectually dishonest to turn this fact on its head and say that because the history is inadequate it is impossible to figure out what happened. That amounts to saying that all of clinical medicine is worthless.

* Note: Dr Reese’s editorial comment on this article (*Quarterly* for end 2005) points out that the author deliberately skipped the past five years of confession literature, which includes more confessions than all the previous years put together. Starling et al., 2004; Ricci et al., 2003; King et al., 2003; Asamura et al., 2003; Starling et al., 1995; Bechtel, Stoessel, et al., 2004; Keenan et al, 2003.

Shaken baby syndrome: theoretical and evidential controversies. Minns RA. Journal of the Royal College of Physicians of Edinburgh 2005; 35: 5-15. This well-written article reviews the published literature from medicine and biomechanics and basically concludes as follows:

1. Shaking can injure the infant brain.
2. Clinicians cannot diagnose the shaking mechanism, but they can diagnose NAHI.
 - a. The clinical diagnosis of NAHI, however, comes with varying degrees of certainty.
 - b. The term “shaken baby syndrome” is best avoided, because it implies diagnosis of a specific mechanism which cannot be clinically diagnosed.
3. Short falls can give a clinical picture similar to NAHI but this is rare.

I have moved the discussion of topic 1 to the “Biomechanics” section, below. --JKR

As to neuroimaging studies, radiology articles have included impact and nonimpact cases. In Barlow’s MRI work on 12 cases with and 7 cases without evidence of impact, all of the non-impact cases had some assortment of subtemporal, suboccipital, and interhemispheric SDH. They were able to demonstrate tearing of the surface veins in 29%, and other primary and secondary brain changes. He notes that there were no confessions of shaking in these cases; the absence of evidence of impact was a clinical deduction.

In summary, under the question of “Can shaking cause brain injury,” the author marshalls the following evidence in support of a “yes” conclusion:

1. Well documented individual cases with confession of violent shaking
2. Evidence from various models “that supports the ‘shaking alone’ mechanism
3. The severe and early brain atrophy that follows cases of shaking (which does not happen in accidental injury cases)

4. Rare adult cases
5. "There are rare cases which have been observed on 'nannycams.'" [This is the first I've heard of that. -JKR]

Author's conclusion: "The cumulative evidence is strongly supportive of the contention that adults do shake young infants, and that shaking alone may produce extensive brain injury."

As to topic 2, whether clinicians can diagnose the shaking mechanism, the answer is no, unless it was reliably witnessed, which is rare. Various features of the clinical picture of NAHI can be produced by a whole variety of mechanisms -- impact, compression, penetration, rotation. Therefore, the term "SBS" should not be used, because it implies that we have diagnosed a specific mechanism. (citing Lazoritz et al., have we changed...?, 1997). So what can we diagnose? We can diagnose NAHI without purporting to determine a specific mechanism. The diagnosis of NAHI rests on three criteria:

1. the history
2. a syndromic combination of clinical, radiological, and ophthalmological features
3. supportive social pathology

As to the history, contributing elements would include a changing history, no history of injury, inadequate history, and delayed rescue.

As to the clinical syndrome, the Scottish cases have broken out into four types of presentation:

1. The hyperacute encephalopathic presentation or cervicomedullary syndrome, usually fatal with CMJ injury and a big black brain due to anoxia. This is what Geddes described.
2. An acute encephalopathic presentation (the most common one), with bilateral SDH, seizures, apnea, hypotonia or decerebration, anemia, and extensive RH. This presentation may or may not have rib fractures.
3. Subacute presentation (non-encephalopathic) with no neurological deficits or imaging brain lesions, but with SDH and some combination of RH, fractures, and bruises.
4. Chronic extracerebral presentation. An expanding head, irritability, vomiting, FTT, and seizures from a chronic SDH.

As to supportive social pathology, the author mentions the risk factors for abuse and NAHI.

Now, the author gives a paragraph to "a very contentious scenario" -- that of SDH + RH with no other injuries and a history of a short fall. "In such circumstances and after thorough investigation, the clinician can only state that these injuries are consistent with an injury of non-accidental origin."

There is no single clinical finding that is pathognomonic for NAHI.

As to the diagnostic approach to possible NAHI, first the history. Then look for signs of malicious injury, which if present will clarify things greatly. If none, then exclude natural causes of SDH. Then, try to find evidence that will characterize the head injury as contact, non-contact, or a combination. After doing these things, the clinician is able to conclude that the brain injury is:

definitely due to a non-accidental cause
consistent with a non-accidental cause
possibly due to a non-accidental cause, or
not due to a non-accidental cause.

But he says that few of the published studies have followed this rigorous condition. Noting the work of Donahoe (evidence-based medicine..., 2003), and the absence of a prospective controlled trial. But the

lack of an evidence base does not negate the syndrome; it just means the support for the syndrome is incomplete.

Finally, as to short falls simulating NAHI, first he notes that short falls causing head injury in infants are quite common. “Undoubtedly fatal and serious injury can occur from low-height falls, but these are exceptionally rare.” In typical low-height falls, we find focal head bruising, extensive skull fractures, and focal brain contusion. We do not find encephalopathy or delayed rescue or a changing history or no history, etc. Discusses Plunkett’s paper.

Discusses RH, accepting the vitreous traction theory as “the most favoured explanation” for extensive RH in AHT.

Discusses the differential diagnosis of fractures: rickets, prematurity, leukemia, drug effects, inherited bone dysplasias, sickle cell anemia, scurvy, congenital indifference to pain, infantile cortical hyperostosis, copper deficiency, CMV, and OI.

But having said all that, he goes on to note that the obvious, that “RH, SDH’s, and multiple fractures individually may be due to multiple causes, but when in combination, they become a reliable pointer to non-accidental injury. Few differential diagnoses include these combinations, [although] birth injury and accidental trauma are the important aetiologies to exclude.”

Dr Reece listed this paper as one of the top ten papers of the past ten years at the 2006 National Shaken Baby Conference.

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.

Dr Duhaime in her note makes the following important point: “Impact and angular deceleration forces are not dichotomous. In the vast majority of clinical injuries they occur TOGETHER. In real-world injuries, as opposed to laboratory experiments in which forces can be controlled and separated, the impact is the event that typically CAUSES the angular deceleration to occur. The impact also increases the magnitude of the injury by shortening the time over which deceleration takes place. Thus, **trying to separate out whether an injury was a contact injury or a diffuse, angular deceleration injury is often, by definition, a futile exercise.** Although there are anatomic features that are clearly related to impact and others that have been shown experimentally to result from the isolated application of diffuse angular deceleration forces, in patients these types of forces typically coexist.” On this point, it is well to give attention to Steve Boos’s comment on another article (Bayly et al., *Deformation of the human brain...*, 2005), in which he says, “Impacts at the end of falls have, at times, been dismissed as unlikely causes of rotation, diffuse strains, and shearing. **Clearly such a dismissal is incorrect.** The crux of the issue is much more complex, involving how much strain, of what nature, in what proportion of the brain, following how much acceleration, sustained over how long an interval, is necessary for various types of injuries. For those of us who turn to biomechanics looking for answers, **the answers are unlikely to be simple and convenient.**”

Dr Duhaime makes another point. That the poor outcome is often related to secondary events, such as apnea and shock, rather than the biomechanical mechanism of the initiating injury.

Dr Hymel comments editorially on this article. (see next below) He says that “painstaking research” is still needed on the central question of whether pure shaking can cause brain injury. He summarizes the published work on both sides of that question. Dr Hymel emphasizes this article’s confirmation of the fact that diffuse bilateral brain injury leads to a dismal outcome, while focal contact injury is more favorable. He refers to the craniocervical junction studies, but does not explicitly connect them with the diffuse injury – dismal outcome data, although I think the implication is there. --JKR

Small steps in the right direction: the ongoing challenge of research regarding inflicted traumatic brain injury. (editorial) Hymel KP. Child Abuse & Neglect 2005; 29(9): 945-947. See above. This editorial summarizes the published research on whether shaking alone can cause severe brain injury. That breaks out into two areas: primary traumatic brain injury and secondary apneic-hypoxic brain injury. As to papers in favor of primary traumatic brain injury, Dr Hymel cites the following papers:

Saternus, Kernbach-Wighton, Oehmichen, 2000, describing autopsy evidence of intervertebral disc and shoulder muscle and brachial plexus injuries
 Raghupathi & Margulies, 2002, on shaking causing SAH and TAI in piglets
 Raghupathi et al., 2004, finding that repetitive shaking caused more injured axons than single shakes in piglets
 Starling et al., 2004, on confession cases

As to papers against, he cites

Plunkett & Goldsmith, 2004, who concluded that “Shaking simply does not generate accelerations that exceed any known injury tolerance values for traumatic brain injury.”

As to craniocervical apnea secondary injury, he cites “a growing body of literature:”

Hadley, Sonntag et al., 1989
 Johnson, Boal, Baule, 1995
 Shannon et al., 1998
 Smith, Andrus, Gleason & Hall, 1998
 Whitwell & Graham, 2000
 Geddes I
 Geddes II

Late neurologic and cognitive sequelae of inflicted traumatic brain injury in infancy. Barlow KM, Thomson E, Johnson D, Minns RA. Pediatrics 2005; 116(2): e174-e175. Found that 36% of the survivors had severe difficulties, 16% had moderate difficulties, and 16% had mild difficulties. Conclusion: AHT has a very poor prognosis. Incidentally, Dr Reece selected this paper as one of the top ten SBS papers of the last ten years at the 2006 National Shaken Baby Conference. I am not going to go into any more detail.

The abused child and adolescent. Platt MS, Spitz DJ, Spitz WU. In: Spitz WU, ed., *Spitz and Fisher’s Medicolegal Investigation of Death: Guidelines for the Application of Pathology to Crime Investigation*, 4th edition. Springfield, Ill., Charles C. Thomas, 2006. “The mechanism associated with shaken baby (impact) syndrome is forceful shaking, causing the head to jerk back and forth followed by impact, against a surface such as a wall or floor, sometimes a piece of furniture, other times a firm cushion or other type of upholstery.” (p. 376) Refers to the NAME position paper of 2001 and the AAP Technical Report of 2001. Discusses the origin of the syndrome from Caffey, 1972, 1974. Says that the term “shaken baby syndrome” “has become subject of much controversy and some investigators are now of the opinion that vigorous shaking of a child cannot cause the injuries once believed to be associated with this mechanism. skeptics believe that a head impact is necessary to sustain the characteristic intracranial injuries previously associated with shaking. A more recent trend is to use the term “shaken baby (impact) syndrome when faced with a child who has abusive intracranial injuries with or without evidence of a blunt impact to the head.” (p. 377) This remains “a vigorously debated topic.” “The lack of an impact site on the child’s head indicates that either no impact occurred or the impact was against a soft surface such as a bed or couch.”

Discusses Duhaime. "On the other hand, many experts (citing the NAME position paper, Kirshner in *The Battered Child*, 5th ed., 1997; Kirschner & Wilson, "The Pathologist's Perspective" in Reece, 1996; Gilliland & Folberg, "Some have no impact injury," 1996; Krous & Byard, Selected controversies, 1999; Byard, Lancet, 2000; Cohle et al., Am J Forens Med P 2000) believe that aggressive shaking of an infant can itself cause death." Citing witness accounts, confessions, and the absence of an impact site at autopsy, but saying that "It is generally accepted that the presence of an impact site partially depends on the nature of the impacting surface." (p. 377) "As the debate continues, it is important to understand that subdural hematoma, subarachnoid hemorrhage, diffuse axonal injury, brain swelling, and retinal hemorrhages can be seen in children who are both shaken and those who sustain a head impact. the mechanism of injury associated with both shaking and impact results in rotational forces that cause shearing injury to the brain and it is likely that both situations play a contributory role. The presence of a scalp bruise or skull fracture indicates an impact, however, coexistent shaking cannot be excluded. The lack of an impact site indicates the possibility of shaking alone or shaking in addition to an impact against a soft surface." (citing the NAME position paper.)

Cites Duhaime, "Head injury in very young children," 1992, for the following list of signs that should lead to consideration of possible abusive head trauma: [I have slightly edited the listing for clarity. --JKR]

- acute [and] remote fractures
- depressed, multiple, or basilar skull fractures
- [facial] contusions
- intracranial injuries
- retinal hemorrhages
- a history that changes frequently
- conflicting statements from those present
- a history that is incompatible with the injuries

Says that classic SBS is under six months, but can go out to several years of age. Cites NAME for the proposition that external injury is absent in 50-75% of cases. The most common external injury is fingertip bruises.

Discusses skull fractures and falls/ drops, (p. 378), saying that short falls can cause linear fractures, but that these fractures "are rarely associated with significant intracranial injury or neurologic sequelae, unless complicated by subdural or epidural hemorrhage. Although linear fractures may be caused by abusive head trauma, multiple, complex, or depressed fractures that involve the parietal or occipital bones and the base of the skull are more common in abused children."

Discusses **stomping**, and gives a photograph (p. 378-379 and 466). [the first time I have ever seen this mechanism mentioned in print.]

Subarachnoid hemorrhage. Commonly associated with AHT.

SDH. More than 90% of AHT cases at autopsy. Cites Ommaya & Yarnell, 1969 and Gennarelli & Thibault, 1982 for the proposition that SDH is due to bridging veins or venous sinuses associated with rapid deceleration-induced strains OR translational forces. Interhemispheric is common in AHT but is not diagnostic for abuse. (citing Ken Feldman in *The Battered Child*, 5th ed., and Plunkett, short-distance falls, 2001). The SDH in AHT serves as an indication that the brain has been subjected to shearing forces. (p. 379)

Discusses the differential diagnosis of SDH in children: aneurysms, AVM's, meningitis, sickle-cell anemia, leukemia, DIC, hemophilia, VWD, ITP, prolonged labor. Says these are all rare causes.

Discusses how to age a SDH.

Discusses REBLEED. Chronic SDH's at risk for rebleed are usually only seen in children with brain atrophy or surgically shunted hydrocephalus. (citing Lee et al., 1998). True rebleed has to be demonstrated histologically, by finding recent hemorrhage in a background of vascular lakes, fibrovascular response, endothelial proliferation, acute & chronic inflammation, and hemosiderin. AND it has to occur in the presence of a grossly observable chronic SDH. Says that should immediately prompt the question: what caused the original SDH? since chronic SDH that are at risk for rebleeding are usually only seen in children with brain atrophy or surgically shunted hydro. And notes Ken Feldman's finding that accidental

cases never had a chronic SDH or mixed acute & chronic SDH. So a rebleed a rebleed suggests abusive head trauma Cites Mark Dias et al., 1998 and Swift, McBride, 2000.

What about coagulopathy observed in 20% of pts with TBI due to the tissue thromboplastin activation of the extrinsic coagulation pathway? Notes that Hymel and Hulka demonstrated that these coagulopathies are secondary to the brain injury, not primary. There should be coags and platelets CBC obtained on admission for these patients. Then inquire after any family history of bleeding. Normal findings on these two inquiries “remove this issue from consideration.”

As to DAI, (p. 383) says this is associated with altered consciousness, is a more severe form of cerebral concussion. “The mechanism of injury consists of angular rotational acceleration induced strains causing shearing injury to the brain.” Cites Duhaime et al., The shaken baby syndrome, NEJM 1998 and Wilman, Bank, 1997 for the proposition that in accidental DAI in children (with a known time of injury), all children demonstrated an immediate decrease in their LOC. It was not necessarily instantaneous incapacitation (note that most of these children survived), but was lethargy, uncons, resp depression, apnea or seizures. (citing Johnson et al., 1995 and Atkinson, Mayo Clin Proc 2000). “In cases associated with respiratory depression or apnea, death probably occurs within minutes to hours outside of a hospital setting. Therefore, children diagnosed with DAI who die or have significant neurologic dys are symptomatic at the time of the injury.” (citing Adams et al., Histopathology 1989). Says an indicator of DAI is punctate or linear streak hemorrhages in the cerebral white matter, becoming more prominent over several days, but often may not be seen because infants’ small vessels stretch. Areas most susceptible to DAI are

corpus callosum
parasagittal white matter
dorsolateral quadrants of the rostral pons
fourth ventricle
aqueduct

In most AHT children it is not possible to demonstrate DAI by conventional methods if the survival is less than 18-24 hours. (Citing the NAME position paper) Even with longer survival, the findings are not guaranteed. The younger the child, the more difficult it is, because of the smaller axons. Even without demonstrating DAI, however, SAH, SDH, and RH are markers of axonal injury.

Discusses Geddes apnea HIE, with only two out of 37 children demonstrating DAI, but 11 having axonal injury in the brain stem and spinal nerve roots.

Discusses Lindenberg & Freytag’s contusion tears at the gray-white junction in infants under five months (giving the same old photograph). Healing leaves gliosis and small areas of yellow discoloration or small cystic spaces. But overall, contusion tears are rare. (p. 384-385)

Increased ICP. Cerebral edema develops within seconds.

As to RH: I am transferring this portion to the section on “RH,” above.

Identification of inflicted TBI in well-appearing infants using serum and CSF markers: a possible screening tool. Berger R, Dulani T, Adelson D, Leventhal J, Richichi R, Kochanek P. Pediatrics 2006; 117: 325-332. According to Dr Reece’s review at the 2006 National Conference on SBS, the authors concluded that serum and CSF NSE and MBP have potential for use as screening tests for iTBI in normal-appearing infants. S-100 is worthless. Thus for NSE had 76% SENS, 66% SPEC. For MBP had 36% SENS, 100% SPEC. In a prospective study of 98 infants in the ER who had symptoms such as ALTE, over 4 episodes of vomiting in 24 hours, seizures, lethargy, fussiness. Had followup at 6 and 12 months. One big difference between the iTBI group and the no brain injury group was time of rescue: iTBI was 49 hours, nonTBI group was 7 hours.

Inflicted head injury in infants - issues arising from the Geddes hypothesis. Jonathan A G Punt, Consulting Pediatric Neurosurgeon (retired). BMJ 2006 March 1; <http://adc.bmj.com/cgi/eletters/91/3/205> Complaining about the way summary statements from doctors get processed into the mass media and into the courts. And also the failure of the peer-review process prior to items reaching the mass media. In particular the Geddes unified hypothesis. Complaining that the flaws in the Geddes papers did not get noted by the press or the bar until the UK SBS litigation was already at an advanced stage (in the Court of Appeal, R v Harris; R v Rock; R v Cherry; R v Faulder [2005] EWCA Crim 1980, [at para 58]). Cites approvingly a paper by Richards et al. (Richards PG, Bertocci GE, Bonshek RE et al. Shaken baby

syndrome. Arch Dis Child. 2006; 91: 205-206.) for condensing the arguments made in the Court of Appeal and making recommendations for the role of doctors in court in the future. "[T]he three papers that constituted the 'unified hypothesis' raise questions as to the quality of the peer review process to which they were subjected prior to publication. In particular, as pointed out elsewhere (6), the second of these papers contained a statement that could not be supported by the data, namely '... it may not be necessary to shake an infant very violently to produce stretch injury to its neuraxis.' It is remarkable that such an unfounded assertion, carrying powerful implications, was permitted to go forward in a distinguished scientific journal..." Footnote (6) refers to the Punt, Bonshek paper of 2004, *supra*. Notes that in the Court of Appeal, Geddes emphasised in her testimony that her theory is just a theory, and stated that she "would be very unhappy to think that cases were being thrown out on the basis that my theory was fact." Complains that such doubts should have been expressed much earlier. "It is a sad day for medical science if mature reflection that leads to an amended view only comes to light under the rigours of cross-examination."

"Shaken baby syndrome": Do confessions by alleged perpetrators validate the concept? Leestma JE. J Am Phys Surg 2006; 11: 14-16. Cites the criminology literature on false confessions. Rehashes the author's previous arguments that biomechanically shaking cannot cause the injuries seen, that the SBS literature consists of case reports, which are an unreliable form of research, and points out that the admittedly strong association between confessed shaking and a particular constellation of findings does not, strictly speaking, establish that the shaking is what *caused* the findings. (In other words, an observed association is not the same thing as a demonstration of cause and effect.) [Ordinarily in science, a demonstration of cause and effect requires an experiment, which is impossible in the case of SBS. --JKR] Also argues that in the SBS cases, the clinicians failed to rule out other possible causes, among which he lists vaccination, drugs, birth injuries, congenital or acquired coagulopathies, and metabolic diseases. Brian Holmgren, JD points out in his review for the Summer 2006 *Quarterly* that, having criticized the SBS literature for relying on case reports, Dr Leestma himself relies on case reports. Holmgren in his review criticizes Leestma for relying on articles that "have been ridiculed in the professional literature," namely Howard, Bell & Utley, 1993, Donohoe, 2003, and Bandak, 2005.

Findings in older children with abusive head injury: does shaken-child syndrome exist? Salehi-had H, Rosas AJ, Rogers KK, Pediatrics 2006; 117: e1039-e1044. Four cases. Dr Block's review in the Autumn 2006 *Quarterly* says:

7 year old with traumatic retinoschisis, SDH, SAH, multiple old and recent injuries, stepfather convicted.

3 year old with pattern injuries etc

2 ½ year old with similar

6 year old with similar.

All perpetrators convicted. The authors postulate that these SBS-type injuries in chil with no skull fractures shows that shaking could be the mechanism. Hard to argue with that, but Dr Block points out that it could be considered circular logic: we think the chil were shaken, we find injuries that we have already defined as shaking, therefore the chil were shaken. But still, with arguably non-impact intracranial injuries and an incompatible history etc., the possibility of shaking is not ruled out. But still, we are inferring *a posteriori*. Where are the nannycam tapes or eyewitnesses? --JKR

Age-related incidence curve of hospitalized shaken baby syndrome cases: convergent evidence for crying as a trigger to shaking. Barr RG, Trent RB, Cross J. Child Abuse & Neglect 2006; 30: 7-16. Reviewed 273 admissions coded as SBS and found that the incidence curve for normal crying is similar but not identical to that for these SBS cases. Crying peaks at 5-6 weeks. SBS peaked at 10-13 weeks with tails to 2-3 weeks and 36 weeks. From Suzanne Starling's review in the Autumn 2006 *Quarterly*.

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. Only 17% had an extracranial injury; rib fxx were present in 14%. Bruising of the head was visible in 47%. Skull fractures were present in 22%. RH in 81%, SDH 89%. Coagulopathy 47%. There is nothing you can do for these patients. 50% died within 24h and 86% within 1 week.

Analysis of caretaker histories in abuse: comparing initial histories with subsequent confessions. Flaherty EG. Child Abuse & Neglect 2006; 30: 789-798. Analyzed 45 confessions and compared them with the initial histories. Showed that crying was the usual trigger.

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. The purpose of this multicenter prospective comparison study was to study differences in OUTCOMES between inflicted and noninflicted head trauma in small children. Six-month followup was used. It was also used to test the hypothesis that inflicted injuries would more often be from noncontact mechanisms (i.e. "rotational cranial acceleration and/or deceleration," i.e. shaking). Between 2003 and 2006 they prospectively acquired 54 hospitalized patients under 36 months of age who had head trauma. Had

Inflicted	Noninflicted	Undet
11	30	13

Under "Noninflicted," the study included the following:

4 MVA's	
26 falls	
3	from heights over 10 feet
2	from heights between 6 and 10 feet
17	from heights less than 6 feet
4	stair falls

The basic findings were that at a level of $P < .001$ the inflicted had the following characteristics compared to the noninflicteds:

1. More frequently had a noncontact injury mechanism
2. Greater injury depth
3. Suffered acute cardiorespiratory compromise
4. Lower GCS
5. More frequent and prolonged impairment of consciousness
6. Bilateral hypoxic-ischemic brain injury
7. Worse mental outcome
8. Worse motor outcome

Here are some other discriminant data taken from table 3:

	Inflicted	Noninflicted	Undet
Mean initial GCS	8	13	13
Injuries per imaging:			
Soft tissue	27%	77%	69%
Skull fx	18%	77%	62%
EDH	9%	7%	15%
SDH	73%	30%	23%
SAH	27%	30%	23%
Brain contus	64%	27%	15%

DAI	27%	0%	0%
Unilat swel	18%	7%	8%
Bilat swel	64%	3%	8%
Death	9%	3%	0%

How did the authors determine the presence of a noncontact injury mechanism? Very simple. They diagnosed contact mechanism by the presence of craniofacial bruising, abrasions, lacerations, [localized] swelling, subgaleal hematoma, cephalhematoma, skull fracture, or epidural hematoma. They diagnosed noncontact mechanisms by the absence of any of these plus the presence of “injuries limited to acute concussion, diffuse axonal injury, or an abnormal subdural collection that extends from the interhemispheric region.” There were two other mechanistic categories: “combined” mechanisms, and “undetermined” mechanisms. Combined mechanism was when there were both contact and noncontact injuries. Undetermined mechanism was atypical head injury [my word], as follows: “injuries limited to subarachnoid hemorrhage, brain contusions, brain lacerations, or an abnormal subdural collection that does not extend from the interhemispheric region.” [Comment by JKR: Shaking denialists will argue that in presuming that their defined “noncontact mechanism” criteria actually determine the presence of a noncontact mechanism, the authors are begging the question. What their criteria do is determine the absence of clinical evidence of impact. That is not the same as saying that they determine nonimpact. To say that we have no evidence of impact is different from saying that we have *excluded* impact. This is the same debate that has been going on for years. These cases were not autopsied. *Viz* Hadley, Sonntag et al., 1989; Sato et al., 1990; Duhaime et al., 1992; Howard, Bell & Uttley, 1993 (I know that paper has been roundly criticized); Gilliland, Folberg, 1996; Taff et al., 1996; Duhaime et al., 1998; DiMaio, 1998; Ewing-Cobbs et al, 1998; Krous & Byard, 1999; NAME Position Paper, 2001; AAP Technical Report, 2001; Rorke, 2003; Geddes & Plunkett, 2004; Starling, 2005; Leestma, 2005; Minns, 2005; Platt, Spitz & Spitz, 2006;

On that basis, the mechanisms found were as follows:

	Inflicted	Noninflicted	Undet
Pure contact	1	19	8
Pure noncontact	8	1	1
Combined	2	7	3
Undet	0	3	1

The 7 noninflicted cases that had combined mechanisms were all falls. These cases are separated out for specific discussion as follows: “Seven of 26 [falls] revealed subdural hematoma... These 7 subjects included 3 with SDH that extended or originated from the interhemispheric region, indicative of a noncontact mechanism of injury. All 3 of these young children fell from a height of only 3 to 6 feet; manifested associated contact injuries; revealed only superficial, focal, cortical brain injuries; and experienced no alterations of consciousness. These cases seem to demonstrate that noncontact (i.e. interhemispheric) SDH of minimal clinical significance can result from a short-distance fall and that the injury thresholds or biomechanical requirements for noncontact SDH and acute concussion, also a noncontact injury, are different.” [Comment by JKR: With only six months followup, I’m not sure it is possible to say that these cortical injuries were of minimal clinical significance. Obviously they were not life-threatening or consciousness-impairing, but the possibility of long-term sequelae such as epilepsy, subdural hygroma, behavioral sequelae, or impaired school performance cannot be excluded.]

Cites specific sources in the literature for the proposition that abusive acts have been linked to:

1. subdural, subarachnoid, and retinal hemorrhaging
2. localized axonal injury in the region of the craniocervical junction and cervical cord; acute respiratory compromise or arrest; loss of consciousness; hypotension
3. secondary diffuse hypoxic-ischemic brain injury with swelling

The diagnostic criteria used to define “Inflicted” were as follows:

1. Cases in which the child’s primary caregiver admitted abusive acts that could be linked to the child’s acute clinical presentation for traumatic cranial injuries
2. Cases in which an independent witness verified abusive acts that could be linked to the child’s acute clinical presentation for traumatic cranial injuries
3. Cases in which a child not yet cruising or walking became clearly and persistently ill with signs of acute cardiorespiratory compromise linked to his/her traumatic cranial injuries while in the care of a primary caregiver who denied any knowledge of a head injury event
4. Cases in which the child’s primary caregiver provided an explanation for the child’s head injury event that was clearly developmentally inconsistent with the parent(s)’ description of their child’s developmental capabilities
5. Cases in which the child’s primary caregiver provided an explanation for the child’s head injury event that was highly inconsistent with repetition over time [meaning repetition of the history, i.e. a changing history --JKR]
6. Cases in which the head-injured child revealed at least two noncranial injuries considered moderately or highly specific for abuse

The diagnostic criteria used to define “Noninflicted” were

1. Cases in which the child’s primary caregiver described an accidental head injury event that was developmentally consistent, historically consistent with repetition over time, could be linked to the child’s acute clinical presentation for traumatic cranial injuries, and occurred in the absence of any noncranial injuries considered moderately or highly specific for abuse
2. Cases in which an accidental head injury event was witnessed independently and could be linked to the child’s acute clinical presentation for traumatic cranial injuries (e.g. MVA)

Diagnostic criteria for “Undet” were

1. Cases meeting criteria for both inflicted and noninflicted etiology
2. Cases not meeting any criteria for either inflicted or noninflicted etiology

As to these criteria for inflicted, the authors comment that they “are free of circular logic and other inherent biases.” They also state that the criteria are conservative, as shown by the fact that these criteria categorized more subjects as undet than they categorized as inflicted.

<http://www.safe4baby.org>

BIOMECHANICS

Whiplash injury and brain damage: an experimental study. Ommaya AK, Faas F, Yarnell P. JAMA 1968 Apr; 204(4): 285-289. A review of their series of rhesus monkey experiments concerning automobile whiplash injury. The authors begin by mentioning their earlier work with monkeys concerning “concussion,” by which they mean traumatic brain injury sufficient to cause loss of aversive reactions. Say that in this work with linear acceleration, they discovered that if the monkeys wore a cervical collar, half of

them did not get brain injury. Why? “The most significant variable affected by the cervical collar appeared to be the amount of rotational displacement of the head on the neck... Because such displacements are readily produced without head impact by a whiplash injury, we predicted that whiplash injury alone should produce cerebral concussion if rotational displacement of head was indeed a significant physical mechanism in head injury by impact.” Says that this prediction was confirmed by experiments with monkey whiplash producing concussion (loss of aversive reactions) at 40,000 radians/sec² “if the duration of such accelerations is greater than 10 msec.” (citing the 1966 Stapp Car Crash Conference, “The role of ‘whiplash’ in cerebral concussion.”) Saying that their more recent work (the work presented in this present article) “has extended and supported these findings and significantly has revealed that in addition to reproducing the entity of experimental cerebral concussion without direct impact to the head, experimental whiplash injury can produce [not only “concussion” but] consistent brain damage...as evidenced by subarachnoid and subdural hemorrhage, cerebral contusions, and breakdown of the blood-brain barrier...” In other words, the full spectrum of brain injuries previously thought to be obtainable only from direct impact. Forty monkeys. Describe their machine, with a monkey chair on a twenty-foot roller track, with no support for the monkey’s head and neck. The chair would be hit from behind by a piston, causing the monkey’s head to snap backward, “so as to mimic a rear-end collision.” Accelerometer. 50% had concussion at 100 G’s, generally lasting for a few minutes. The experimenters then waited 24 hours after the experiment, and sacrificed the animals by perfusion-fixation with 10% formalin. Performed gross pathology on the brain and spinal cord. Results: 40 monkeys hit: 19 had clinical “concussion.” Of these, 15 had “surface hemorrhages which were primarily subdural in nature.” The surface hemorrhages they show in figures 3 and 4 appear to me to be focal subarachnoid hemorrhage. They state that as to the location of these surface hemorrhages, “[T]he parietal parasagittal zones, the medial supracollosal surfaces of the two cerebral hemispheres, the tips of the frontal and temporal lobes, the inferior orbitotemporal and temporo-occipital (particularly the latter) cerebral surfaces, and the brain stem, including the upper cervical cord, were primarily involved.” “The cortical draining veins enter the sagittal sinus in the parasagittal zone and thus are particularly liable to rupture. We found that subdural bleeding in these areas was by far the commonest visible lesion.” With one exception, none of the injured animals had any parenchymal lesion by gross examination. Of the 31 without concussion, none had any gross brain injury or hemorrhage. Figure 5 is a plot of the acceleration duration against the acceleration. It is a downsloping curve. It shows that the range of accelerations used was from 12,000 to 100,000 radians/sec². The range of durations was from 1 to 13 msec. On fig. 5, all the injuries were above a downsloping curve which starts at 100,000 R/sec² with 3 msec and curves down to 13,000 R/sec² and 11 msec. The longer the duration, the less radial acceleration it took to give injury. In the comment, they say that they plan to repeat this in a series of species with progressively increasing brain weight, e.g. squirrel monkey, rhesus monkey, chimpanzee), in order to be able to use scaling to predict the injury threshold for whiplash brain and cord injury in humans. They intend to assume that the level of injurious rotational acceleration is inversely proportional to the two-thirds power of the mass of the brain. (Their basis for making this assumption is drawn from their own work presented at the 1967 Stapp Conference.) Suggest that the threshold of injury for the human CNS will be in the range of 6,000 to 7,000 R/sec². [Duhaime et al in 1987 will report that the radial accelerations obtainable in their doll model by manual shaking are 1,138 R/sec², and by impact 52,000.] Discuss the rel to automobile whiplash symptoms, stating that these experiments suggest that CNS injury with subarachnoid hemorrhage is quite possibly present in a significant number of automobile whiplash patients. They mention in passing that “shear strains generated by rotational acceleration of the head and its contents” is the proposed mechanism of concussion, just as they believe they have demonstrated it is for surface bleeding.

Cerebral concussion and traumatic unconsciousness: correlation of experimental and clinical observations on blunt head injuries. Ayub K. Ommaya and T. A. Gennarelli. Brain 1974; 97: 633-654 A review article going over the history of research on traumatic concussion. They give a schematic chart of the biomechanics of head trauma. In this chart, starting with mechanical event, the progression bifurcates to “static” effects and “dynamic” effects. “Static” or static loading means forces applied slowly (duration over 200 msec), usually when the head is slowly crushed. The static effects have focal and diffuse effects. Both focal and diffuse would include strains and shearing. Shearing breaks out into four subcategories: focal concussion, [diffuse] cerebral concussion, primary brain lesions, and skull fractures. All of these lead to biologic response, including secondary brain lesions. In the real world, static loading usually leads to only focal injuries. Dynamic loading (rapid loading) breaks out into impact and impulse. Impulse is where

the head is moved suddenly without an impact. Dynamic loading injures the brain both through the stresses and strains of inertial loading (movement of the brain), and through contact phenomena, such as skull bending and wave propagation. The authors do not go into contact phenomena in detail. They concentrate on inertial loading, the main cause of traumatic unconsciousness in both impact and impulse loading events.

“Until recently the relative significance of the two components of inertial loading -- translatory and rotatory or angular movements of the head, was undecided. (reviewing the literature to the effect that some workers have thought that only rotational could cause LOC, others that both rotational and translational). Our recent experiments studying these components in relative isolation have shown that ... **rotation of the head appears to be necessary for loss of consciousness as well as productive of diffuse and focal lesions of the brain...** Translation of the head in the horizontal plane on the other hand produces essentially focal effects only (cortical contusions and intracerebral hematomas).” (Citing acceleration of squirrel monkeys up to 1400g; Gennarelli et al., 1971; Ommaya et al., 1973). Rotational loading tends to produce lesions at brain surfaces and at zones of changes in density of tissue. [Compare Lindberg & Freytag, 1969, Geddes et al., 2001. -JKR] Theoretically the tissue strains causing damage should decrease from the surface to the center of the brain. Disconnection should occur at progressively deeper circumferential ‘layers’ of the brain mass as shear strain input increases. Traumatic unconsciousness should not occur until this reaches the ascending reticular activating system in the mesencephalon. (Theoretical model.) But, material properties of the brain tissue and cranial geometry such as inner skull contour and dura, vessels, etc. will influence this in complex inhomogeneous ways. Plus there could be contact and translational loads in the event as well as impulse and rotational loads. Thus a simple spherical model will not be predictive. But the authors stick with their model of progressive severity of impairment of consciousness depending on progression inward of the disconnections from the surface down to the mesencephalon (rostral brain stem) depending on the magnitude of the (rotational) inertial loading.

The authors describe certain biomechanical discoveries they made with squirrel monkeys and rhesus monkeys and chimpanzees. One was that during impact they could protect the animal from concussion by having the monkey wear a rigid cervical collar. Another was that the rotational acceleration threshold for concussion was twice as high with impulse loading as it was with impact loading. (Ommaya et al, 1971b). They developed a tolerance curve to rotational loading to predict the injurious rotational acceleration threshold for man (1970, 1971). Then they did experiments with pure inertial loading. Their squirrel monkey helmet machine (illustrated) would give pure rotation or pure translation up to 1230 g without impact. The rotated animals had concussion; the translated animals did not. They refer to this experiment as “head shaking -- rotation.” All twelve rotated monkeys had concussion, SDH, and SAH. Most had parenchymal petechiae. Few had cortical contusion, none had ICH. (The squirrel monkey has a relatively larger brain and head.) In “head shaking -- translation,” no monkeys had concussion, the SDH’s were focal only, and no SAH, no parenchymal petechiae, some had ICH. Hemorrhages in the brain stem were remarkably scarce in either group.

Discusses human clinical material. Claim that their “concentric layer’ hypothesis of rotational injury is supported by the clinical progression of recovery from traumatic coma with consciousness and memory recovering last. Brain pathology is discussed. Refer to work on subdurals by Ommaya and Yarnell (1969) attributing SDH’s in rearward falls and whiplash injury of adults to “rotational components of inertial loading.” They note from the work of Strich and others on tears and neuronal lesions that this work is consistent with their hypothesis that the brain stem should be the least vulnerable area to inertial loading.

The authors admit that pathologic studies in experimental animal brains have limited human applicability. Some work apparently had shown that brain stem and cervical cord lesions in animals were the basis for primary concussion, which would contradict their theory. They suggest scaling models to human, but agree that scaling models would need to be validated.

The shaken baby syndrome: a clinical, pathological, and biomechanical study. Duhaime A-C, Gennarelli TG, Thibault LE, Bruce DA, Margulies SS, and Wiser R. *Journal of Neurosurgery* 1987; 66: 409-414. Retrospective review of 48 cases suspicious for child abuse admitted with either

- (a) RH plus SDH or SAH, or
- (b) SDH or SAH plus interhemispheric blood.

13 fatalities, all were autopsied. "All fatal cases had signs of blunt impact to the head., although in more than half of them these findings were noted only at autopsy."

The much-discussed experiment with an accelerometer model showing that the angular accelerations at the cranial center of mass developed by shaking were too small to reach the estimated thresholds for SDH and DAI causation estimated by scaling from the earlier primate model of Gennarelli et al, (1982) (see below under "Head injury -- fall versus inflicted.") The peak tangential acceleration obtainable by shaking was 9g, while that obtained by slamming was 428 g. The impulse durations were 106 msec and 21 msec respectively. The peak angular velocities were 60 and 548 sec(-1), and the angular accelerations were 1,138 and 52,475 sec(-2) respectively.

The article also includes clinical data from 48 allegedly shaken babies aged 1 mo to 2 years. 63% had clinical, radiological, or autopsy evidence of impact to the head, 37% did not. All of the 13 autopsy cases had scalp contusions with or without skull fractures.

Duhaime's accelerometer model has been criticized by Hadley (1989) and Gilliland (1996) on the ground that the "head" of the doll model was packed tightly with wet cotton, which did not allow for an extraaxial space or secondary movement of the "brain" vis-à-vis the "skull," but this criticism does not reach the biomechanical argument of Gennarelli et al. that the rupture threshold for bridging veins cannot be attained by shaking. As Plunkett puts it, relying on this work, "I have no doubt that you can kill a baby -- probably even practically tear its head off -- by shaking it. But you cannot cause a subdural hematoma that way." (Plunkett J, personal communication, 2001.) And subdural hematoma is almost (note I said "almost") a *sine qua non* for the definition of SBS; SDH is almost always present in fatal SBS cases. I'm not sure about nonfatal. (See NAME Position Paper, 2001 saying that SDH is "probably always present" in infant AHT and reported in 98%.)

But note that this SDH-threshold argument is not the only biophysical threshold argument that bears on SBS. There is also the axon-rupture threshold argument. Bearing in mind that as Kaufhold, Spivack and Case have taught, the essential lesion in AHT is not the SDH, which in and of itself is not harmful, but rather is the axonal or neuronal injury of which the SDH is an indicator. But these authors posit (See NAME Position Paper, 2001) that the rupture threshold for infant bridging veins is *lower* than that for infant brain parenchyma (!). See also Reece & Kirschner, 1998, emphasizing brain injury. Kleinman argues that the Ommaya-Gennarelli primate shearing thresholds obtained on myelinated (adult primate) brain do not apply to unmyelinated infant brain with its higher water content. (*Diagnostic Imaging of Child Abuse*, 2d ed., 1998, p. 289.) He also argues from the larger subarachnoid space/ CSF volume of infants allowing more movement of the infant brain. He also argues that repetitive oscillatory motion in shaking could generate cumulative effects on the brain tissue, quite apart from any single-impulse shearing threshold. He further adduces apnea/ infarction as an independent brain injury mechanism in shaking, anticipating the work of Shannon (1998) and Geddes (2001).

Leestma (2005) abstracts three cases from this article in his collection of all the published cases in which there was an admitted history of shaking. As follows: "In case 3, the child apparently had a history of a fall or having been hit or admittedly shaken by someone (not specified). No apparent cranial impact was noted and no skull fracture seen. Apparently, no retinal hemorrhages were noted. Case 11 had a history of an apparent fall or having been hit and shaken by someone (not specified). Injuries [included RH and SDH]. In case 13, the history was similar to both cases with an apparent fall and/or having been hit or shaken by someone (not specified). There were skull fractures, and other injuries [SDH] were noted."

Head injury in very young children: mechanisms, injury types, and ophthalmologic findings in 100 hospitalized patients younger than 2 years of age. Duhaime A-C, Alario AJ, Lewander WJ, Schut L, Sutton LN, Seidl TS, Nudelman S, Budenz D, Hertle R, Tsiaras W, Loporchio S. *Pediatrics* 1992 Aug; 90(2): 179-185. 100 consecutively admitted head injury patients under 24 months. 24 inflicted, 32 possibly

inflicted, 44 accidents. All had opth. RH were found in 10: 9 abuse and one fatal high-speed MVA. All 10 RH pts had SDH. Discusses translational versus rotational mechanisms. Short falls translational are not of great clinical consequence, even when they cause fx, unless an EDH occurs. Higher falls translational cause complex, depressed, or basal fractures or multiple fxx as well as focal brain contusions and SAH; unless the translational force is extreme, damage is predominantly focal and recovery of global neurol function is usually rapid. In contrast, it has been shown experimentally and clinically that more significant diffuse brain injury results from the introduction of a significant **angular component** to the head's deceleration. Angular (rotational) deceleration leads to much more brain deformation and shear strain...and at progressively greater angular decelerations the phenomena of concussion, SDH, and DAI will occur. (This is taken from Genarelli's work on monkeys, but here explicitly transposed to humans.) SDH were uncommon in accidental injuries, occurring in only three ch inv in MVA. In contrast, SDH and diffuse SAH are common in inflicted injury in very young ch, occurring in 13 of 24 pts in this series. ... "The mechanism postulated is that of a child being held by the perp who shakes, swings, or throws the ch, **the head thus moving in an arc**, stopping abruptly against a surface. Previous autopsy studies and biomech using infant models suggest that shaking alone does not generate sufficient deceleration forces to cause the SDH and brain injuries seen in these children (citing the 1987 biomechanical study.) The frequent radiologic or clinical findings of blunt impact in series of "shaking" injuries corroborate this conclusion, as does the rarity of an *unsolicited* history of shaking. [italics added --JKR] For these reasons, we now refer to this syndrome as the '**shaken impact syndrome**.' The presence of fractures and bruises will be determined by the surface against which the rotating head decelerates."

Gives Table 4 which lists the injury types that can be expected from various common mechanims:

Fall under 4 ft	Concussion Linear fx EDH Ping-pong fx (if hit corner) ?Depressed fx
Fall over 4 ft	Above injuries Depressed fx Basilar fx Multiple fxx SAH Contusion ?SDH ?Stellate fx
MVA	Above injuries SDH DAI

"...[I]t is clear that RH can occur under a variety of circumstances, including vaginal delivery, spontaneous SAH, systemic HTN, intracranial HTN, thoracic or abdominal trauma, and in-hospital resuscitation. Whether superimposed hypoxia or ischemia with reflow exacerbates the finding remains unknown. Traumatic retinoschisis resulting from acceleration/deceleration forces applied to the eye has also been postulated as a mechanism for RH. The latter may be particularly relevant in very young children because of the more solid consistency or the vitreous body in the infant and the stronger adhesions at the vitreoretinal interface. Threshold values for the degree of decleration required to result in RH have not been established. Since this study was completed we have seen three additional pts with well-witnessed accidental head injuries who had acute RH..."

Diffuse axonal injury: its mechanism in an assault case. Takeshi Imajo MD, Am J Forens Med Path 1996 Dec; 17(4): 324-326. This adult male was multiply kicked in the side of the head by assailant, survived 13 days. Had DAI with a microscopic lytic lesion in the corpus callosum. Dr. Imajo posits low-acceleration coronal rotational injury with freely moving head.

Techniques for developing child dummy protection reference values. Klinich KD, Saul R, Auguste G, Backaitis S, Kleinberger M. Report by the child injury protection team, NHTSA, October, 1996. Cited in Cory and Jones, 2003, as postulating a 50% concussion probability at only **4,000 sec(-2)** peak angular acceleration for a pediatric auto vs. pedestrian model.

Abusive head trauma? A biomechanics-based approach. Kent P. Hymel, Faris A. Bandak, Michael D. Partington, Ken R. Winston. Child Maltreatment 1998 May; 3(2): 116-127. See under "Head injury -- fall vs. inflicted." These authors import results from the transportation industry and military studies of head-injury mechanics. This world is divided into contact and noncontact injuries. As far as noncontact injuries or injuries that are partly noncontact: large subdurals (i.e. not confined to the area under an impact site or fracture) are caused by anteroposterior angular acceleration that is sudden and short. DAI is caused by slower angular acceleration in the coronal plane. DAI leads to immediate LOC.

Age-dependent material properties of the porcine cerebrum: effect on pediatric inertial head injury criteria. Kirk L. Thibault and Susan S. Margulies. Journal of Biomechanics 1998; 31: 1119-1126. Reviewed by Betty in Quarterly for Jan 2001. Took cerebra from baby pigs at age 3 days and one year. Subjected them to oscillation at varying frequencies from 20 Hz to 200 Hz and measured the shearing modulus (tolerance to oscillation.) Found that the shearing modulus increased with age and with increasing frequency. Hence was more susceptible at lower age and at low frequency. Betty Spivack in her review in Quarterly points out that the suggested SBS frequency as per Duhaime's experimental model of 1987 is 3–10 Hz, below the freq used here. The authors think that a 3 day old pig cerebrum approximates the viscoelastic properties of a one month old human cerebrum, but Betty thinks that more likely 5-6 month human. If so, then to approximate a one-month human brain you would have to use a fetal pig. Betty says that at low frequencies these data are consistent with low tolerance for human early infant. The authors apply their material data retrospectively to the shaking rotational accelerations measured by Duhaime et al., and find that even adjusting those accelerations for this new viscoelastic modulus, **the accelerations remain well below the injury thresholds for "concussion, SDH, and DAI."** Betty points out that since the present experiments were performed on isolated brain tissue without skulls and without any pathology done, no statements are warranted about concussion, SDH, or DAI. Betty points out that the authors also state that shearing injury is possible with linear decel if impact; Betty says there is no support in the published literature for this. Betty says that this paper gives important data for us "to begin to understand" the biomechanical properties of the human infant brain, which is "crucial" to an understanding of accidental and non-accidental traumatic brain injury in infants.

Infant rat model of the shaken baby syndrome: preliminary characterization and evidence for the role of free radicals in cortical hemorrhaging and progressive neuronal degeneration. Smith SL, Andrus PK, Gleason DD, Hall ED. J Neurotr 1998; 15: 693-705. Said by Levin (2001) to be a DAI model. Shook rats in canisters. This paper is cited by Hymel (2005) as one of the papers supporting craniocervical apnea – HIE as a common sequela of shaking.

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. (on Medscape). See above under 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. Discusses the possible mechanisms of RH as being in need of further study. Does not comment in detail on how such a minor impact could have caused an SDH.

Traumatic axonal injury: practical issues for diagnosis in medicolegal cases. Geddes JF, Whitwell HL, Graham DI. Neuropathology and Applied Neurobiology 2000; 26: 105-116. See above under "Shaken." Contains a discussion of biomechanics.

Maturation-dependent response of the piglet brain to scaled cortical impact. Duhaime A-C, Margulies S, Durham SR et al. J Neuros 2000; 93: 455-462. At ages of 5 days (human infant by brain maturation level), one month (toddler by brain maturation level), and four months (adolescent by brain maturation level). Autopsied seven days after injury. Injured tissue increased with increasing age. Betty Spivack

quotes the conclusion in the *Quarterly* for July 2003: "...the immature brain is particularly well suited to resisting injury, which ... contradicts the notion of infant fragility. This would suggest that the poor outcome seen in clinical series of head-injured infants does not reflect intrinsic increased vulnerability ... but likely represents different magnitudes of injury, different injury types (such as inertial injury and/or subdural hematoma seen in NAI), or other factors such as superimposed hypoxia/ischemia." Betty comments that the resistance of the infant-cognate brain tissue to impact injury may explain the good outcome of infants who fall. Betty compares the same authors' J Neurotrauma 2002 art on whiplash vulnerability of newborn piglets, which showed high vulnerability to TAI, SDH, and apnea.

Traumatic axonal injury after closed head injury in the neonatal pig. Raghupathi R, Margulies S. J Neurotrauma 2002; 19: 843-853. Found high vulnerability to whiplash-induced TAI, SDH, apnea. Compare the low vulnerability to impact injury found by the same authors in J Neurosurg 2000. Hymel (2005) cites this article as having concluded that "rapid rotation of the piglet head without impact results in SAH and TAI similar to that observed in children following severe brain injury." See also the same group's follow-on experiments with shaking piglets and finding that if they shook them twice instead of just once, they got significantly more foci of injured axons. (2004, below)

A flask full of jelly: the first in vitro model of concussive head injury -- 1830. Feinsod M. Neurosurgery 2002; 50: 386-391. (Haifa) Guy in 1830 struck such a flask and observed concussive waves in the jelly. Carol Jenny observes in the *Quarterly* for July 2003 that "we do not know the basic biophysical properties of the infant brain." See Duhaime, 2000 on piglet brain.

Biomechanics and neuropathology of adult and paediatric head injury. Ommaya AK, Goldsmith W, Thibault L. Br J Neurosurg 2002 Jun; 16(3): 220-242. This is not experimental work, but a review article and argument in favor of biomechanical modelling as a way of predicting age-related injury severity from mechanism by means of scaling from known experimental data to unknown human infant and adult cases. It would be the last word of the late Professor Goldsmith, who died in 2003.

Injury biomechanics for aiding in the diagnosis of abusive head trauma. Pierce MC, Bertocci GE, Berger R, Vogeley E. (Pittsburgh Children's Hospital) Neuros Clin N Amer 2002 Apr; 13(2): 155-168. Additional biomechanical research is needed to bridge the gap between clinical observations and experimental predictions. The answer to the question, "Is it possible" is based on clinical experience and objective reasoning.

Anthropomorphic simulations of falls, shakes, and inflicted impacts in infants. Prange MT, Coats B, Duhaime A-C, Margulies SS. J Neurosurg 2003 Jul; 99(1): 143-150. Duhaime's group built a new dummy of a 1 ½ month old infant with a 1.13 kg head and a 9.2 cm radius of rotation to the cranial center of gravity at a 10.6 pound total body weight. Its skull was a hollow plastic molded on a toy doll head purchased at the toy store and made with a plastic chosen to have deformability characteristics half way between those of infant skull bone and suture. This model skull was covered with a model scalp made of 1.25 mm of latex. Model is intended to be more biofidelic than the 1987 one. In this experiment, unlike the 1987 one, they were able to measure angular velocity directly, not just acceleration. They measured total change of angular velocity, and also angular acceleration, including peak change of angular velocity and peak angular acceleration. They did three things to the dummy:

shook it
slammed it
dropped it.

The slams and drops were onto three different surfaces:

lab bench
lab bench covered with a ¼" carpet pad
4" foam rubber

They measured

maximum change in velocity (peak to peak)
continuous angular acceleration
peak angular acceleration
pulse duration

They compared the resulting data with the very limited empirical data available from animal and cadaver experiments, using scaling (while acknowledging that scaling itself has not been validated for this use.)

Their basic conclusions were the following:

1. The magnitudes found in shaking and falls of 3 feet or under are **unlikely** to produce TAI or SDH.
2. The magnitudes found in falls of 5 feet onto concrete or thin carpet **could cause** TAI or SDH.
3. The magnitudes found in slamming **would be likely** to cause TAI and SDH.

Magnitudes:

	peak accel rad/sec ²	delta-V rad/sec	pulse duration msec
1-ft fall			
onto concrete	42,000	70	9
onto ¼" carpet	30,000	60	9
onto bed	1,000	18	40
3-ft fall			
onto concrete*	89,400	83	4
onto ¼" carpet	18,000	80	7
onto bed	2,000	25	35
5-ft fall			
onto concrete	80,000	85	3
onto ¼" carpet	70,000	85	3
onto bed	5,000	20	11
slam			
onto concrete*	173,000	275	3
onto ¼" carpet	160,000	170	35
onto bed			
shake*	2,640	56	170

definitions: In all cases except the ones with asterisks, I have read the values off of bar graphs.
These are average values averaged over 134 falls and 61 shake-slams.
The ones with asterisks are exact measured values given in the text for a single representative sample event.

peak accel: the highest accelerometer reading
delta-V: the absolute change in velocity from maximum forward to maximum backward
pulse duration: the elapsed time from peak forward velocity to peak backward velocity
concrete: in the case of slams, a stone lab benchtop, having an elastic modulus in the range of 20-60 Gpa.
in the case of falls, a concrete floor having a similar elastic modulus.
carpet: a ¼" carpet pad over lab bench. It had an elastic modulus of 621 kPa.
bed: a 4" foam pad, having an elastic modulus of 24.8 kPa

Comparing the hard-surface slam magnitudes against the shake magnitudes, they found that the slam magnitudes were high multiples of the shake magnitudes, to wit:

peak acceleration	39X
delta-V	3X
pulse duration	-53X

Shaking had a delta-V (peak-to-peak change of velocity) similar to that of a one-foot fall onto hard surface.
 Shaking had a peak acceleration similar to that of a 3-foot fall onto a bed.
 Shaking had a pulse duration greater than that of even a 1-foot fall onto a bed.

Comment by JKR: The very fact that these workers and the medical community have found it necessary to perform this research demonstrates that there is still significant scientific uncertainty about the two areas:

- (a) head injury: fall versus inflicted?
- (b) shaken: can it cause major brain trauma?

As to question (a), the present authors remark that "Evidence exists to support the hypothesis that short falls do not cause serious injury and the critical height for a fall to cause death is substantial (>10 ft). (citing references which I will give below). Simultaneously, however, others contend that relatively short falls can occasionally cause injuries associated with high mortality rates, such as SDH's, epidural hematomas, and skull fractures. (citing references which I will give below). Because the mechanical responses experienced by the head and the injury tolerances associated with shaking, shaking with impact, and falls have not yet been established, the differentiation between accidental and inflicted head injury is problematic." (pp. 1443-144)

As to question (b), the present authors remark that in their previous (1987) work, they were not able to measure head velocity directly, but had to infer it based on certain geometric assumptions (such as assuming a fixed center of rotation), and they did not study falls, and their doll was not all that biofidelic. Also, that they used scaling both there and here to predict the injury thresholds. That said, their data do not support the hypothesis that shaking can cause brain concussion. [Geddes et al., (I), 2001, above, have a comment in passing that their neuropathological findings on 53 AHT children they believe support this view. They found true DAI only in cases with very severe impact trauma. --JKR]

Injury thresholds continue to be a problem. "Regional [i.e. within the brain] tissue thresholds specific to the infant would be required to predict injury on the basis of local intracranial stresses or strains produced by the rapid rotations. Such thresholds are currently unavailable for the pediatric population." (p. 148) Therefore, "[T]he results of the dummy tests cannot be used to predict whether such rotations are sufficient to cause injury." (p. 148) [meaning rotation alone --JKR]

The authors acknowledge that scaling is a problematic approach to estimating injury thresholds, even apart from the regionality problem. ("[D]ifferences in species, age, material properties, geometry, and direction make scaling experimental angular acceleration and velocity measurements to infants problematic when based on differences in brain mass alone." (p. 149)) For one thing, there are no baseline animal or cadaver data to scale from in the case of maximum acceleration and delta-V for the more severe types of falls and for shaking and for slamming. For another thing, the simple hinge neck or the adult cadaver neck may not move the way a flexible infant neck moves, so that an error may be present in the numbers that are being scaled. And the tissue-injury threshold problem. Nonetheless, the authors scaled to brain mass from what baseline data are out there from primate and cadaver biomechanical experiments. [See the 2002 article of Ommaya, Goldsmith and Thibault (Larry) supporting scaling by brain mass, Br J Neurosurg 2002 above, which the present authors do not cite, as it may have been published after they wrote their article. But they might not have cited it anyway, since it contains no new data. --JKR] There are scalable data available for the two extremes of types of events -- minor falls, and inflicted slams. As to minor falls, the authors compare their physical data to the empirical data obtained by Weber on dropping infant cadavers and by Pincemaille et al. on boxers who did not suffer injury, and find that the low force range the present authors found for one and three-foot falls correlates well with the lack of injury found by these earlier researchers for a comparable force range. Specifically, the authors note that all their falls onto foam (bed) and their one-foot falls onto carpet pad did not scale to the level of forces needed to produce SDH and TAI as found in the primate experiments of Abel, Gennarelli et al., 1978, Gennarelli, Thibault, Adams et al.,

1982, and Margulies, Thibault, Gennarelli, 1990. For one-foot falls onto concrete and 3-foot falls onto carpet pad, the authors appraise the scaling data as inconclusive for the potential to produce SDH and TAI based on the primate work and human cadaver drop experiments. For the more severe falls (3 feet and 5 feet onto concrete, 5 feet onto carpet pad), as noted there are no specific baseline experimental biophysical data to scale to. But the authors comment that the maximum-acceleration data they obtained for these falls exceed the peak accelerations that caused intracranial bleeding in the adult cadaver drop experiment and SDH and TAI in the primate experiments for nonfall events. Therefore, the authors conclude that their data “support at least the possibility of intracranial injuries caused by these most severe falls...” (p. 148)

As for shaking and slamming, the experimental literature does not provide baseline data for acceleration and delta-V. They note that the available data from cadavers and primates indicate that slamming against foam (but not shaking) got close to but did not reach SDH/TAI thresholds scaled from primates and cadavers. “To summarize, there are no data demonstrating that the delta-V and max accel experienced during shaking and inflicted impact against foam cause SDH or TAI in infants.” (p. 148)

As to slamming against hard surfaces, again there are no baseline data for this type of event. However, the authors point out that “The majority of inflicted impacts against these hard surfaces produced a delta-V and max accel greater than the scaled rotational responses that produced fatal acute SDH in adult primates and intracranial bleeding in adult cadavers.” (p. 149) “Given this experimental data, angular velocities and accelerations measured during inflicted impacts against hard surfaces would likely produce SDH and, possibly, TAI in an infant.” (p.149)

The authors acknowledge two weaknesses of their present doll: (1) The skull lacks sutures and does not mimic the deformability of a true infant skull. (2) The neck is a simple hinge. (They do not mention it, but Carole Jenny’s soft model out in Hawaii may get around these difficulties.)

Can shaking alone cause fatal brain injury? A biomechanical assessment of the Duhaime shaken baby syndrome model. Cory CZ, Jones MD. (Cardiff School of Engineering). Med Sci Law 2003 Oct; 43(4): 317-333. This refers to the 1987 model; this does not take account of Duhaime’s 2003 work, above. These guys built a replica of the 1987 model. Owing to vagueness in the published description of that model, they had to build theirs in an adjustable way, to allow for various parameters that might be consistent with the published description and which might affect the angular acceleration of the head. For example, they constructed three different necks -- one hinge and two rubber. With such adjustments, the authors were able to get the angular acceleration up to **9,000+ sec(-2)**, which is within Duhaime’s predicted boundary condition for concussion (Head Injury Criterion, or HIC). Additionally, they say, review of the literature indicates that Duhaime et al may have used an incorrect HIC. These guys followed predicted tolerance limits proposed by Sturtz, 1980. Duhaime’s scaling limits required 10,000 for concussion (or 8,000 if you use scaled values from Thibault & Margulies, 1988), and 6,000 for SDH. But Ommaya’s work quoted in Klinich et al., 1996 gives a 50% probability of concussion at 4,000. (This was from a model used in studying pediatric auto vs. pedestrian accidents.) See figure 2 and table III. The authors discuss the problems with scaling.

“At this present stage the authors conclude that **it cannot be categorically stated, from a biomechanical perspective, that pure shaking cannot cause fatal head injuries in an infant.**” Parameters require further investigation to increase the biofidelity.

Traumatic axonal injury is exacerbated following repetitive closed head injury in the neonatal pig. Raghupathi R, Mehr MF, Helfaer MA, Margulies S. J Neurotrauma 2004; 21: 307-316. See below under “Biomechanics.” Cited by Hymel (2005) as having “subjected piglets to either single (n=5) or double (n=6) nonimpact axial rotations of the head and discovered significantly *more foci* of injured axons following *two* rotational loads.” This work was a followon to their 2002 piglet experiments which found that rotation caused SAH and TAI similar to that seen in SBS.

Animal models of shaken baby syndrome: revisiting the pathophysiology of this devastating injury. Bonnier C, Mesple B, Gressens P. (Brussels) Pediatric Rehabilitation 2004 Jul-Sep; 7(3): 165-171. Shook mice. 75 % of survivors had focal brain lesions consisting of hemorrhagic or cystic lesions of the white matter, corpus callosum and cerebellum. All showed late white matter atrophy.

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. In this chapter, Dr Starling uses the term “shaking” as her term for abusive head trauma. She summarizes the shaking debate as follows [with some emendations by me --JKR]:

Critics of Duhaime’s proposal that impact is necessary make the following arguments:

1. Duhaime’s use of primate data did not take account of immaturity features of the infant.
2. Duhaime’s model only measured peak acceleration.
3. Duhaime’s model did not address repetitive shaking.
4. “Models are only beginning to capture the characteristics of the immature brain and skull [to say nothing of neck; see Zimmerman’s discussion in *Inflicted Childhood Neurotrauma* symposium, 2003, above under “Cervical spine and neck,” discussing the papers of Hadley et al. and Geddes et al., who found trauma of the cervicomedullary junction. --JKR], and none have addressed the differences between a single blow or whiplash and multiple shakes, nor do models address the role of secondary injury in the neurological outcome.” p. 41
5. Six of Duhaime’s 13 infant autopsies did not show evidence of impact, and others, for example Gilliland, have shown cases with no impact injuries. Citing Hadley et al., 1989, Alexander, Sato et al., 1990, Gilliland & Folberg, 1996, and Pounder’s shaken adult, 1997.
6. Dr Starling’s point about secondary injury and its role in the neurological outcome deserves to be enumerated separately. Severe secondary injury seems to be much more a feature of abusive head trauma than of accidental head and neck trauma. Therefore this by itself becomes a diagnostic feature of AHT. (Obviously, severe secondary injury may be caused by delayed rescue, as in the case example given on p. 42 by Dr Starling). But more than that: severe secondary injury is a feature of shaking, that is, cases with a shaking history and cases without evidence of impact. -- JKR

The evidence base for shaken baby syndrome. Response to editorial from 106 doctors. Reece RM. BMJ 2004 May; 328: 1316-1317. As to biomechanics, the research is still being done. It is “premature” to conclude from it that shaking cannot cause infant brain injury. And note all those confessions.
Comment by JKR: This last item, on biomechanics, is interesting. Note that the 106 doctors did not claim that biomechanically concluding that shaking cannot cause (primary) brain injury is impossible. They only said it is “premature.” They seem to be admitting that the biomechanical research is pointing more and more in that direction, and that it’s only a matter of time.

Animal models of shaken baby syndrome: revisiting the pathophysiology of this devastating injury. Bonnier C, Mesple B, Gressens P. (Brussels) *Pediatric Rehabilitation* 2004 Jul-Sep; 7(3): 165-171. Shook mice. 75 % of survivors had focal brain lesions consisting of hemorrhagic or cystic lesions of the white matter, corpus callosum and cerebellum. All showed late white matter atrophy.

Shaken baby syndrome: theoretical and evidential controversies. Minns RA. *Journal of the Royal College of Physicians of Edinburgh* 2005; 35: 5-15. This well-written article reviews the published literature from medicine and biomechanics and basically concludes as follows:

1. Shaking can injure the infant brain.
2. Clinicians cannot diagnose the shaking mechanism, but they can diagnose NAHI.
 - a. The clinical diagnosis of NAHI, however, comes with varying degrees of certainty.
 - b. The term “shaken baby syndrome” is best avoided, because it implies diagnosis of a specific mechanism which cannot be clinically diagnosed.
3. Short falls can give a clinical picture similar to NAHI but this is rare.

As to topic 1, whether shaking can injure the infant brain, the author states that there are a significant number of published pure-shaking cases to provide us with a clinical database for study of this question. About 33% of the published cases are shaking-only cases, he states. [Compare this statement with the statements of Leestma, above. –JKR] Thus about 1/3 of published non-accidental SDH's have no evidence of impact. "This by itself is strong evidence in favor of the syndrome." Indeed, he points out that even one well-documented case of SDH from shaking would prove that the syndrome exists. And he gives such a case, apparently his own patient, with a confession that the child was "shaken angrily in the air" without impacting on any surface. [This patient survived and hence was not an autopsy case -- a weakness of this item of evidence adduced. –JKR] He then discusses more theoretical approaches to the question, under five heads:

- predisposing anatomy
- animal models
- computer models
- biomechanical models
- neuroimaging

As to predisposing anatomy, the author cites five factors that predispose infants to suffer brain injury and SDH from shaking. 1. large head. 2. weak neck. 3. large extracerebral space. 4. laxity of the pachymeninges. 5. high water content of the brain.

As to animal models, the author briefly reviews the articles of Ommaya, Faas & Yarnell (1968) demonstrating concussion and surface bleeding in rhesus with pure impulsive loading, Genarrelli & Thibault (1982) with the helmet showing fatal SDH from angular acceleration of 100-3,000 g, but noting that the acceleration magnitudes were probably greater than could be induced by manual shaking, Duhaime et al. (1987) finding that shaking frequency was 4-10 Hz, Ommaya (1993) and others considering that there may be a natural resonant frequency for the infant head-neck-body system, since primates have a natural resonant frequency of 5-10 Hz. Mechanical properties of animal brain tissue have been studied by Thibault & Margulies (1998) and Prange & Margulies (2002), and have found that both mature and immature porcine brain tissue is nonlinear in its response to strain, and thus is "viscoelastic", and the immature porcine tissue was less stiff than adult tissue at the lower levels of strain, suggesting that infant brain might have a lower threshold for injury. Also Raghupathi & Margulies (2002) finding that immature unmyelinated axons may be more susceptible to deformation than adult axons, again suggesting a lower threshold for axonal injury in infants.

As to computer models, people have used these to study bridging vein loading and CSF buoyancy/ tentorial protection of the bridging veins against translational and rotational acceleration. Zhou et al. (1996) computer-modelled angular acceleration of a simulated human head whose biomechanical properties were scaled from [adult] animal data, and deduced peak angular accelerations and decelerations of 7,000 sec(-2) and 9,000 sec(-2) respectively, leading to bridging vein stretch ratios [strain?] of 1:383 during sagittal rotation. [The stretch ratio apparently correlates with the likelihood of vein rupture.] Morison (PhD thesis, 2002) deduced that the bridging vein stretch ratio is highly sensitive to shaking frequency, especially in the frequency range of 2 to 5 Hz, and that the average bridging vein will rupture at stretch ratio of 1:5, while some veins may fail at ratios of as little as 1:15. Shaking at 4 Hz can produce a stretch ratio of 1:26. "Therefore, this model shows that SDH may well be a possible result of manually shaking a baby." As to CSF buoyancy/ tentorial protection of the bridging veins, Morison's work showed that these are highly protective against translational acceleration, but not against rotational. And in shaking, 93% of the strain on bridging veins comes from rotation, not translation. "Rotational accelerations are therefore extremely dangerous to humans and this has been well acknowledged since Holbourne, the primate experiments of Ommaya and Genarelli, and the observations of woodpeckers by May et al. This current model gives support to the hypotheses of Hodgson et al that the CSF protects against translational acceleration perfectly but effectively acts as a lubricant to brain rotation." One other thing: The bridging veins slant forward toward the falx. Therefore, the most harmful point in a shaking motion is the posterior extremum, when the brain moves backward in the skull, placing the most stretch on the bridging veins.

As to biomechanical models (dolls), the author says it is difficult to construct reliable models because of the absence of infant biophysical data, with the resulting need to extrapolate from animal data. He reviews the generally held view that primary brain injuries are focal or diffuse. Focal injuries may be contact or inertial, and diffuse injuries are rotational. The types of rotational diffuse injuries are:

- SDH and SAH
- sphenoid impact
- gliding contusions
- TAI
 - lacerations at the gray-white interface
 - corpus callosum tears
 - cerebellar peduncle tears
- craniocervical junction injury
- diffuse RH

[I have reorganized the author's categorization somewhat to conform to US usage. –JKR]

The author then mentions the secondary brain injuries of hypoxic/ reperfusion injuries, cerebral edema, seizures, infarctions, herniation, with resulting systemic hypotension, hypoxemia, and pyrexia. He then goes on to discuss the doll models. Duhaime (1987): Impact produced peak accelerations up to 50 times that of shaking. Scaling from animals predicted that the shaking accelerations would not be enough to produce injury. "They deduced that while there may be no visible soft tissue injury with impact against a soft surface, there can be dramatic pathological loads experienced by the brain." Then his own experiments done on a doll in Edinburgh. The doll was intended to model a two month-old human infant. They found that an adult can maximally shake such a doll for an average time of 24 seconds, with an average frequency of 3.5 Hz, reaching an average peak linear acceleration of 4,200 cm sec⁻². [Doesn't give angular acceleration here; the actual report is in press. –JKR] Shaking the doll, they found that the peak angular accelerations were 326 to 890 sec⁻² (4 to 15 g) when shaken by adult men. Their peak values were **less** than the average values obtained by Duhaime. In another study, de San Lazaro found peak accelerations of 7 g for violent shaking. What can be concluded from all these dolls? Apparently not much. The main problem being the invalidity of scaling from animal data.

Rigid-body modelling of shaken baby syndrome. Wolfson DR, McNally DS, Clifford MS, Vloeberghs M. *Journal of Engineering Medicine* 2005; 219(H): 63-70. (Nottingham) Reviewed by Kent Hymel in the *Quarterly* for Autumn 2005. According to Dr Hymel's review, these experiments were directed toward neck stiffness, and failed to achieve the acceleration thresholds for brain injury. Dr Hymel comments, "Just because we can't yet model SBS does not mean shaking is benign."

Shaken baby syndrome: a biomechanics analysis of injury mechanisms. Faris A. Bandak. FSI 2005 Jun; 151(1): 71-79. In this article, Prof Bandak looks at the tensile strength of the infant neck as available from four articles in the literature, and then calculates the probable effect of violent shaking on that neck, which he concludes is going to be either a neck fracture or a decapitation. In the Winter 2006 *Quarterly*, Betty Spivack critiques this by saying that all of the four studies cited by Bandak involved static ("quasistatic") loading in which infant human or animal necks were stretched by suspended weights. In contrast, shaking involves dynamic loading. Biologic tissues, she says, are viscoelastic. They are much more resistant to sudden dynamic loading than to slow stretch loading. Therefore, she says, the rupture thresholds derived from static loading are completely inapplicable.

Mechanics of acute subdural hematomas resulting from bridging vein rupture. DePreitere B, Van Lierde C, Vender Sloten J et al. (Belgique) *Neurosurgery* 2006; 104: 950-956. These guys whanged the backs of the heads of ten cadavers of older adults with a big padded steel hammer, gradually increasing the velocity and the pulse time (by varying the amount of padding). Head fixed with accelerometers. Performed 18 whacks. Got six SDH equivalents (extravasation of radioopaque dye from the superior sagittal sinus. Steve Boos in the Winter 2007 *Quarterly* reports that bridging vein rupture occurred at between 5,267 and 13,411 radians per second acceleration, and pulse durations of between 5.2 and 15.4 milliseconds. He reports that "There was a trend for decreasing acceleration threshold as pulse duration increased. There was also a trend for decreased threshold on second impacts." Steve makes the very useful interpretive

comment comparing this work with the primate studies. “The results suggest that the peak acceleration threshold for subdural hematoma at long-duration accelerations (those more closely resembling shaking) [is] about half that of the threshold at shorter [pulse] durations.” He also takes note of the apparent lowering of the threshold in those cadavers that were hit twice. Noting that shaking involves repetitive accelerations. He notes that the parameters measured here were different from the primate studies, namely

here

peak acceleration
pulse duration

primate studies

peak acceleration
peak change in velocity

But these differences could be converted numerically. [I note that the doll studies do include pulse duration. –JKR] His overall assessment is that “[T]his study provides a toehold supporting arguments that shaking without impact is biomechanically consistent with subdural hemorrhage. More firm footing will require much more work...”

HEAD INJURY -- FALL VS. INFLICTED

See also “Shaken” and “RH” and “Walkers” and “Bunk beds” and “time of injury – head.”

Reece & Ludwig, 2d edition (2001, pp. 62-68; 193-195): The first discussion is by Randy Alexander, Carol Levitt, and Wilbur Smith. They say that sorting out accidental from abusive head trauma is a “troublesome dilemma” for clinicians, particularly when the injuries are less severe. However, falls are “an infrequent cause of death.” “[S]evere head injuries purported to be accidental, unless related to a moving vehicle accident, are very likely to be the result of abuse, particularly if injuries are ascribed to falls from short heights. Difficulty remains in sorting out accidental from abusive injury when the head injuries are less severe.” The literature is confused, but contains 1,732 short falls, 1,037 stairway falls. As to short falls, they cite

- Kravitz, 1969 (330 falls, 105 neurol sx, 1 SDH)
- Helfer, 1977 (246 children falling off of beds or sofas, no CNS injuries)
- Bilmire & Myers, 1985 (84 infant head admissions w CT: no ICH or RH from falls)
- Nimityongsukul, 1987 (76 children fell oob, no CNS injuries but arguing that a tiled floor could be more dangerous)
- Greenes & Schutzman, 1988 (14 infants **with** intracranial injuries from short falls)
- Selbst, 1990 (68 bunk beds, 1 skull fx w SDH)
- Chadwick, 1991 (65 falls of 5-9 feet no deaths; 10 alleged falls of 4-9 feet, 7 deaths fr SDH)
- Williams, 1991 (44 witnessed falls under 10 feet no life-threatening; 53 unwitnessed falls under 10 feet, 2 deaths)
- Lyons & Oates, 1993 (124 or 200 falls from cribs & beds, no life-threatening)
- Levitt & McCormick, (336 uncorroborated falls from less than 8 feet, 9 SDH, of which 7 were later determined to be abusive)
- Tarantino, 1999 167 infants under 10 months fell less than 4 feet by history, excluding acceleration injuries. 12 skull fxx, no ICH. Two with ICH turned out to be false histories of fall. An infant who is dropped appears to be at greater risk (of significant head injury) than one who just falls.
- Mayr, 1994 (103 falls from high chairs, no ICH)
- Joffe & Ludwig, 1988 (363 stairway falls. 4/10 who fell with adult suffered skull fxx. 24 walkers had head injuries.)
- Chiaviello, 1994 (Stairways: 3 fell in the arms of their caretakers 2 got SDH's.)
- Chiaviello, 1994 (46 walker stairs: 1 fatal SDH, 4 other nonfatal ICH)
- Levitt & McCormick (ongoing study) (30 infants w accidental intracranial injuries: 9 were

walker-stairs. These 9 incl 3 SDH, 3 large intracerebral hematomas, 2 small brain contusions, 1 SAH)

Smith, 1997 (260 walkers: 10 skull fxx incl 3 depressed with ICH)

Mayr, 1994 (143 walkers: 19 skull fxx, no ICH)

Gruskin & Schutzman, 1999 (236 falls, 2 SDH, 4 EDH, 3 contus, 1 SAH, 2 cerebral edema.

“They do describe **four infants** of 72 infants younger than 12 months who had intracranial abnormalities from falls 3 feet or less.”) (Are these the same infants as Schutzman’s 1988 infants?)

Reece & Sege, 2000 (10% of acc had SDH vs. 46% of AHT)

“...support the conclusion that ... **severe intracranial injuries ascribed to short falls likely indicate abusive injury.**” The authors give a full-page chart summarizing the above articles and others.

The second discussion is by Jan Bays. She says that significant intracranial injury with a history of a fall presents clinicians with “a dilemma.” Citing Hall, 1989 for the proposition that falls are the third leading cause of traumatic death in children 1 to 4 years old. [Cf. Ommaya’s 2002 assertion of the same proposition, below, and Cindy Christian and Tina Duhaime’s negative comment thereon. Note also Dr Kirschner’s negative comments on the Hall article, below, as “a defective study.” –JKR] Yet, AHT is the most common cause of intracranial injury in those under 1 year old. Without drawing some sort of magic boundary at one year of age, “how does the clinician sort out accidental from nonaccidental causes?” First she separates simple gravitational falls from falls involving [rotational?] acceleration forces. Then she reviews the articles by Kravitz, Helfer, Billmire & Myers, Lyons & Oates, Williams, Chadwick, Tarantino, and Reece & Sege. She concludes that “In summary, a history of an accidental fall of less than 5 feet is plausible if it results in **either no injury, soft tissue injury with no neurologic sequelae, or, rarely, if the infant impacts an edge, in a linear parietal skull fracture with no subdural bleeding and no CNS damage.**”

Discusses intracranial hemorrhage, including spontaneous, as a separate category. See also under “Terson’s syndrome.”

Knight’s Forensic Pathology, Third Edition (2004). Discusses skull fractures on pp. 464-466 and intracranial injury on pp. 469-471. As to intracranial injury, the authors look at Howard, Bell & Utley (1993), who studied 28 cases of SDH in infants under 18 months all of whom fell short distances or from own height. 47% had a skull fracture and an SDH. They refer to Aoki’s work finding SDH after falls from a sitting position. State that “there is considerable controversy in the literature” as to the distance of a fall needed to cause head injury. There is no doubt, citing Weber, that skull fractures can occur from low falls. But skull fracture is usually not accompanied by any brain injury. “The evidence that low falls may cause brain or meningeal lesions is much less convincing than that proving skull fractures -- but the possibility exists and cannot be dismissed by inflexible, dogmatic opinion.” [because biomechanical experimentation on infants is impossible.] As to biomechanics, says that subdural bleeding likely depends on the **strain rate** rather than the absolute deceleration. Strain rate is the rate of change of deceleration and the duration of deceleration. “A head impacting on an immovable surface after a fall is an example of a high strain-rate injury...” [while shaking is a low strain-rate injury]. “Subdural bleeding from bridging veins is likely to occur from high strain-rate injury, even though this may be a low-energy injury, insufficient to cause cerebral tissue disruption. By contrast, low strain-rate injuries are more likely to cause cerebral contusion, with vascular damage only if concomitant high energy is generated.” (p. 470) [They seem to be referring to Gennarelli, Thibault, 1982, below. –JKR]

Accidental falls from elevated surfaces in infants from birth to one year of age. Kravitz H, Driessen G, Gomberg R, Korach A. Pediatrics 1969; 44(suppl): 869-876. This is one of the only studies of this type that focuses specifically on infants; see also Billmire & Myers, 1985. Reports two studies performed by these pediatricians in their clinics. (1) A retrospective questionnaire survey of the parents of 200 clinic patients who had a history of a fall. These were mostly lower-class families in an urban public clinic. (2) A prospective questionnaire survey of the parents of 336 upper-class children in a suburban private practice who had a history of a fall. Total 536 infants. The two groups were compared. The urban group had more instances of multiple falls. Many of the infants did not have any injuries. As far as injured ones, first falls resulted in 154 injured infants out of 200 in the clinic group (an injury rate of 77%), and 101 injured infants

out of 336 in the private group (an injury rate of 30%.) As to injury from first falls, Table IV shows that as to skull fractures the private group had 2/101 for a 2% fracture rate while the clinic group had 1 fx for an 0.6% fracture rate. (note that these are peculiar denominators: they use only the subgroup of infants who had an injury, not the whole group who fell.) As to SDH, the private group had 1 for a 0.3% rate and the clinic group had 0. Total 3 out of 536 had skull fractures for an overall fracture rate of 0.5%, 1 out of 536 had SDH for **an SDH rate of 0.2%**. No fatalities.

Head injury due to falls from heights. Cummins BH. Injury 1970; 2: 61-64. Average fall distance of children was 19 feet. 62% of those under 2 years old had a skull fracture. No LOC. One EDH, no SDH.

The significance of skull fractures in children. Hardwood-Nash et al. Radiology 1971; 101: 151

Cerebral concussion and traumatic unconsciousness: correlation of experimental and clinical observations on blunt head injuries. Ommaya AK, Gennarelli TA. Brain 1974; 97: 633-654. See under "Shaken."

Acute SDH in infancy and childhood. Gutierrez FA, Raimondi AJ. Child's Brain 1975; 1: 269-290. Quoted by Wilkins (Head injury -- abuse or accident, 1997). A series of infants and toddlers with SDH where both abuse and falls were identified as causes.

Injuries in children sustained in free falls: an analysis of 66 cases. Smith MD, Burrington JD, Woolf AD. J Trauma 1975; 15: 987-991. Long falls. Had to fall four stories (30 ft) to get fatal. 34 fell less than one story. 32 fell one to eight stories. Overall had 10 skull fractures, 32 extremity fractures, 0 rib fxx, several visceral injuries all over two stories. 2 deaths, both fell more than four stories. [But I have personally autopsied toddlers who fell two stories and were killed. -JKR]

Injuries resulting when small children fall out of bed. Helfer RE, Slovis TL, Black M. Pediatrics 1977; 60: 533-535. Parental questionnaires and hospital incident reports of 246 ch under 5 who fell out of bed. Of these, 85 fell 36 inches or less. Of these 85: 57 no inj, 17 cuts & scratches, 20 bruise, 1 skull fx w no seq. Other breakdown: 161 fell at home: of these there were 2 skull fxx, 3 clavicle, 1 humerus, 0 rib fxx. 85 fell in the hospital: of these, 1 skull fx. **No deaths, no serious sequelae.** The AAP's abstract ("Guide to References...") notes that of all the at-home falls regardless of height, the parents reported that 80% had no observable injuries; of the in-hospital falls, 67% had no injury. The AAP's abstract states, "The authors conclude that physicians should be very suspicious when serious injuries are said to have been caused by a fall from a bed, sofa, or crib." A possible limitation of this study would be the older average age of the subjects, as opposed to infants. This article is referred to by Zumwalt & Hirsch in chapter 13 of Helfer & Kempe's *The Battered Child*, 4th ed. (1987), where these authors note that the head injuries reported in the article are much less extensive than the massive skull fracture demonstrated by Zumwalt & Hirsch as having been caused by a six month old being dropped by a sibling.

Children can't fly: a program to prevent childhood morbidity and mortality from window falls. Spiegel CN and Lindaman FC. Am J Publ H 1977; 87: 1143. In Los Angeles, there is no law requiring screens or any type of childproofing of upper-story windows.

Snyder et al., 1977. 100 falls of chil and adults. Had to fall 15 feet to get life-thr inj

Unsafe playgrounds. Reichelderfer et al. Pediatrics 1979; 64: 962-963. Argued for the use of pads on playgrounds by presenting the biomechanics of falls onto different surfaces. A 3-inch fall onto concrete gives 150 g's, while an eight foot fall onto wood chips gives only 35 g's. 50 g's is the injury threshold for the head.

Dr Hirsch's earlier (1980) work arguing that in loss-of-balance falls the center of gravity becomes displaced relative to the feet, and it becomes a rotational fall for purposes of the head. (Dawson, Hirsch et al., HumP 1980; 2: 155)

Cerebral injury in child abuse: a changing profile. McClelland et al. Child's Brain 1980; 7: 225.

Biomechanics of acute subdural hematoma. Gennarelli TA and Thibault LE. J Trauma 1982 Aug; 22(8): 680-686. States that there are basically three mechanisms of an acute SDH: (a) fracture-SDH, (b) contusion-SDH, where blood enters the subdural space from a large brain contusion, and (c) tear of bridging veins. They studied type (c) which is the most common. Studied CT data on 38 hospital patients of all ages who suffered acute SDH that was not fracture-related or contusion-related. Also performed experiments on 128 Rhesus monkeys fitted into a tight-fitting accelerometer helmet that distributed the loading all over the head and prevented localized impact. The center of rotation was in the lower cervical area. It appears that the rotation of the head was in the sagittal plane. Three parameters were independently varied: (a) acceleration, (b) time-duration of the application of the acceleration, also called the "impulse duration," and (c) rise-time to peak acceleration. This latter parameter corresponds to the "strain rate" being applied to the bridging veins. They specifically designed their test bed to be able to vary the rise-time so that they could vary the strain rate (rate of increase of strain) of the bridging veins. Strain rate is the rate of increase of strain, where strain is $(L_f - L_o) / L_o$, L_o being resting length and L_f the stretched length of the bridging vein. Their real focus was to study the accelerative parameters needed to break the bridging veins. Strain rate corresponds to the rise-time of the angular acceleration, which, as noted, they were able to vary independently with their helmet. They argue that the behavior of bridging veins is highly viscoelastic, which means that whether they will break depends on how abruptly they are stretched (the strain rate,) as well as how far they are stretched (the strain.) They show that this viscoelastic model of a bridging vein correlates well with theoretical predictions and their observations for creation of SDH. Production of SDH had a threshold rise-time when peak angular acceleration was controlled, or in other words, when peak angular acceleration was plotted against the reciprocal of the rise-time (a directly controllable stand-in for the strain rate,) the result was that SDH only occurred outside of an inverse straight line (Fig. 3). Thus, production of SDH required the combination of either low peak acceleration with very fast rise-time, or high peak acceleration with slower rise-times. Thus, "the viscoelastic behavior of the bridging veins precludes a single ultimate strain or ultimate stress criterion for failure." This is apparently to distinguish bridging veins from the monotonic models that were used by earlier workers to estimate concussion thresholds. They want testing of fresh vein specimens in order to establish the precise location and shape of the tolerance curve.

Discussion: "This study demonstrates that ASDH due to ruptured bridging veins occurs because of head acceleration that is associated with rapid rates of acceleration onset (i.e., high strain rate.)" They correlate this fact with various real-world observations. One is the fact that impact is not necessary to produce this type of SDH: take boxers, football players whose heads are snapped, and shaken babies (posited as a specific example; but note that this statement predates their and Duhaime's later work done in 1987.) Another is the fact that bridging-vein SDH's usually happen with moving head impact against a hard surface such as falls and assaults, involving impact against a hard surface with very fast rise-time, and seldom in MVA's where the surfaces are padded or breakable/ bendable. (They point out, though, that while such surfaces protect against SDH, they may actually make DAI more likely.)

Acceleration was varied from 100 to 3,000 g, also measured as 100,000 to 300,000 sec^{-2} , impulse duration was varied from 2 to 12 msec, and rise-time was varied from 0.1 to 6 msec. Fig. 1 shows that the angular acceleration threshold for SDH was about 1×10^5 radians per second squared, and the pulse duration threshold was about 3.5 msec; all the SDH's were at angular accelerations greater than about 1×10^5 and pulse durations longer than 3.5 msec. These data have been used in a controversial way to argue that short falls can give SDH if the stopping time is right.

Ten years' experience of falls from a height in children. Barlow B, Niemierska M, Ghandi RP, Leblanc W. J Pediatr Surg 1983; 18: 509-511. 61 falls. 23% were killed. 3 floors = 0 mortality. 4-6 floors = 50% mortality. 4 of the survivors had rib fxx. In the October 98 issue of Child Abuse Quarterly, Betty Spivack compares this paper favorably to the later effort by Lau et al (FSI 1998, see below).

Experimentelle Untersuchungen zu Schadelbruchverletzungen des Sauglings. Weber W. Zeitschrift fur Rechtsmedizin 1984; 92: 87-94. He dropped 15 dead babies aged from newborn up to 8 1/2 months from a height of 82 cm (about 32 inches) onto three different floors (tile, carpet over tile, linoleum over tile). All 15 received linear skull fractures. One had bilateral fractures. Three had fractures crossing a suture. See also his papers from 1985 and 1987.

Bleeding disorders and non-accidental injury. O'Hare AE, Eden OB. Arch Dis Child 1984; 59: 860-864. Discusses spontaneous SDH.

Skull fracture and the diagnosis of abuse. Hobbs. Arch Dis Child 1984; 59: 246

Infantile acute subdural hematoma: clinical analysis of 26 cases. Aoki N and Masuzawa H. J Neurosurg 1984; 61: 273-280. By the term "Infantile Acute SDH," (IASDH) as used in Japan, the term means "accidental," i.e. noninflicted. All 26 had RH. Hence Rekate in a letter (J Neuros 62: 216-217) said that these authors might have missed the dx of SBS in some of these cases, because of the lack of expected external bruising. In the next article below, which is Aoki's reply to that letter, Aoki comes back and argues that SDH + RH could just as well be accidental, it is not diagnostic of abuse. RH is not detailed. Levin (2000a) comments that this article "deserves special mention since it reports findings that are in complete disagreement with all other literature on the subject. Aoki and Masuzawa report 26 children, 3 – 13 months old, **who sustained acute SDH following minor falls**, most often simply falling backwards from a sitting or standing position onto a soft tatami mat... All 26 pts had 'retinal haemorrhage and dark red semicircular preretinal haemorrhage around the optic nerve heads.' Two photographs centred on the optic nerve heads are provided which show extensive dot, blot and flame haemorrhages in one eye with a large preretinal haemorrhage in the other, There is no papilloedema, These findings are unlike any ever reported int the literature or observed by me in an accidental head trauma victim and would otherwise suggest that the babies were shaken. In fact, there are many concerns about this paper which suggest that this may indeed be the case. The authors do not state if complete investigations were done and, to my knowledge, there were no child abuse teams formed at that time in Japan. In addition, some of the ch had findings on angiography suggestive of infarction, a finding which wd be inconsistent with such mild accidental trauma..." and goes on to say that Japan was way behind the West in recognition of child abuse, concluding that "it is very likely that the children reported by Aoki and Masuzawa represent missed cases of SBS rather than accidental head injuries." (Levin 2000a at 181-182.) See 1986 article below.

Subdural hematomas in infants. (letter) Rekate HL. J Neuros 1985; 62: 216-217. Arguing that Aoki & Masuzawa might have missed a number of SBS cases in their 1984 work, above. That accepting a parental history of a short fall in infants or toddlers with SDH + RH fails to consider the entity of SBS, and that a child abuse workup was not done in these cases. Answered by the authors with 1986 paper, below, where the authors essentially deny the existence of SBS, at least in Japan. Answered by Rekate et al. 1989 autopsy study of five shakens showing no impact injury. (Neurosurgery 1989; 24: 536).

[On the biomechanical fragility of the infant skull.] Weber W. Zeitschrift fur Rechtsmedizin 1985; 94: 93-101. (Translation made by John Hunsaker, MD of the Kentucky Justice Cabinet.) Continuing the 1984 work, they have now dropped 35 additional babies, again from 82 cm. This time they dropped them onto softly cushioned surfaces, and obtained 5 parietal fractures. Concludes that the parietal bone is a zone of fragility because it has no diploe. See also the 1987 paper. [Knight states that "the diploe of infant skulls are absent or only partially formed, according to age." Saukko & Knight, *Knight's Forensic Pathology*, 3d edition (2004), p. 466. --JKR]

Falls in children and youth. Garretson LK and Gallagher SS. Peds Clin N Amer 1985 Feb; 32(1): 153-162. (have) A review of epidemiology and prevention. In childhood, falls were the fourth leading cause of traumatic death, behind MVA, fires, and drowning. Falls were the most frequent cause of injury bringing children to the emergency room. Falls in childhood are class-correlated (Kravitz, acc falls from elevated surfaces in infants, 1969; Spiegel, Children can't fly, 1977) and urban. Falls were reverse race-correlated -- higher risk in whites. What children fall from: in cases of head injury in order of freq:

stairs	42%
furn	28%
bed or changing table	18%
tree	4%
bike	3%
window	3%

ladder	1%
infant seat	1%
roof	-%

In toddlers, “injuries from stairs and steps predominate, while beds, tables, and chairs are also common injury vehicles.” Contact surface is important. Skull fractures are common in falls, especially in infants. (Boulis, 1978).

Serious head injury in infants: accident or abuse? Billmire ME and Myers PA. Pediatrics 1985; 75: 340-342. Studied 84 infants 0-1 yr admitted to U Cincinatti with head trauma requiring a CT scan. Ages 3 weeks to 11 mos, avg 4.6 mos. 54 were acc, 30 AHT. 40/54 acc had only a linear skull fx w no seq. When uncomplicated skull fractures are excluded, 95% of all the serious head injuries to infants were AHT. The one accidental case was an unrestrained passenger in a car collision. That one was the only acc that had intracranial bleed, while 11 abuses had intracranial bleeds. No RH seen in non-abuse. Need to check what *kind* of intracranial bleeds these had.

Subdural hematomas in abused children: report of six cases from Japan. Aoki N and Masuzawa H. Neurosurgery 1986 Apr; 18(4): 475-477. The purpose of this work was to answer American critics (Rekate et al., 1985) who argued that these authors’ earlier article (above) could have misdiagnosed cases of SBS as accidental IASDH, particularly since RH was present in all of them. Here the authors present six cases of AHT, of which five have head or face bruises and the other one had abdomen bruises. State that in Japan, all abused children have bruises, as in these cases. Suggest that the same is probably true in the US, and argues that US physicians need to reevaluate their tendency to diagnose AHT based solely on SDH + RH when there are no other injuries. A history of a short fall leading to an SDH with RH is not *per se* suspect. “These findings of bruises and skull fractures suggest a different mode of trauma from that causing IASDH, for which trivial head injury, such as falling down while sitting or standing, is exclusively responsible.” The authors impliedly cast doubt on the Anglo-American theory of whiplash (citing Guthkelch); but how would they explain case 1 which had no head bruises and no history of a fall? [In this study, one pt had a history of a fall, and in that case the history was obviously spurious.] In this study, 2 had RH, 3 did not, 1 was not examined for RH. RH not detailed. All but one of these six abuse cases are toddlers. “In conclusion, RH and SDH without external signs of trauma in Japan is usually attributed to accidental, trivial head injury (IASDH;) in the United States, it is usually attributed to child abuse. It is important to be aware of these tendencies when investigating the cause of SDH in infants.” Case 1 age 1 yr 11 mos presented comatose after becoming drowsy and suffering a grand mal at home. Exam showed no bruises on the head or face. Had multiple bruises on the abd region. Had anisocoria / larger R pupil. + R RH. Craniotomy evac 30 gm clotted R SDH. Pt recovered with moderate L hemiparesis. Case 2 a 3 y o emaciated boy presented obtunded with fever and grand mal. Bruises on face and head. +RH. Sutural diastasis. Bilat SDH. Brown CSF. Treated nonoperatively, remained tetraplegic. Case 3 a 1 yr 9 mos boy history of falling off a table, had a sz several hours later. Kept at home in a comatose state for one month. No skull fx. CT sh high-density convexity SDH w interh. Observed. Remained comatose. Case 4 an emaciated 3 yo boy w hx of battering, LOC. DV. Multiple burns. No RH. No fx. Bilat SDH. Resolved without operation. Case 5 an infant age 3 mos w multiple bruises of the head and face, LOC, presenting w spasticity & a tense fontanelle. No RH. Linear fx R frontal. Bilat acute & chronic SDH’s + interh. Survived w drainage. Case 6 a 3 y o girl with a R forehead bruise, coma, anisocoria, large R pupil, no skull fx, CT acute R SDH, resolved nonoperatively and recovered completely w conservative man. No RH. According to Sunderland (see Wilkins, 1997), this controversy “has also now involved British neurosurgeons,” citing Howard, Bell & Uttley, 1993.

Incidence, severity, and external causes of pediatric brain injury. Kraus JF, Fife D, Cox P, Ramstein K, Controy C. Am J Dis Chil 1986 Jul; 140: 687-693. Epidemiologic study of hospital and coroner’s records of pediatric brain injury up to age 15 from San Diego County for 1981. Causes: falls 35%, recreation 29%, MVA 24%. Under falls, fig 2 gives falls from a height 60%, same-height 10%, unk height 30%. The brain injury rate per hundred thousand children was 100 for chil 0-4 (the highest age group). In the less-than-1 age group, from all mechanisms, 75% were mild, 12% mod, 13% severe or fatal. In infants, assaults were more likely to cause severe brain injury (56% severe) than all mechanisms together (24% severe.) Bicycles were frequently involved in older children.

The likelihood of injuries when children fall out of bed. Nimityongskul P and Anderson L. J Ped Orthop 1987; 7: 184-186. 76 pediatric falls out of bed in hospitals. 1 skull fracture without sequelae, 1 tibia fx, 0 rib fxx, 14 scalp and face hematomas. No deaths, no serious sequelae. Concluded that serious injuries are extremely rare when chil fall OOB.

Falls in urban children. Meller JL. Am J Dis Chil 1987; 141: 1271-1275. Retrospectively reviewed 48 admissions for fall from a height. Had a number of deaths.

[Predilection sites of infantile skull fractures following blunt force.] Weber W. Zeitschrift fur Rechtsmedizin 1987; 98(2): 81-93. Translated by John Hunsaker, MD of the Kentucky Justice Cabinet. Weber has now dropped 80 dead infants from newborn up to 14 months and two toddlers from a height of 82 cm onto hard and padded surfaces, obtaining an unspecified number of fractures (*voire* 1984, 1985 ff.). He posits that the thin areas, primarily of the parietal bones, without diploe, are vulnerable. He repeats that these fractures are entirely consistent with the mechanism of falling off a changing table.

Stairway injuries to children Joffe M and Ludwig S, Pediatrics 1988 Sep; 82 (No. 3 pt 2): 457-61. 363 falls down stairs in children 0 - 11 yrs old. No life-threatening injuries, no ICU admissions. 22 children had skull fxx (all under age 2 yrs); most of these were in the arms of a caretaker who also fell down the stairs. Overall, 7% had fxx, 1% had concussions. 11% had no injury. A fall down a stairway is actually a series of much smaller falls. The first one is the longest -- a fall from somewhat greater than child's own height onto the second or third stair. Gives diagrams. Please compare this article to Computer simulation of stair falls by Bertocci GE, et al., below (Arch Ped Adol Med, 2001). q.v.

Injuries to children younger than one year of age. Rivara FP, et al. Pediatrics 1988; 81: 93-97. 191 injuries studied: 146 accidental and 45 abusive. Accidental injuries were overwhelmingly nonsevere; abusive injuries were commonly severe and multiple injuries. There were 8 cases of abusive rib fxx, 0 cases of accidental rib fxx. Abuse group also had 13 CHI, 7 skull fxx, 7 LE fxx, 30 UE fxx.

The mortality of childhood falls. Hall JR, Reyes HM, Horvat M, Meller JL, Stein R. J Trauma 1989 Sep; 29 (9): 1273-1275. Dr. Kirschner calls this "a defective study" from the Chicago ME's office that did not challenge any of the histories, did not get complete medical records on the cases. (Hall was a surgical intern.) The records of the Cook County ME from Jan 1983 thr Dec 1986. "The prehospital and hospital records of these patients are not kept with the autopsy reports due to legal reasons; however, a brief summary was available in most of the charts." There were 44 pediatric deaths due to falls (5.9% of all pediatric deaths). 8 were high height, 18 mid-height, and 18 low-height (under 3 feet). From the abstract: **"Forty-one percent of the deaths occurred from 'minor' falls such as falls from furniture** or while playing." "Of the patients with 'minor' falls, 38% had parental delay in seeking medical attention, with deterioration of all." In the low-height group of 18 deaths, "These children all died from head injuries without any associated injury. Nine children had a delay in definitive treatment of greater than 4 hours with deterioration of eight. ... The one DOA in this group was an 8-month-old girl who fell off a couch onto a hard wood floor and was dead on arrival at the hospital. Her autopsy revealed a large acute subdural hematoma." The authors address the issue of possible occult abuse, saying that "While some of the 'minor' falls may have been secondary to abuse despite negative investigations (all of these had intense police investigation to rule out abuse), it is important that two of these falls did occur under medical observation. **'Minor falls' can be lethal**, especially in a toddler, and must be evaluated." Goes on to say that "[I]t is extremely rare to have visceral, thoracic, or non-skull fracture injuries in children who fall from less than 3 floor. It is, in fact, possible to suggest that if these injuries are found in a child with a fall from less than 3 stories, one should suspect abuse as the etiology of the injury." (citing Barlow, Niemierska, 1983 and Smith, Burrington, 1975 for the proposition that it is extremely rare.) This paper is cited by Wang et al. (2001) for the proposition that "Overall, falls account for 5.9% of childhood deaths."

Do retinal hemorrhages occur with accidental head trauma in young children? Alario A and Duhaime T (abstract) Am J Dis Child 1990; 144: 445. 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.** But see Cindy Christian's 1999 paper with some small RH from three atypical home accidents.

Bunk bed injuries. Selbst SM et al. Am J Dis Child 1990; 144(6): 721-723. See under "Bunk Beds."

Home accidents to children under 2 years of age. Tursz A, LeLong N, Crost M. Paediatr Perin Epidemiol 1990; 4: 408-421.

Deaths from falls in children: how far is fatal? Chadwick DL, Chin S, Salerno C, Landsverk J and Kitchen L. J Trauma 1991 Oct; 31(10): 1353-1355. 1984-1992 experience at the trauma center of CHSD. 338 falls of children under 6. 317 inf and ch w hx of a fall. Seven were fatal. Of these, five were undocumented short falls that had old fractures or multiple impact sites on the head. One fell from the second story and had a depressed skull fx, unwitnessed. 6 deaths were from head trauma, either SDH or cerebral injury. He concluded that all seven deaths were abusive. Note that 100 chil who fell less than 4 feet had 6 deaths, 65 chil who fell 5 - 9 feet had no deaths, 118 chil who fell 10 - 45 ft had one death. "When children incur fatal injuries in falls of less than 4 feet, the history is incorrect."

Injuries in infants and small children resulting from witnessed and corroborated free falls.

Williams R. J Trauma 1991; 31: 1350-1352. 106 witnessed falls and 292 uncorroborated falls under 3 years of age. Corroborated: one death fell 70 feet. Uncorroborated: 2 deaths fell less than 5 feet. Three corroborated skull fx fell under 10 feet: all three had small depressed skull fractures without LOC, caused by falling 4 or 5 feet onto an edged surface. Three depressed skull fractures in the corroborated group and two deaths and a number of severe injuries in the uncorroborated group. The AAP's abstract says: severe injuries were common in the uncorroborated group; witnessed falls were not generally associated with serious trauma. The authors concluded that **serious injury from uncorroborated short falls is highly suspect**. This article comes into discussion in Plunkett, 2001, *infra*.

Pediatric falls from heights. Musemeche C, Barthel M, Cosentino C, Reynolds M. J Trauma 1991; 31: 1347-1349. 70 children who fell more than 10 feet, usually out of buildings. 39 had significant head trauma, with 19 closed head trauma, 17 skull fractures, 2 subdurals, 1 epidural. 23 extremity fractures. 0 rib fractures. 3 patients had residual deficits.

Head injury in very young children: mechanisms, injury types, and ophthalmologic findings in 100 hospitalized patients younger than 2 years of age. Duhaime A-C, Alario AJ, Lewander WJ, Schut L, Sutton LN, Seidl TS, Nudelman S, Budenz D, Hertle R, Tsiasar W, Loporchio S. Pediatrics 1992 Aug; 90(2): 179-185. One fatal MVA had RH. 9/24 NAI had RH. Includes one walker-stairs injury.

Diffuse axonal injury by simple fall. Imajo T and Kazee AM. Am J Forens Med Pathol 1992; 13(2): 169-172. DAI is the second most common lethal head injury (after SDH.) DAI usually happens in traffic accidents, but it may occasionally result from falls from a height. Previously, it has not been associated with a simple fall or a fall of a distance not more than the victim's own height. We report herein a case of DAI from a simple fall. A 62 y o ETOH fell while drunk, hit back of his head.

Head injuries from short-distance falls. Root IR. Am J Forens Med Pathol 1992; 13: 85-87. Can be fatal.

Position statement on identifying the infant with nonaccidental central nervous system injury (the whiplash-shake syndrome.) Luerssen TG, Bruce DA, Humphreys RP. Pediatr Neurol 1993; 19: 170.

Epidural hematomas in children. Schutzman SA et al. Ann Emerg Med 1993; 22: 535

Fatal falls in childhood: how far must children fall to sustain fatal head injury? Report of cases and review of the literature Reiber GD. Am J Forens Med Pathol 1993; 14(3): 201-207. Sacramento Medical Examiner's Office 1983-1991. 22 falls in children under 5 years. 19 short fall histories led to 17 child abuse dx, **2 confirmed accidentals** -- one 6 ft, one 3 ft. One was from a top bunk. One was an accelerated fall from a rocking chair.? 3 falls from a high height fatal. This paper said to be "misleading" by Betty Spivack, *infra*. See also Betty Spivack's literature review in SIGCA printout in file 1998.

"Linear accel/decel has not been shown to be a significant factor in brain injury; instead it is the angular accel/decel which is far more significant. This has been known since the 1960s." [In other words, a short fall would have to have a rotational component in order to cause brain injury. Betty suggests that these two short falls may have had a rotational component. But, then, why can't any number of other short falls also have a rotational component? --JKR] Betty dissects this article in Chapter 5 of Lazoritz S and Palusci VJ, eds. *The Shaken Baby Syndrome: A Multidisciplinary Approach*. New York: The Haworth Abuse & Trauma Press, 2001. There she states that this article is "disturbing in its implications." (p. 70) She goes on to say that Reiber relied on experimental deceleration data from some experiments reportedly conducted at the Franklin Institute by Reichelderfer et al. in 1979, as reported in an article entitled "Unsafe playgrounds." These data, which Betty gives in tabular form (p. 74), show that falls onto concrete give the following impact times and (translational) decelerations for an unspecified (presumably hard) object dropped onto concrete:

	3"	6"	12"	48"
terminal velocity	1.2 m/s		2.4 m/s	4.9 m/s
impact duration	0.6 ms		0.5 ms	
deceleration	150-200 g	250-300 g	475-525 g	

Betty argues that these data are misapplied by Reiber in three respects:

1. The unspecified "object" that was dropped almost certainly was not biofidelic to the human head, because abundant clinical experience shows that comparable falls onto concrete by real persons seldom cause significant brain injury. Current experimental data with biofidelic models show deceleration times in the range of 1-3 ms for concrete, twice or more those of Reiber's Franklin Institute data. If these more likely impact durations are substituted into the equations for deceleration, the resulting decelerations are below the threshold value of 50g suggested by Reiber. "High decelerations are possible, but only if impact times are extremely small." (p. 73) Betty observes, concerning biofidelic models, that "minor changes in the model can have a major impact upon the apparent outcome of modeled events." (p. 75)
2. The velocities obtainable with short falls "starting from rest" are low. "Viscoelastic materials, such as brain, have injury criteria dependent upon velocity rather than acceleration for short-duration events." (p. 73)
3. The decelerations obtainable in simple drop experiments are purely translational. The primate brain is very resistant to translational injury. "Short falls, especially the extremely short falls hypothesized by Reiber, allow little or no possibility of significant rotational velocity or accelerative changes." (p. 74)

The pathophysiology of infant subdural hematoma Howard MA, Bell BA, and Uttley D. (British radiologists). Br J Neuros 1993; 7: 355-365. Has been cited to support the view that falls can cause fatal SDH. Also see Aoki, 1986; Greenes, 1998; Plunkett, 2001. Retrospective review of the charts and CT scans on 28 babies up to 18 mos over a 20-year period. All had findings consistent with an impact injury. "Our findings do not support shaking as the only cause of infant SDH formation and also suggest that non-accidental injury is a less common cause of SDH than it is believed to be." Cited by Wilkins, 1997, to the effect that some child pts are "at increased risk" for SDH caused by minor injury, and also that non-accidental injury is possibly overdiagnosed." See also *Knight's Forensic Pathology, 3d ed.*, 2004, above.

Falls from heights: a problem not just in the Northeast. Lehman D, Schonfeld N. Pediatrics 1993; 92: 121-124. L A County 1986-1990. 93 children with falls from buildings greater than 1 floor. 2/3 of them had fractures. 30% required ICU admission. 10% had residual neuro impairment. One death in a 20 month old who fell three stories onto concrete.

Falling out of bed: a relatively benign occurrence. Lyons TJ and Oates RK. Pediatrics 1993; 92: 125-127. Australia. 207 children in-hospital incident reports under 6 yrs who fell out of beds or cribs in a hospital. 176 had no injury. 31 had some observable injury. 29 of the injuries were trivial; 2 were

clinically significant: 1 clavicle fx in a 22-month-old , one linear skull fx in a ten-month-old crib fall with no LOC.

The rarity of serious head injury in day care centers. (letter). Chadwick DL and Salerno C. J Trauma 1993; 35: 968.

Population-based study of fall injuries in children and adolescents resulting in hospitalization or death.

Rivara FP, Alexander B, Johnston B, Soderberg R. Pediatrics 1993 Jul; 92(1): 61-63. (UW). Reviewed all fall-related hospital admissions of children and adolescents up to 19 years old in Washington State for 1989 and 1990. Total 2658 fall-related admissions, comprising 29% of all pediatric trauma admissions. In the 0-4 age group, 11.2% were for same-height falls; of these, 9.4% were slip and fall, 1.4% were collision with another person. 52% in this age group were falls from one level to another, such as from playground equipment, beds, tables, and chairs. 22% were "not specified" as to height or mechanism. So in the 0-4 age group, had:

52%	falls from one level to another
11%	same-height falls

Types of injuries are not broken out by age. However, the authors state that in the 0-4 age group, 42% had head trauma, compared with only 14% of adolescents. The single most common mechanism of fall-related injury was fall from one level to another, and 70% of these injuries were to children under 10 (while injuries from same-height falls were more concentrated in the adolescent age.) "Few injuries appear to result when children fall from low heights such as beds and couches. (quoting Helfer, 1977, Nimityongsk, 1987) Thus, it appears that a greater height and/or more unforgiving surfaces are needed to produce significant injuries such as reported in this study. Common principles of injury control can apply to most of these injuries... The placement of barriers to prevent falls, whether they be from changing tables, bunk beds, stairs, balconies, or playground equipment... Proper surfacing materials can serve to absorb the impact of falls and prevent injury for such items as both indoor and outdoor play equipment" (citing refs.) "Falls from stairs are a problem at the extremes of age." (citing Joffe & Ludwig).

Blunt trauma in children: causes and outcomes of head versus extracranial injury. Lescohier I and DiScala C. Pediatrics 1993 Apr; 91(4): 721-725. This is a statistical article about trauma in general, from the National Pediatric Trauma Registry. Under 15 years. All blunt trauma. 8639 children. Divided into Extracranial only, Head only, and Both. 50% extracranial only, 50% head only or both. Falls were the leading cause of injury in the head-only group, while traffics predominated in the both group and the extracranial group.

Paediatric head trauma: influence of age and sex, I: epidemiology. Barney J, Favier J, Froidevaux A-C. (Geneva) Child's Nervous System 1994 Nov; 10(8): 509-516. From the abstract: a consecutive unselected series of 1,812 chil up to 15 years old admitted for head injury over a period of 8 ½ years, divided into five categories of injury: benign injury, EDH, SDH, open brain laceration, brain contusion. Pts were divided into three age groups: group I = 0-3, group II = 3-9, group III = 9-15. Group I was subdivided into subgroup Ia (0-1) and subgroup Ib (1-3). Also broken out by sex. Boys were 2X girls overall, but the sexes were about equal when it came to road accidents. Road accidents were responsible for 94% of the deaths. The abstract also says, "Falling was the most frequent cause of injury." There were only 8 cases of SDH; 4 of these were in subgroup Ia. This paper is relied on by Ommaya et al. (2002) as showing that SDH occurs with low-height falls, but Christian and Duhaime in their review (Child Abuse Quarterly for January 2003) point out that of the four SDH's in subgroup Ia, three were from injuries classed as "miscellaneous," which included child abuse. There is another paper, part II:

Paediatric head trauma: influence of age and sex, II: biomechanical and anatomo-clinical correlations.

Barney J, Froidevaux A-C, Favier J. (Geneva). Child's Nervous System 1994 Nov; 10(8): 517-523. Cited by Denton & Mileusnic, AJFMP Dec 2003 (below) for the proposition that "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."

Falls and childhood deaths: sorting real falls from inflicted injuries. Chadwick DL. The APSAC Advisor, Winter 1994. Updated 1999 in the ICAN "Unified Response" newsletter, see below.

Stairway-related injuries in children. Chiaviello CT, Christoph RA, and Bond GR. Pediatrics 1994; 94(5): 679-681. Studied 69 ER pts under age 5 prospectively. Median age 2 yrs. 78% had no significant injury. 22% had significant injury: 11 had concussions (16%). 5 had skull fx* (7%). 2 had cerebral contusion.* 1 had SDH.* 1 had a C-2 fx. Total 90% had head & neck injuries. 6% had extremity injuries, 4% truncal injuries. No deaths. No patient had injuries to more than one body region. "These studies [this one plus the one previous study by Joffe & Ludwig 1988] together suggest that some of the most severe injuries occur when infants fall with an adult while being carried on the stairs." **"Severe head injury is compatible with a stairway-related fall.** However, injuries involving multiple body regions, or severe truncal or extremity injuries should prompt a search for an alternate mechanism, including intentional trauma."

*2 skull fxx, 1 cerebral contusion, 1 SDH occurred while being carried downstairs by an adult.

Comment by Cindy Christian at the Second National SBS Conf 1998: two circumstances in which stairway falls cause serious head injury in infants: (1) walkers, (2) being carried by an adult who falls. See also under "Walkers & Strollers." See also Joffe & Ludwig, 1988, the other major art on stairways.

Crush injuries to the head in children. Duhaime AC, Eppley M, Margulies S, Heher KL, Barlett SP. Neurosurgery 1995; 37: 401-406

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.

Radiographic Atlas of Child Abuse: a case studies approach. Harris VJ, Lorand MA, Fitzpatrick JJ, Soter DK. New York: Igaku-Shoin, 1996. ISBN 0-89640-258-4. "A fall out of bed is one of the most deadly histories elicited in pediatrics." p.22 "Depressed skull fractures are very unusual in the first year of life and may be caused by hitting an edge rather than a flat surface, or by a direct blow to the head with a hard object. Depressed skull fractures that are said to be the result of a short fall to a flat surface are suspect." p. 30. "Epidural hematomas have been described in chil under 2 years of age who have fallen less than 4 feet." p. 35. See above under "Fractures -- In general."

Mechanisms of primary head injury. Gennarelli TA and Meaney DF. Ch. 259 In: *Neurosurgery*, 2d ed., Wilkins RH and Rengachary SS, eds., New York: McGraw-Hill, 1996.

Epidural hemorrhage: is it abuse? Shugerman RP, Paez A, Grossman D, Feldman KW, Grady MS. Pediatrics 1996 May; 97(5): 664-668. Retrospective review of all children age up to 4 years with EDH or SDH. 93 cases.

	Acci (n=56)	Abuse (n=31)	Undet (n=6)
SDH (n=59)	25 (42%)	28 (47%)	6 (10%)
EDH (n=34)	31 (91%)	2 (6%)	0 (0%)

Correlates the two types of hematomas with mechanism. EDH from an impact consistent with a fall. Most of them result from short falls. SDH: "...rotational accelerations that occur with shaking, with blows to the head that initiate its rotation on the axis of the neck, or with rapid deceleration of the body during which the head remains free to rotate (as in auto crashes in which the passenger's body is restrained. Such forces result in disconjugate motion of the brain and skull..." (citing Duhaime, Gennarelli, Thibault, SBS, 1987; Gennarelli, Thibault, SDH, 1982; Ommaya, Faas, Yarnell, Whiplash injury and brain damage, 1968). The authors note that in their material, the actual incidence of abuse in SDH patients was probably underestimated, because the CPS had a policy of not diagnosing abuse in SDH-only cases; they required extrinsic injuries. Comments by JKR: 1. The paper does not break out a history of fall or short fall as a historical mechanism for the hematomas, although the authors comment on biomechanical-theoretical grounds that EDH's are biomechanically consistent with falls on a hard surface, while SDH's are not.

2. The fact that two EDH's did result from abuse is concerning; given that the percentage (6%) was substantial, it means that a history of a fall in an EDH cannot automatically be accepted. This would seem to be even more true in a fatal case, since EDH's promptly treated are seldom fatal, and fatality could imply delayed rescue.

Kim Oates, oral presentation at the 1997 San Diego conference. (Have handout).

He reviewed 207 documented falls in children under six years old. Fell out of bed. No serious injuries.

One simple skull fx and one clavicle fx. 29 chil had superficial injuries.

"Falls from bed are unlikely to cause serious injury."

Leestma, JE. Forensic neuropathology. Chapter 12 in *Pediatric Neuropathology*, edited by Serge Duckett. Baltimore: Williams & Wilkins, 1995. Pp 243-283 (have)

Leestma, JE. Forensic neuropathology. Chapter 11 in Garcia JH et al., eds., *Neuropathology: the Diagnostic Approach*. St.Louis: C.V. Mosby, 1997. Pp 475-527.

Cerebrovascular response in infants and young children following severe traumatic brain injury: a preliminary report. Adelson PD, Clyde B, Kochanek PM, Wisniewski SR, Marion DW, Yonas H. *Pediatr Neuros* 1997; 26: 200-207. (have)

Syncope after immunization. Braun MM. *Arch Pediatr Adol Med* 1997; 151: 255-259. From the VAERS there were 697 incidents of syncope with 6 skull fxx or intracranial bleeds in vaccinees 12-28 years old.. Some had EDH and/or coup or contrecoup contusions, one SDH. [These findings make no sense. -JKR]

Comparison of intracranial computed tomographic findings in pediatric abusive and accidental head trauma. Hymel KP, Rumack CM, Hay TC, Strain JD, Jenny C. *Pediatr Radiol* 1997; 27(9): 743-747.

They compared 39 accidents with 39 abuses.

Closed-head injury.

	<u>Acci</u>	<u>Ab</u>
Interhemispheric	5 %	44 %
SDH	10 %*	41 %
Large subacute collections	0 %	21 %

They compared the statistical frequency of six particular CT findings as to accidental and inflicted cases of closed-head injury (CHI). 39 accidental children versus 39 inflicted children.

	<u>Acc</u>	<u>Ab</u>
Intracranial findings	13%	51%
Interhemispheric hemorrhage	5%	44%
SDH	10%	41%
Large nonacute extraaxial fluid collection	0%	21%
Basal ganglia edema	0%	13%
Posterior fossa hemorrhage	0%	10%
Frontoparietal shearing tears	3%	3%

*Three infants with accidental CHI had a skull fracture with a small underlying SDH at the site of impact. Note that this type of SDH in Hymel's view is biomechanically different from the diffuse convexity SDH. (See his biomechanics article.) Small localized SDH under a fracture is strictly fracture-related and can be due to focal-loading linear deceleration. Diffuse convexity SDH is due to distributed-load long-duration rotational deceleration.

Head injury -- abuse or accident? Wilkins B. *Arch Dis Child (Br.)* 1997 May; 76: 393-397. This is a literature review by an Australian pediatric intensivist. He states that this is a "dilemma." (Note the use of the same word by two different authors in Reece, Second Edition, and in the 2001 NAME Position Paper. -JKR) "Publicised court cases, with widely divergent medical opinions, illustrate the dilemma of

distinguishing between inflicted and accidental causes, especially when there are no other signs of abuse but just an uncorroborated, alleged accident, often a fall. Although there has been resistance to diagnosing abuse, there may also be over enthusiasm to do so, and although there is an increasingly prevalent opinion that short falls can never cause serious injury, **this, too, is still open to debate.** The literature reports a few severe injuries from low-height falls, citing Hall, 1989, where the victims fell while under medical observation; Reiber, 1993. 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." Goes on to discuss Raimondi's own work (Gutierrez & Raimondi, 1975) in a series of infants and toddlers with SDH where "both abuse and falls were identified as causes." Also refers to Howard, Bell & Utley, 1993, to the effect that some child pts are "at increased risk" for SDH caused by minor injury, and also that non-accidental injury is possibly overdiagnosed." Also citing neurosurgical literature (Livingston, 1991; Rimel, 1982; Dacey, 1986) for the proposition that "Subdural haematoma contributing to neurological deterioration, cerebral oedema or death has been described in adults and children after only moderate or trivial head injury." He then discusses biomechanics. First, how much acceleration is needed to cause brain injury? On this, he refers to experiments including the Philadelphia group (Duhaime SBS, 1987, achieving only 9G by shaking while 285 G was needed to cause SDH, a figure extrapolated from anesthetized monkeys, Gennarelli, Thibault, 1982; Australian road-accident research suggesting that less than 150G can cause fatal concussion if the impact was to the side of the head, referring also to Hanigan's tin-ear article of 1987 which calculated that with side blows a much lower acceleration could cause brain injury and SDH. Second, dynamics of falls and blows. A 1.2 m fall onto packed earth can give 200G, as determined with a 1.5 kg dynamometer ball drop experiment (giving all the physical numbers), but then there is scalp-skull deformation and the unknown effects on the cranial contents, such that these figures "cannot be calculated for the brain." Citing Popovic et al., 1994 for the proposition that deceleration is greater for smaller objects and harder surfaces. Suggests that the main factor for brain injury may not be acceleration but kinetic *energy* imparted to the brain and the bridging veins. Kinetic energy is proportional to the integral of acceleration over distance. "The time-course and magnitude of these energy changes in the brain cannot be measured or calculated." Third, Linear and rotational motion. Citing the Philadelphia group (Gennarelli, Thibault, Adams et al., primate DAI, 1982; Duhaime, SBS, 1987; Gennarelli, Thibault, SDH, 1982) on the need for rotational injury to cause DAI or SDH, but saying that work was limited to an axis of rotation outside the skull (in the neck.) What if the axis of rotation is *inside* the skull? This could happen with a blow to the side of the jaw, an oblique strike by a car, or in a rolling fall) It will cause "true rotation" of the brain in the skull, (instead of with the skull) with "the motion transferred from skull to brain via shear rather than compressive forces. The bridging veins are potentially stretched more and there is likely to be less damping by CSF, so kinetic energy stored in the brain may be dissipated in tearing these vessels....[and DAI]." "True rotation might be rare in simple falls but explain rare cases of severe injury." Goes on to discuss Gennarelli's argument that the rate of increase of acceleration could relate to bridging-vein rupture, whereas a softer impact could cause DAI, because of possible effects of resonance and damping on the motion of the brain. As to why side-impact blows may be more damaging than occipital impact: either because resonance and damping could differ in side blows, or because they may produce true rotation. Conclusion: "In the absence of clear signs of abuse we cannot jump to the conclusion that injury is non-accidental just because there is brain injury or subdural haemorrhage, especially if the alleged fall height is greater than in 'household' falls." Brain physics cannot be derived accurately from skull physics. Hence "There are too many variables and unknowns to allow a categorical statement that a certain fall did or did not injure a child." "The medical appraisal [as distinguished from the psychosocial investigation] may not be conclusive about whether an injury is compatible with the stated history." There follows a commentary by a discussant. The discussant refers to Weber's cadaver experiments in which skull fractures were easily caused by short drops. Cites Duhaime for the proposition that "Acute SDH and axonal shearing injuries have been created by shaking anaesthetised primates," and questioning the value of experiments on simplified models. Goes on to sermonize that "Many of us have exhibited logical inconsistency by believing parents who say they have shaken their baby but disbelieving those who deny it, and we believe parents who describe a fall where no significant injury occurs but disbelieve others when injuries are found."

Depressed skull fracture in a 7-month-old who fell from bed. Wheeler D, Shope T. Pediatrics 1998; 100: 1033-1-34. From San Diego. 24" fall onto a toy car led to a ping pong fx. No brain injury. Abuse ruled

out. Child did well. Fits perfectly with Hymel, 1998. Compare the case report of Denton & Mileusnic, AJFMP Dec 2003, where the nine-month old child died a delayed death from falling 30" off a bed with a linear parietal fracture and fatal brain swelling.

Occult intracranial injury in infants. Greenes DS, Schutzmann SA. Ann Emerg Med 1998 Dec; 32(6): 680-686. 101 infants under 2 yrs seen in ER for reported head injury. 19 had intracranial injury without overt symptoms. Small subdurals at the point of impact and epidurals were most common. 7 had injuries from falls of less than 3 feet. Cited by Plunkett (Am J Forens Med Path 2001 Mar) as reporting seven children with falls from 0.6 to 1.5 meters who had SDH, no LOC, and no Sx. But note that these were fracture subdurals.

Traumatic neuropathology (monograph). Pearl GS. Clin Lab Med 1998 Mar; 18(1): 39-64. Includes adult and child abuse areas.

Neuroimaging, physical, and developmental findings after inflicted and noninflicted traumatic brain injury in young children. Ewing-Cobbs L, Kramer L, Prasad M, Canales DN, Louis PT, Fletcher JM, Vollero H, Landry SH, Cheung K, Pediatrics 1998 Aug; 102(2): 300-307. A prospective clinical study of inflicted and noninflicted head injury. Prospective clinical study of 40 brain-injured children from 1 month to 6 years old, comparing 20 Inflicted (I) cases with 20 Noninflicted (NI) cases. Most of the NI were automobile accidents, some falls. Found that they were equally severe from a neurological point of view both early and late (neuro status was not a discriminant). SDH were not seen in falls, but were seen in MVA. SDH was 16 inflicted to 9 noninflicted automobile. EDH was 3 NI to 0 I. ICH was 6 NI to 1 I. Preexisting brain injury (cerebral atrophy or encephalomalacia) was seen only in the I group and was present in 40% of them (8/20). RH was a discriminant, present in 14 I and 0 NI. RH could be either unilateral or bilateral. Location of fractures differed, with the NI group having mainly facial fractures, the I group having rib tib-fib, femur. The perpetrators were 30% natural fathers, 5% mothers, 15% boyfriend or girlfriend, 10% babysitters. See next.

Acute neuroradiologic findings in young children with inflicted and noninflicted traumatic brain injury. Ewing-Cobbs L, Prasad M, Kramer L et al. Child's Nerv Syst 2000; 16: 25-34. See previous; these two studies go together. Total of 60 patients observed prospectively. The accidental group were older. The inflicted group had significantly more acute SDH and more interhemispheric; also more preexisting brain abnormalities. But had *less* shearing injuries than acc. (!) Acc group had more skull fxx. Compare the findings of Dashti and co-workers at Rainbow Babies 1999 in comparable prospective study, below.

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. Walker stairs, adult carrying stairs, infant carriers, counters, beds. Opth was documented in all. 6 had RH: of these, the most severe was multiple bilateral posterior-pole hems. None had peripheral hems, large hems, or traumatic retinoschisis. Quite a number of skull fractures and SDH's. No deaths.

Abusive head trauma? A biomechanics-based approach. Hymel KP, Bandak FA, Partington MD and Winston KR. Child Maltreatment 1998 May; 3(2): 116-127.

Loading apparently means any type of energy transfer.

Static loading = very slow, e.g. squeezing, crushing. [As to static loading, one may wish to look at the more recent paper on lateral crush injuries of the head by Tortosa and colleagues from Spain. Bitemporal head crush injuries: clinical and radiological features of a distinctive type of head injury. Tortosa JG, Martinez-Lage JF, Poza M. J Neuros 2004 Apr; 100: 645-651. Finding that "Static forces applied to the head in a transverse axis produce fractures in the skull base that cross the midline structures without producing significant cerebral damage. Stretching of the cranial nerves at the skull base explains the near-universal finding of paralysis of these structures, whereas an increase in the vertical diameter of the skull accountf for the occurrence of diabetes insipidus in the presence of an intact function of the anteriorf paituitary lobe." Names this neuroendocrine complex the "syndrome of bitemporal crush injury to the head." --JKR]

Dynamic loading = very fast, less than 200 msec. Includes:

- Impact (contact) loading (cranial collision)

- Impulsive loading (acceleration of the head)

“Strain” is deformation by a load. “Strain” is deformation per unit length.

“Stress” is force per cross-sectional area. There are three types of strain:

- Compressive strain (shortens the impacted element)

- Tensile strain (lengthens the impacted element)

- Shearing strain (distorts the element)

When a moving impactor strikes the stationary head,

- Cranial deformation is favored when

- A narrow area of impact

- A fixed head

- Cranial acceleration is favored when

- A wide area of impact (distributed loading)

- A freely movable head

Three types of cranial acceleration

- Translational (when the brain's center of gravity moves)

- Rotational (when the brain's center of gravity does not move)

- Combination of the two

Two main categories of primary cranial injuries

Categories (You can have both categories of injury in the same victim.)

- Contact injuries, caused by the impact of an object on the fixed head. They cause focal strain

- Essentially these injuries are caused by the fixed-head deformations (strain).

- They include scalp injury, skull fracture, underlying EDH, SAH, or small localized underlying SDH. Also underlying parenchymal brain injuries. Also distant injuries such as BSF and contrecoup injury.

- Noncontact injuries are caused by cranial acceleration, (of the movable head) regardless of whether there is impact.

- They cause injury by three subclasses of intracranial mechanisms:

- Subclasses

- Brain moves relative to skull causing rupture of bridging veins

- Strain occurs inside the brain

- Combination of the two -- frequently seen in impact injuries

Don't forget the role of secondary cranial injuries -- cerebral ischemia due to apnea, blood loss, space-taking effects of hematomas, and cerebral edema.

Tissue response to contact or noncontact loads are of two broad categories: stress (internal forces) and strain (deformation, $L1 - L0 / L0$.) The mechanical property of a tissue is how it converts stress into strain and vice versa. Tissue generally resists deformation in very abrupt force (hence low strain). Slower force allows more tissue deformation (high strain). Low strain (not very deforming) force tends to protect the cranial contents even if there is high internal stress; high strain (very deforming) force tends to injure it even if the internal stress is lower. This explains why infants rarely have serious head injuries from falls: low strain.

As to subdural hematomas, they differentiate contact subdurals from noncontact subdurals. (p. 119). (Elsewhere, Dr. Hymel has used the terms “focal” to apply to contact subdurals and “diffuse” to apply to noncontact subdurals. (San Diego Conference, 2000)). Contact subdurals are small localized subdurals that occur at the impact point where there was deformation (strain) on the skull. Tend to be associated with impacts against a narrow object. Noncontact subdurals do not result from skull strain, but result from “superficial tensile (stretching) and/or shearing strains to vascular tissues caused by differential acceleration of the skull and brain, resulting from anterior-posterior, cranial acceleration of relatively short duration and high magnitude.” (citing Gennarelli & Thibault's subdural article of 1982). *Je vous signale:*

one should bear this distinction in mind whenever one reads articles reporting the finding of “subdural hematomas” NOS. In none of the articles in the literature is this distinction reported; hence the biomechanical meaning of those findings is obscure. –JKR.

Concussion and its more severe cousin DAI are caused only by angular or rotational acceleration, not translational and not fixed-head impact. Angular or rotational acceleration produces parenchymal strain. This contention is ascribed to British wartime experiments and Ommaya & Gennarelli’s 1974 concussion experiment on squirrel monkeys as extended by Adams, Gennarelli & Graham’s DAI experiment (1982). Contact and noncontact subdurals, however, were also caused by translational acceleration. Angular acceleration produced every known type of cranial injury with the exception of skull fracture and epidural hematoma. Ommaya & Hirsch 1971 did an experiment on monkeys involving rotational acceleration produced by either contact loading or impulsive loading. They found that it took higher velocity to produce concussion (i.e. parenchymal injury/ parenchymal strain) by impulsive loading than it did by contact loading. Therefore impact injuries involving rotational acceleration should be more severe than impulsive-loading (pure spin or shake) injuries at the same velocity or energy level.

Next there is the question of the *duration* of the cranial acceleration (1968). Longer duration of acceleration produced more concussion for the same amount of acceleration than did shorter duration acceleration, because the longer duration allowed for more parenchymal strain (deformation) to develop, apparently because of the elastic properties of brain.

Next there is a discussion of the *direction* of acceleration. For noncontact (angular) acceleration, 1982 monkey experiments showed that sagittal-plane angular acceleration produced SDH but no DAI, while coronal-plane angular acceleration produced DAI. Also, as the direction was changed from sagittal toward coronal, the injury effect of a given acceleration increased, but apparently SDH decreased.(?) Result: noncontact SDH results from sagittal-plane acceleration of short duration and high magnitude. DAI results from coronal acceleration of long duration and lower magnitude.

How to explain the clinical observation that AHT victims commonly have a combination of cranial injuries that is not characteristic of any one injury mechanism described in the experimental work? They often have fractures, SDHs *and* DAI all together, even though these types of injuries are biomechanically separable. You have to posit, in effect, multiple injuries -- multiple accelerations.

Discusses DAI as found in infants. Immediate LOC is expected and ordinarily observed clinically, but they are not doctrinaire about this: “Although LOC may evolve over time in response to secondary hypoxia-ischemia, immediate LOC at the moment of injury is the clinical hallmark of primary diffuse brain injury.” (None of Adams’s 45 patients (1982) had a lucid interval.) Axonal swelling and retraction balls. Radiological findings: parenchymal petechiae in the cc, dorsolateral quadrants of the rostral brainstem, and other central portions. Pathology: infants under 5 months had no histologic findings by conventional stains but were seen by silver stains (1987), and no tears or hemorrhages. Contusion tears have been known since Lindenberg & Freytag 1969.

"Severe or fatal cranial injuries in humans are most often the result of primary acceleration strains -- not contact strains. In the presence of severe or fatal head injuries, the critical question is whether or not the biomechanical history explains significant cranial *acceleration*." 117 (Compare the view of Wilkins (1997) that it may be whether it explains significant cerebral kinetic energy.)

Go on to discuss the accidental fall-versus-inflicted problem, since a fall is the most frequent explanation offered by caregivers of head-injured children. The authors posit two biomechanical classes of primary injury that you should look for: I. Contact injuries (soft tissue injury, skull fracture, focal impact site intracranial hemorrhage, focal impact-site parenchymal injury (i.e. fracture contusions and coup contusions), and focal away-from-impact-site parenchymal injury (i.e. contrecoup contusions.)) II. Acceleration injuries (diffuse SDH, concussion, and DAI). “Determine if isolated contact, isolated acceleration, or combined contact and acceleration injury mechanisms were required” (to cause the primary injuries that you find.) For each primary injury that you find, look in Tables 5, 6 and 7 to see the biophysical description of the injury mechanisms that could have produced this particular injury. Table 5 is

for contact injuries: (subpart I for head hit by a moving object, subpart II for moving head hitting a fixed object). Table 6 is for noncontact injuries: (subpart I pure impulsive loading, subpart II hit by a moving object, subpart III moving head hits a fixed object). Table 7 is for when you find both contact and accelerative-type injuries: (subpart I head hit by moving object, subpart II moving head hits object.) In the case of an alleged household fall, the nub of the issue comes down to subparts II of tables 6 and 7. These tell us to look for the deformability of the surface, the speed of the impact, the direction of the impact, etc. The tables are slightly simplified vis-à-vis the more detailed research discussion given above, particularly when the direction of loading might be neither straight-sagittal nor straight-coronal. Also, these tables do not go into the question of a center of rotation within the head, due perhaps to an oblique impact off the center of the head possibly causing brain spin (Wilkins, 1997).

In concluding, the authors mention the unfortunate fact that only one biomechanical study of actual infant abusive head trauma has ever been done (Duhaime SBS, 1987). Therefore, the following biomechanical unknowns remain to be studied regarding AHT: (1) the specific possibilities of producing a (diffuse) SDH from a fall; (2) better definition of DAI in young infants; (3) creation of age-specific biophysical fall models. (I have rephrased these items for clarity.)

Fatal falls from a height: the use of mathematical models to estimate the height of a fall from the injuries sustained. Lau G, Ooi P L, and Phoon B. FSI 1998; 93: 33-44. Derives a formula using multivariate analysis. But Betty Spivack in her comment in *Child Abuse Quarterly*, October 1998 comments unfavorably on both the mathematics and the overall validity of this approach, saying that Barlow's 1983 paper is more reliable. She points out that the authors have great difficulty in accounting for the variability in injuries. The authors' formula only accounts for 46% of the variability in the height of a fall, indicating that other factors play a large role in determining the injuries suffered in a fall from a substantial height.

Subdural hematomas in children under 2 years: accidental or inflicted? A ten year experience. Tzioumi D and Oates RK (Australia). *Child Abuse & Neglect* 1998 Nov; 22(11): 1105-1112. Retrospectively reviewed 38 children with SDH's. 21 nonaccidental (55%), 15 accidental (39%), 2 natural disease – nontraumatic etiology (6%). Ophthalmoscopy was done on all 21 of the nonaccidental and on 5 of the accidental: of these, 16 (84%) of the nonaccidental had RH, none of the accidental. RH were bilateral in 7, unilateral in 6, and absent in two shakens. Patients were divided into “shaking” and “impact” groups depending on skull fractures or a history of impact. The four things that predicted non-accidental injury were:

1. Young age
2. RH
3. Rib & long bone fractures
4. Delayed rescue

Inflicted	SDH	55%
Accidental	SDH	39%
Spontaneous	SDH	6%

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.

Bilateral pediatric skull fractures: accident or abuse? Arnholz D, Hymel KP, Hay TC, Jenny C. *J Trauma* 1998; 45: 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. CT was normal, baby did well. The authors comment that bilateral fractures are highly suspicious for abuse, and rarely seen in accidents. Dr. Kleinman comments that this can mimic two blows because of the hemorrhages on opposite sides of the head. They do not refer to the baby cadaver drop experiments of Weber, 1985, 1987, which support this mechanism. --JKR

Role of coagulopathy in patients with head trauma. Lozance K et al. J Clin Neuroscience 1998; 5: 394-398. Markedly abnormal coags correlated strongly with outcome. Kent Hymel advises us to consult also Pediatrics 1997; 99: 371.

Subdural hemorrhages in infants: population-based study. Jayawant S, Rawlinson A, Price J, Schulte J, Sharples P, Sibert JR, Kemp AM. BMJ 1998 Dec; 317(7172): 1558-1561. England. A retrospective study of all children under 2 who had SDH in South Wales and West England, excluding neonates. 33 cases (age range 3 weeks to 17 months) of which 28 were under 1 year. 27 of the 33 cases were considered highly suggestive of abuse (82%); abuse was confirmed by the medicolegal authorities in 21. 14 had a history of shaking. 19 infants had other evidence of abuse (fractures, torn frenulum, bruises, burns, bites, induced hypernatremia.) 16 had fractures. Of the 28 infants, 27 had oph exam, and 12 of these (44%) had RH. One SDH arose from a road accident and had multiple skull fractures with no RH. Conclusion: most SDH in this age group are due to child abuse, but in a few the cause is unknown. The overall incidence of SDH in children under 2 in South Wales was found to be 12.8/100,000 children per year. The incidence in infants was 21/ 100,000. The incidence of non-accidental SDH was found to be 10/ 100,000/ year. Both SDH and non-accidental SDH were more common in infants than in older children. "Our paper provides the only population-based case series of which we are currently aware. We show that the majority of subdural haemorrhages in children under 2 years of age are due to child abuse." "Subdural haemorrhage in infants and young children presents major challenges in diagnosis to doctors, social workers, and courts. In clinical practice, it is often difficult to deduce whether a subdural haematoma in an infant is caused by accident or abuse." They recommend a thorough abuse workup in all SDH cases because of the high incidence of NAI. They give the risk factors. "Previous physical abuse in an infant is a significant risk factor for subdural hemorrhage and must be taken seriously by child protection agencies." Also gives information on the perpetrators (boyfriend in 5, father in 6, mother in 3.) Does not give any details as to the morphology of the hematomas or the RH.

The epidemiology of urban pediatric neurological trauma: evaluation of, and implications for, injury prevention programs. Durkin MS, Olsen S, Barlow B, Virella A, Connolly jr ES. Neurosurgery 1998 Feb; 42(2): 300-310. A survey of the North Manhattan Injury Surveillance System (NMISS) from 1983 to 1992. Includes brain, spinal cord, peripheral nerve. From the abstract: Chil under 1 year were the highest incidence of both major and minor inj. "Traffic accidents and falls were the leading causes overall (38 and 34% respectively)." Further: "Falls were the most common cause of neurological injury in children younger than 4 years, accounting for more than half of all cases in both infants (136.4 incidents/ 100,000 pop/yr) and preschoolers (55.43 incidents/100,000 pop/yr). ... Assault was a major cause of neurological injury in infants (24.9 incidents /100,000/yr)..." Fig 3 shows that in the under 1 age group, falls were about 60% of all incidents, with non-gun assault being about 15%. Compares these findings to the Kraus (San Diego) study of 1986. Injury incidence and severity were class-correlated.

Short vertical falls in infants. Tarantino CA, Dowd MD, Murdock TC. Pediatric Emergency Care 1999 Feb; 15(1): 5-8. Retrospective chart review of 167 infants at or under ten months admitted to an ER with a history of falling less than four feet. The study population was specifically limited to infants and to histories of short vertical falls as opposed to older children hurt while running and climbing. Excluded all walker falls, car seats,* stairs, all accidents related to walking, falling on objects, and being fallen on by a caregiver. Mean age 5 mos, max 10 mos. 10% had to be hospitalized. 55% rolled off a bed, 16% rolled off a couch, 10% fell off other objects, 20% were dropped. Overall, 85% had minimal or no injury, 15% (n=25) had significant injury: 7 long bone fracture, 18 closed head injury including 12 linear skull fractures and 2 intracranial bleeds. The two intracranial bleeds were later found to be child abuse: one had an initial history of rolling off a couch, which was changed to being hit in the head with a stereo speaker thrown during a domestic dispute; the other also had an initial history of rolling off the couch, which was changed to shaking by the father. Excluding the two child abuses, the only mechanism that led to any significant injury was **being dropped. No child sustained an intracranial hemorrhage from a short vertical fall.** Being dropped was overrepresented among the significant injuries, as opposed to falling.

	Minor or no injury (142)		Significant injury (25)	
Fell	119	(84%)	14	(56%)

Dropped	<u>23</u>	(16%)	<u>11</u>	(44%)
	142	100%	25	100%

The authors note the comparison to the two stairway papers (Chiaviello, 1994 and Joffe & Ludwig, 1988) in which being carried was a risk factor for more serious injury. "The biomechanics of a fall from a caretaker's arms may be different. Further studies may help to clarify the velocity and forces of being dropped from a caretaker's arms versus rolling off a bed or other stationary object." [But compare Denton & Mileusnic, 2003, reporting a 9 month old who rolled off a bed, had a 64 hour lucid interval, and died of TAI/ skull fx. See under "Time of Injury -- Head." --JKR]

Briefly summarizes the short-fall literature: Helfer, Duhaime, Nimityongskul, Lyons finding that short falls rarely if ever result in serious injury; while "other investigators have reached different conclusions:" Williams showing three depressed skull fractures in the corroborated group and two deaths and a number of severe injuries in the uncorroborated group; Hall finding 18 deaths and concluding that ; "minor falls can be lethal;" States that "A significant amount of debate exists in the literature regarding the type and extent of injuries in children resulting from a short fall."

* Falls in car seats are a known cause of head injury. See under "Car Seats," above.

Diffuse axonal injury in infants with nonaccidental craniocerebral trauma: enhanced detection by beta-amyloid precursor protein immunohistochemical staining. Gleckman AM, Bell MD, Evans RJ, Smoith TW. Arch Pathol Lab Med 1999 Feb; 123(2): 146-151. Beta-APP study used 7 SBS brains and 7 control brains (which included 1 HIE and one cerebral edema). Used paraffin blocks. Five SBS brains showed + APP in all sections including medulla, cerebellum; more severe changes in the brain stem, less in the subcortex. Most sections were negative by H&E. Controls were all negative.

Falls from heights among children: a retrospective review. Lallier J. J Pediatr Surg 1999 Jul; 34(7): 1060. Cited by APRI 15(7), 2002 for the proposition that "for a fall to be fatal, the vertical distance generally needs to be 20 feet or more," and that "The caretaker's story of the fall -- i.e. how far it was, the surface onto which the child fell, and what body part(s) the child landed on -- will be of crucial importance in determining of the fall was truly accidental."

Highchair accidents. Mayr JM, Seebacher U, Shimpl G, Fiala F. (Graz) Acta Paediatrica 1999; 88: 319-322. 103 babies aged 7 mos to 2 ½ years. 15% had skull fracture. 69% had soft-tissue injuries of the head or face, 2% had limb fractures. **No fatalities. No SDH, no SAH.**

Monkeybar injuries: complications of play. Waltzman ML, Shannon M, Bowen AP, Bailey MC. Pediatrics 1999; 103:e58. Many different kinds of injuries; consistent with multiple-broken fall.

Epidural hematoma in infants: a different entity? Ben-Adani L, Flores I, Spektor S, Umansky F, Constantini S. (Tel Aviv). J Trauma 1999; 46: 306-311. Usually from low-height falls. Some presented fully conscious, some unconscious.

Current patterns of inflicted head injury in children. Dashti SR, Decker DD, Razzaq A, Cohen AR. Pediatr Neurol 1999; 31: 302-306. Rainbow Babies Jan 95 thr Dec 97 retrospective chart review of 405 charts ages 0-18 adm w head injury. AHT was in 38 pts (9%). AHT was ages 1 mo to 10 years, average age 5 mos. 26% had a history of a witnessed fall. 47% had RH, while 0 of 367 accidental had RH. 9 pts (24%) had skull fx -- 7 linear, 2 depressed. 63% had SDH. 10% had EDH. Dr. Reece points out that when this study pop is stratified for age under 2, the incidence of AHT becomes similar to the studies by others.

Head trauma in children younger than 2 years: are there predictors for complications? Karen D Gruskin and Sara A Schutzman. (Harvard). Arch Ped Adol Med 1999 Jan; 153(1): 15-20. A retrospective chart review of the charts of 278 children under 2 evaluated in the ER for head injury.

227	minor injuries
39	uncomplicated skull fracture
12	intracranial injury

Their answer was that they could not isolate a complex of ER findings that would successfully predict the likelihood of serious complications. (Complications being LOC, emesis, sz). 278 patients had the following mechanisms of head injury:

236	falls (the falls are further substratified by type of fall, including 57 stairs and 20 dropped)
11^^	MVA
11	hit by a moving object
2	collided with object
3	unknown
14	NAI

The incidence of complications (LOC, emesis, Sz) did not differ between the patients with and without skull fracture or intracranial injury. The incidence of skull fracture/intracranial injury was much higher in the under-12 months age group (14% vs 1%) A low-height fall (under 3 feet) did not exclude complications. This is correlated with Duhaime's finding (100 infants, 1992) that while complications rarely resulted from low-height falls, skull fractures were as likely from low-height falls as from high-height falls. One death in entire study pop.

Literature review by David Chadwick in "Unified Response," the child fatality review team newsletter published by the Los Angeles County Inter-Agency Council on Child Abuse and Neglect, Winter 1999, p. 11. For address see end. He draws the following conclusions:

1. "Given the availability of prompt and appropriate medical care, falls contribute minimally to deaths in childhood. Death from a fall is now considered very unlikely when the fall is less than twenty feet, and accumulating experience may soon extend that.
2. "Epidural hematoma may occur as a result of a short fall and may cause death occasionally when care is delayed or the condition is not recognized.
3. "Each year pediatricians and emergency physicians see numerous infants and young children with skull fractures and short fall histories. Most ... recover uneventfully. ...
4. "Older statistics indicating that falls are an important cause of death in children less than five years of age may well be substantially contaminated by cases of inflicted injuries presented as falls."

The acute reversal sign: comparison of medical and nonaccidental injury patients. Rao P, Carty H, Pierce A. Clin Radiol 1999; 54: 495-501. Cited by Starling in her AHT chapter in Giardino & Alexander, Child Maltreatment, 3d Ed. (2005) for the proposition that interhemispheric SDH is the most characteristic of AHT as opposed to nonabusive HT.

Evaluation of infants with subdural hematoma who lack external evidence of abuse. Morris MW, Smith S, Cressman J, Ancheta J. Pediatrics 2000 Mar; 105 (3 Pt 1): 549-553. See above under "Shaken." Eval 9 ch w lone SDH (no other ev of abuse), because these cases "present a difficult challenge to child protection workers." In 8/9 pure SDH's without RH, bruises, or any other ev of trauma, ages 11 days to 15 months, SCAN investigation "uncovered the circumstances of the trauma," and led to a diagnosis of inflicted cerebral trauma. All 8 who had RH were found to be abuse. 1 without RH was UNDET. Lone SDH is usually abuse.

Childhood head injuries: accidental or inflicted? Reece RM and Sege R. Arch Pediatr Adol Med 2000 Jan; 154(1): 11-15. Retrospective chart review of 287 head trauma admissions to Tufts Univ Hosp aged 1 week to 6 ½ years, and thereby is able to compare 54 cases of definite abusive head trauma with 233 cases of definitely accidental head trauma. See <http://www.archpediatrics.com/>. This article uses a statistical probability approach to allow an anterior probability of a head injury being accidental or inflicted based on the type of injury.

All age groups:

<u>Type of injury</u>	<u>Abused</u>	<u>Acc</u>	
SDH	46% had (25/54)	10% had (23/233)	P=.001

SAH	31% “ (17/54)	8% “ (19/233)	P=.001
RH	33% “ (18/54)	2% “ (5/233)* **	P=.001
Skin injuries✓	50% “ (27/54)	16% “ (37/233) (!)	P=.001

* No RH in accidental falls of under 4 feet. “RH are, if not diagnostic, compelling findings; most are seen in abusive head trauma.”

** 18/54 AHT had RH, while 5/233 Acc HT had RH. Of these 5, 4 had obvious mechanism such as GSW face, fall from a great height, or MVA. Includes a discussion of RH. See also under “Walkers.”

✓ old and new skin injuries consistent with inflicted origin

Age under 3:

Type of injury

SDH	50% had	10% had	P=.001
SAH	33% had	9% had	P=.001
RH	35% had	3% had	P=.001

History given:

MVA	0%	23%
Fall	17%	58%
No history of injury	56%	0%
Infliction	24%	0%

History of a fall less than 4 feet -- all ages

<u>Overall</u>	15% (8/54)	27% (62/233)
----------------	------------	--------------

Of these,

Simple linear skull fx	50% (4/8)	61% (38/62)
Complex skull fx	12% (1/8)	8% (5/62)
SDH	38% (3/8)	8% (5/62)
SAH	38% (3/8)	2% (1/62)
RH	25% (2/8)	0% (0/62)

These numbers do not look all that discriminative. Other than RH, if a short-fall victim had one of these findings, how much anterior probability of abuse or accident could be assigned based on these numbers? Plus, the P values are not given, and are probably not in the range of statistical significance. –JKR

<u>Mortality</u>	13% (n=7)	2% (n=4)
------------------	-----------	----------

Walkers -- had 18 cases, all accidental, none fatal

Simple skull fx	72% (n=13)
Complex skull fx	22% (n= 4)
SDH	6% (n= 1)
SAH	0% (n= 0)
RH	0% (n= 0)

Includes a discussion:

Cites the following sources as having studied head injury -- accidental versus inflicted:

Caffey, 1946
 Kempe, Silverman et al., Battered child, 1962
 Guthkelch, 1971
 Caffey, 1972
 Caffey, 1974
 Duhaime et al., book, 1977
 Merten & Osborne, 1983
 Ludwig & Warman, SBS 20 cases, 1984
 Billmire & Myers, 1985
 Gennarelli & Thibault, 1985
 Hanigan, Tin ear, 1987
 Duhaime, Gennarelli et al., SBS, 1987
 Bruce & Zimmerman, 1989
 Hadley, Rekate, et al., Infant whiplash, 1989
 Alexander, Crabbe, et al., Serial abuse in shaken, 1990
 Alexander, Sato, Impact/ shaking, 1990
 Duhaime et al., very young children, 1992
 Levitt, Alexander, Smith chapter on AHT in *Reece*, 1st ed., 1994
 Gilliland & Folberg, Shaken -- no impact, 1996
 Taff & Boglioli, 1996
 Duhaime, Sutton, 1997
 Kleinman, 2d ed., 1998

Head trauma is the second leading cause of mortality in the pediatric age group, after MVA -- about 10/100,000. The third leading cause is leukemia. Gives figures for TBI in children:

MVA	37%
Falls	24%
Sports	21%

But in children under 1 year, 17% of all TBI and 56% of all serious TBI were inflicted. In another study, 24% of TBI under 2 years were inflicted. Abusive head trauma was more severe than accidental:

Injury Severity Scores of 20 to 75:

Abusive	22%
Accidental	6%

Cites Ewing-Cobbs et al. for the earlier data that abused had a much higher frequency of SDH (80% versus 45%) and of RH (70% versus 0%).

Conclusions:

1. The incidence of abuse in this population of serious head injuries was high, warranting a high clinical index of suspicion in the setting of a young child with a severe head injury. In the under-3 age group, 1/3 of all the patients were abused; if you take out motor vehicle accidents, it was 49%.
2. SDH, SAH, and RH were sentinel injuries for abuse. RH was nearly diagnostic.* There were 5 accident victims who had RH: of these, 4 had massive witnessed head trauma. "In a clinical setting, there would be no difficulty in differentiating these children from those whose injuries were caused by abuse."

* But in the paragraph on "caveats," the authors note that fundoscopy was not performed on all patients.

3. This study corroborates the previous findings that short falls do not cause serious injury in children, excepting epidural hematomas. "Subdural hematomas and SAHs are seldom seen and retinal hemorrhages are virtually never seen in short falls. When these lesions are seen, the veracity of the history of a fall is open to serious question." (citing Duhaime et al., Very young children, 1992; Chadwick et al., How far is fatal? 1991).

4. Walkers. Briefly reviews walkers. One death per year according to CPSC. 29,000 injuries in 1991. In our study, 83% of the walker head injuries were simple linear fractures and 22% were complex skull fractures. 16% had SDH. No RH, no deaths.

5. Outcomes were more severe in the abuse group: 6X mortality. Due to injury severity and possibly to delayed presentation.

6. Offers a clinical classification scheme:

Inflicted

Brain injury with

no history of trauma	OR
history of trauma incompatible with developmental level	OR
witnessed infliction	OR
confession	OR
other inflicted injuries	

Accidental

Brain injury with

history of trauma corroborated by more than one adult	AND
history of trauma compatible with developmental level	AND
no other inflicted injuries	

Child abuse and unintentional injuries: a 10-year retrospective. DeScala C, Sege R, Li G, Reece R. Arch Pediatr Adol Med 2000 Jan; 154: 16-22. See under Epidemiol. RH were 27.8% of abused vs .06% of accidentals. (The accidentals were overwhelmingly falls and MVA's.) RH were in 18 out of 54 abused chil and 5 out of 233 accidental chil: of these 5, 4 were GSW, fall from a height, or MVA. See Dr. Sege's discussion on Medscape at <http://www.medscape.com/medscape/pediatrics/>

Letter of Jan E. Leestma, MD, MM, April 12th, 2001 (in file) on possible own-height or high-chair fall versus AHT in a 16 mo infant at the baby sitter's who admits that she was angry at the baby and grabbed his ear. Saying that a fall from the infant's own height of 30 inches could develop over 300 g's of acceleration at the head and could cause massive SDH and RH. Saying that the pathogenesis of RH is unproven. See also Dr. Leestma's chapter in the textbook *Pediatric Neuropathology*, Serge Duckett, Ed. (1995).

AAP Policy Statement May 2001: Falls from Heights: Windows, Roofs, and Balconies (RE-9951) (have). 126 children under 14 were killed by falls from a height in 1998. Fatalities generally over two stories. Risk factors for fatal falls from a height include: a history of previous major unintentional injury to the patient or his siblings, developmental delay, seizure dso, hyperactivity, history of neglect, poverty, single-parent, inadequate child care, recent moves, illnesses, job changes. I should add lack of local legislation prescribing childproofing of buildings.

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 (mainly HIE). While limiting unnecessary imaging procedures. See the same authors' papers of 1999 and 1998.

Demonstration and interpretation of bridging vein ruptures in cases of infantile subdural bleedings.

Maxeiner H (Berlin). J Forensic Sci 2001 Jan; 46(1): 85-93. This guy used postmortem x-ray to demonstrate ruptured bridging veins, including counting the number of ruptured bridging veins, in postmortem cases of infants with both minimal and non-minimal subdural bleeding. States that "[T]he prevalence of several bridging vein ruptures combined with a SDB of [significant] volume, in an infant dead or in deep coma upon presentation, is not compatible with the supposition of a minor fall as the cause. We have not observed such findings as the result of a minor accidental event for more than 15 years."

Computer simulation of stair falls.

Bertocci GE, Pierce MC, Deemer E, Aguel F. Arch Ped Adol Med 2001 Sep; 155(9): 1008-1014. This is free-falls not walkers. Stair surface characteristics and slope affected the likelihood of upper-leg injuries in this unvalidated computer model. The purpose was to model stair falls as a possible mechanism of femur fractures in ambulatory children. Begins with the statement that "Unfortunately, it is often difficult to determine the validity of caregiver-stated scenarios, since so little is known regarding pediatric biomechanics and injury risk associated with these relatively common falls. A better understanding of the influence that specific fall-environment factors have on injury biomechanics in children is needed." "Although a limited number of studies describing relationships between the biophysics of a particular fall and associated injuries in children exist, additional studies are greatly needed to advance the forensic science to detect child abuse." This article is reviewed by Betty in *Child Abuse Quarterly* for January 2002. Betty says the authors' model predicts that the risk of femur fracture increases with "increasing slope, decreasing coefficient of friction,...and increasing number of steps fallen." She notes that this result contrasts with the empirical finding of Joffe & Ludwig (1988) to the effect that the number of stairs fallen did not affect injury likelihood or severity. [And I would add that it contradicts their conclusion that a stairway fall "is actually a series of smaller falls." --JKR] Betty comments that this paper "is very important and exciting," but notes that the authors' research up to this point has not attempted clinical correlation. She also points out that the fracture force threshold for the infant femur is not known. Also that the study did not include stair pitches steeper than 8:8. She considers that these findings can perhaps be reconciled with Joffe & Ludwig's conclusions on the following basis: (1) in Joffe & Ludwig's study, the number of bony injuries (25) was so small that it was not statistically possible to differentiate the effect of distance fallen on the occurrence of bony injuries. (2) Joffe & Ludwig's conclusion of lack of distance fallen effect referred to their whole study group, which was almost entirely (93%) soft-tissue injuries. What she doesn't say is that J&L's conclusion as to lack of distance fallen effect is unwarranted on their own data and potentially refuted by these authors' work.

Accidents and resulting injuries in premobile infants: data from the ALSPAC study. Warrington SA et al. Arch Dis Child 2001; 85: 104-107. 3357 falls in 2554 infants.

Fatal pediatric head injuries caused by short-distance falls.

Plunkett J. Am J Forens Med Path 2001 Mar; 22(1): 1-12. Reports 18 cases of fatal pediatric short-fall head injuries culled from US government databases. He presents them in ascending order of age, from 12 months to 13 years., many on playground equipment, but only 12 were witnessed by other than the caregiver. Fall distances ranged from 2 feet to 10 feet with most around 2 – 3 feet. Thirteen had SDH; of these, 7 had interhemispheric. None had DAI. Only six had any eye examination recorded, none by an ophthalmologist (but Dr Plunkett points out in later argumentation that the examiners were highly experienced pediatric intensivists); of these six, four (4) had RH; of these, 3 had bilateral "extensive," one had NOS. All the RH occurred in cases with SDH. He states that, "RH may be caused experimentally either by ligating the central retinal vein or its tributaries or by suddenly increasing intracranial pressure." (citing Smith, Kearns & Sayre, 1957 and Lehman, Krupin, Podos, 1972.) "Any sudden increase in ICP may cause RH." (citing Kirschner, "mistaken dx," 1985; Weedn, RH after CPR, 1990; David and Jain on bungee jumping, 1994.) "Vasospasm secondary to traumatic brain injury selectively increases venous pressure."

On biomechanics of SDH and TBI, (which is the core of his article), he says that "Research in TBI using [animal models] has shown that a force resulting in angular acceleration produces primarily diffuse brain damage, whereas a force causing exclusively translational acceleration produces only focal brain

damage. (citing Ommaya, 1995.) A fall from a countertop or table is often considered to be exclusively translational and therefore assumed incapable of producing serious injury. However, [it] must have an angular vector unless the force is applied only through the center of mass.” Therefore, argues that on theoretical grounds it should be able to cause an SDH if the magnitudes are right. [Note Wilkins’ earlier comment to the same effect, “True rotation might be rare in simple falls but explain rare cases of severe injury.” Wilkins, 1997. -- JKR] “An injury resulting in a SDH in an infant may be caused by an accidental fall.” (Citing Aoki, 1984; Greenes & Schutzmann, 1998; Howard, Bell, Uttley, 1993.) (In later argumentation, see Am J Forens Med P 2002 Mar, Dr Plunkett elaborates on this rotation possibility by commenting concerning the earlier studies of pediatric falls that, “[N]one of the other studies describes the position of the body at the beginning of the fall, the initial velocity, the part of the body that first struck the ground, or the characteristics of the impact surface. These cited studies do not allow even a kinematic analysis, let alone a kinetic evaluation.” This comment indicates that Dr Plunkett considers these biomechanical considerations forensically significant. --JKR) High-strain impacts (short pulse duration and high rate of deceleration onset), typical of falls onto hard surfaces, are more likely to cause SDH than low-strain impacts (long pulse duration and low rate of deceleration onset) typical of MVAs with deformable surfaces. [This supports Duhaime’s results (1992) but not Ewing-Cobbs’s results (1998) -- JKR.] The duration of deceleration for falls on a hard surface is usually less than 5 msec. Experimentally, impact duration longer than 5 msec will not cause SDH unless the angular acceleration is above $1.75 \times 10(5)$ radians per second squared. (citing Gennarelli & Thibault, 1982; I thought they said 3.5 msec. -- JKR) [“Strain” is deformation per unit length. “Stress” is force per cross-sectional area.]

This article is commented on by John Stirling MD in the July 2001 issue of *Child Abuse Quarterly*. Dr. Stirling makes the following points: 1. Dr. Plunkett is not the first researcher to point out that short falls can be fatal in small children; there have been many others. However, such fatalities are rare. Noting that Dr Plunkett obtained these 18 cases from a database of 75,000 accidents over eleven years. 2. Six of the falls were unwitnessed, and two others had preexisting medical conditions, so that leaves ten out of 75,000. 3. Most of the SDH’s in these cases were “the large or rapidly expanding type seen not infrequently with impact injuries.” 4. These were not infants. These are ambulatory children mostly in playground settings. This amounts to a “selection bias;” therefore the meaning of the data for infants is unclear since they were not represented in the sample. 5. The lucid intervals lasted less than one hour in all but four cases, usually 15 minutes or less; in the four remaining cases, two were complicated by cerebral infarcts, one was an EDH, and one had a large SDH with a “remarkable” 48-hour delay. 6. None of these patients had an ophthalmologic evaluation, so little can be concluded from the RH data.

This article is also commented on by Betty Spivack in Am J Forens Med Path 2001 Oct; 22(3): 332-334 and by Alex Levin in Am J Forens Med Path 2001 Dec; 22(4): 417, and Bethann Schaber in Am J Forens Med P 2002 Mar; 23(1): 101-103. See below. See also under “Time of Injury -- Head.”

See also another literature review at:

http://www.cincinattichildrens.org/programs_services/152/tools/falls.asp

Fatal pediatric head injuries caused by short distance falls. (letter) Alex V. Levin. (Comment on Plunkett, above). Am J Forens Med Path 2001 Dec; 22(4): 417. See also earlier letter by Betty Spivack in Nov issue.

Dr Levin first criticizes the RH data in Dr Plunkett’s article. Four of the 18 victims had retinal hemorrhages, none examined by an ophthalmologist. Dr Levin points out that the descriptions of the RH are difficult to interpret.

Second, Dr Levin states that the incidence figures deducible from the article are in line with previously reported figures for the incidence of RH in severe accidental head injury in children (3%); Dr Plunkett’s figures suggest an even lower incidence. Noting that Dr Plunkett had to search tens of thousands of accidental-fall records to find four cases with RH.

Third, as far as the “low-height” aspect, two of the four cases with RH were falls from swings; these Dr Levin says could be higher velocity and not mechanically consistent with a short-distance fall. As far as the other two, one had papilledema, which can give RH by itself. That leaves one other with “extensive” RH, “maybe this article reports an *extremely* rare circumstance where multiple factors coincide to allow RH (maybe even ‘extensive’ RH) to occur following accidental trauma that is less than that which is usually needed...”

Dr Plunkett responds to Betty and Alex:

1. Death in infant TBI is due either to (a) cerebral anoxia or edema secondary to focal injury, or (b) a space-taking SDH. For focal injuries causing fatal anoxia or edema, he posits (1) contact cortical contusion, (2) cortical laceration from fracture, (3) brainstem (or cord) contusion caused by either hyperextension or primary traumatic (mass-movement) herniation. He denies that death in infant TBI results from DAI. For these propositions he cites Oehmichen, 1998 (who found that AI-like changes were indistinguishable in anoxic brains and traumatic brains), and the three Geddes articles. [Therefore, he is implying, if no brainstem or cord contusion or primary hern is demonstrated, these deaths have to be from contact injury. If stem or cord contusion is demonstrated, then whiplash is implicated in causing respiratory arrest. He is also implying that SDH is caused [only] by contact injury in fall cases. Not clear whether he is referring to Hymel's focal contact SDH's and Hymel's diffuse angular-deceleration SDH's or both.]
2. The physics of a slam is identical to the physics of a fall. [Thus denying that there is any biomechanically significant radial component to a slam. The two mechanisms (fall and slam) cannot be biomechanically distinguished, therefore they cannot be pathologically distinguished as long as only a single impact is demonstrated.]
3. Says that pure shaking causing fatal TBI is physically impossible. Not just SDH, as previously argued, but any fatal TBI. Says that Ommaya's 1968 monkey experiment with pure impulsive loading used the lowest possible injury threshold (concussion) and needed accelerations of at least 10,000 r/s², which would be 5,000 ft/s² or 156 G, far in excess of shaking. Says that Caffey relied on this Ommaya paper in his 1974 shaken paper, and that Ommaya subsequently advised Caffey that he (Caffey) had misinterpreted Ommaya's data.
4. AS to RH, he argues that the correct denominator for RH/ fatal short falls is 4/18, not 4 over tens of thousands. Fatal short falls are rare. What we have is at least one third of them having extensive RH. As far as the quality of the eye examinations and descriptions, he says that the eye examinations were performed by a highly experienced neurosurgeon and a highly experienced pediatric intensivist, and one can draw one's own conclusions from there as to their analytical usefulness for scientific purposes.
5. He repeats his review of the mechanism of RH (see above) as either (a) central vein occlusion by vasospasm or edema or otherwise, or (b) increased ICP. States that "There is no evidence that 'shaking' can cause retinal hemorrhage..." "If someone has a theory for a cause of retinal hemorrhage different from those that I discuss in the study, then please do the appropriate experiments and prove it."

Fatal pediatric head injuries caused by short-distance falls. (letter) Schaber B, Hart AP, Armbrustmacher V, Hirsch C. Am J Forens Med P 2002 Mar; 23(1): 101-103. Makes the following points about Dr Plunkett's article (I have included Dr Plunkett's replies):

1. All of the youngest-age falls were unwitnessed. (the ones under 23 months). To this Dr Plunkett makes no reply.
2. The infant skull and brain have different biomechanical properties at different developmental ages, which are not taken into account when accidents in children of one year are lumped in with children of eight or ten years. To this Dr Plunkett replies that there is only one published study of the biomechanical properties of the infant skull (Margulies, Thibault, 2000), and therefore we don't know what these differences are. We can reasonably assume that there are differences, and it is precisely from studies like this that some of those differences may begin to emerge. By lumping the ages together he did not mean to imply that there are no differences or that all the accidents are equivalent, only to present the information for what it may be worth, as an answer to the contention of Williams and Wilman that fatal short falls and lucid intervals never happen.
2. Some of the younger-age cases did not have a full autopsy. (Suggesting that child abuse was not definitely ruled out.) Dr Plunkett acknowledges that the lack of autopsies limits the interpretation. He has never claimed otherwise. (He could point out, but doesn't, that some fatal child abuse cases have also lacked full autopsies.) He goes on to say that several of these non-autopsied cases came from medical-examiner jurisdictions in which they were "signed out" by physician chief medical examiners. He fails,

however, to develop the statistically interesting implication of this line of argument: that the absence of case reports of fatal falls and lucid intervals could be partly due to sampling bias. Witnessed accidents are simply signed out; only the suspicious deaths are autopsied. Therefore an autopsy population may be inherently biased toward suspicious deaths. This bias becomes more significant when you consider that both fatal abuse and fatal falls are statistically uncommon: a bias in a small sample is more difficult to correct for than the same bias in a large sample, because the differences between the sample groups are less well characterized.

3. As to lucid intervals, in the younger age group (under 5), there were only four cases that were both witnessed and autopsied. These had lucid intervals from none to 3 hours, as follows:

Case 6: no lucid interval. Side impact. Small localized SDH. DAI. Herniated from cerebral edema after 4 hours.

Case 5: few minutes lucid interval. Impact to the side of the forehead. Large SDH. Hern from cerebral edema after 12 hours.

Case 8: few minutes lucid interval. Complex fracture of the frontal-temporal bones. Cortical fracture contusions. EDH, SDH. Cerebral edema, hern on the second hospital day.

Case 9: 3 hour lucid interval. EDH.

The authors do not develop explicit conclusions from this, making only the two points that (a) lucid-interval histories have to be carefully correlated with the anatomic findings, and (b) in these four instances the correlation was good (“the lucid intervals were consistent with the neuropathologic findings at the time of autopsy.”) What they seem to mean by that is that the DAI case had no lucid interval, as would be expected, the two SDH front-impact cases had brief lucid intervals consistent with rapid bleeding and swelling, and the EDH case had a longer lucid interval as is common in EDH cases. (Others have made the point that EDH cases do not belong in this sample because their pathology is different; Wilman noted the EDH exception back in 1997. So what you really have, to develop Dr Schaber’s argument a bit further, is two documented independently witnessed cases of lucid intervals of a few minutes. These were in children aged 23 months and 3 years. –JKR)

3. Say that these falls were atypical of the usual infant falls dealt with by medical examiners because these falls possibly involved a rotational component for the head and brain. Criticize Dr Plunkett for not discussing this possible rotational component of the head/ brain in these falls from moving playground equipment. Pointing out Dr Hirsch’s earlier (1980) work arguing that in loss-of-balance falls the center of gravity becomes displaced relative to the feet, and it becomes a rotational fall for purposes of the head. (Dawson, Hirsch et al., HumP 1980; 2: 155). To this Dr Plunkett replies that (a) he did discuss this limitation of the data in his Appendix, and (b) unfortunately both in his data and that of all other articles, there is not enough information available to state a kinematic analysis of the falls. Thus the rotational-head issue is left open.

4. These accidents are rare. Dr Plunkett replies: (a) They may not be as rare as the raw NEISS numbers might make them appear; bear in mind that the NEISS is designed to capture all incidents, even those (the great majority) that involve no injury, only the potential for injury. (b) Whether they are rare or not is scientifically unimportant. No matter how rare they are, we need to understand how the injuries and lucid intervals happen, [because any forensic case can involve a rare event.]

5. These accidents involve unique scenarios (i.e. scenarios that would be easily distinguishable from the histories in possible child-abuse cases.) This point has been made by others. Dr Plunkett’s answer to it is that even so, understanding of less unique and more legally relevant scenarios could come out of attempting to understand the medicine and the biomechanics of these unique accidents (or at least acknowledging that they occur). He laments the fact that the biophysical data in these scenarios are inadequate for kinematic reconstruction and pathological analysis. There is nothing he can do about that; he doesn’t hide from it, he just says this is what the available information is.

Comment on Plunkett article, by a member of the SIGCA-MD-L listserv, late 2001.

(This came in as an attachment, and I have unfortunately lost the message it was attached to; there is no name on the document itself. JKR) This physician has gone through the Plunkett article and broken the cases out by age. They break out as follows:

Ages 0 to 1	0 cases
Ages 1 to 2	5 cases
12 m	unwitnessed, skull fx, SDH, no RH, no autopsy
14m	unwitnessed, cerebral edema with hern, no RH, no autopsy
17m	unwitnessed, SDH, edema, no RH, autopsied
20m	unwitnessed, occ fx, SDH, SAH, bilat multil RH
23m	witnessed videotaped fall, 5 min lucid, SDH, bilat RH, autopsied
Ages 2 to 3	3 cases
26m	witnessed SDH edema bilat multil RH, autopsied
3y	witnessed 3 ft fall cerebral edema, no autopsy
3y	witnessed 2 ft fall complex skull fx, cerebral contusions, edema
Ages 4 to 6	5 cases
4y	witnessed 7 ft fall EDH
5y	unwitnessed 7 ft fall SDH no autopsy
6y	unwitnessed 8 ft fall SDH edema no autopsy
6y	witnessed 10 ft fall malignant cerebral edema
6y	witnessed 3 ft fall SAH SDH edema infarct
Ages 7 to 14	5 cases
7y	witnessed 8 ft fall cerebral infarct sec to vertebral artery thrombosis
8y	witnessed 3 ft fall SDH
10y	witnessed 5 ft fall SDH AVM +bil RH
12y	witnessed 6 ft fall occ fx w contrecoup contus
13y	witnessed 6 ft fall occip fx, SDH edema

The author comments as follows:

1. Four of the five under 2 years were unwitnessed and Dr Plunkett admits that those injuries could be from some cause other than the reported fall, and that "it is possible that another person caused the nonobserved injuries."
2. The bilateral RH cases were of the following ages:
20mos 23mos 26mos 10y
3. The lucid intervals were based entirely on the histories, not on clinical observation.
4. Ambiguity as to whether the SDH's were the cause of death, or was it edema?

The cause of infant and toddler subdural hemorrhage: a prospective study. Feldman KW, Bethel R, Shugerman RP, Grossman DC, Grady MS., Ellenbogen RG. Pediatrics 2001 Sep; 108(3): 636-646. A prospective case series from both the hospital and the ME's office of 66 children under 3 admitted with SDH. They excluded patients with any predisposing factors for a bleeding diathesis or known preexisting cranial pathology. 39 (59%) were abuse, 15 (23%) were accidental, and 12 (18%) were undet. Mean age of abused was 8.7 months +/- 8.1, accidentals was 19 +/- 10. All accidentals were MVA or other well-documented major trauma. Long bone or rib fractures were found on 20 (51%) of the abused and only 1 of the accidentals. Chronic or mixed acute & chronic SDH were found only in the abused (44% had them) or the Undet (67%). RH was present in 72% of the abused children (all 39 abused children had eye examinations.) **RH was present in 1 out of 3 accidental children** who had eye examination, and it was typical of increased ICP (a few RH surrounding the optic disc in one eye only). The presenting history in the abused group was minor fall or no history of any trauma. Conclusion: **one fifth of SDH were nonabusive in origin.**

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. According to Carolyn Levitt's review in *Child Abuse Quarterly* for Jan 2002, this was a questionnaire survey to parents of infants under six months. Collected 3,357 short falls in 2,554 infants. Including 50 walker falls. 14% of all falls had visible injury. 1% had serious injury, defined as concussion or fracture. There were no cases of intracranial injury.

Lethal subdural bleedings of babies -- accident or abuse? Maxeiner H. (Berlin) Med Law 2001; 20(3): 463-482. Maxeiner is able to demonstrate torn bridging veins at autopsy. From this demonstration he can rule in trauma. He also analyzed his own material and Berlin registry material 1978-1999 and found that **“not a single death of a child due to an undoubted minor fall was recorded.”** From the abstract: the cause of SDH in infants “has become quite controversial in recent years.” Saying that some authorities have favored an accidental cause.

Injuries from falls in the pediatric pop: an analysis of 729 cases. Wang MY, Kim KA, Griffith PM, Summers S, McComb JG, Levy ML, Mahour GH. J Pediatr Surg 2001 Oct; 36(10): 1528-1534. The point of this article is that the customary EMS triage categories of “high-level” and “low-level” for pediatric falls are worthless; they do not predict injury severity or the frequency of intracranial injury. There is an increased frequency of severe organ injury with high-level. “Low-level” for EMS triage purposes means under 15 feet; in this article the authors state it includes “heights of one story down to 6 or 7 feet,” but in fact the studied material includes falls down to own-height falls. Figure 2 shows 180 patients with falls of 0 to 4 feet. Table 1 shows that 56 patients suffered own-height falls (52 “tripped,” and 4 fell during a seizure.) Children’s Hospital of Los Angeles’ trauma database. Retrospective chart review of 729 patients under 15 years old who were seen in the CHLA ER after a fall from any height; median age 2 years, age range less than three months up to 15 years. 393 low-level and 336 high-level. Mortality was 1.6%: (2.4% for high-level and 1.0% for low-level.) Found 12 deaths: 4 low-level. All 4 low-level deaths were from head injury. Table 2 shows that one low-level death was a 3 year old who tripped, had an orbital fracture, ocular injury, and **subdural hematoma**. As to high-level deaths, half were head injury and half other injuries. Low-level falls were mostly from playground equipment, also bicycles, windows, shopping carts, trees, being dropped, and falling down stairs, especially in walkers (8.5% of all cases). As to head injuries, “Interestingly, high-level falls were not associated with higher incidences of intracranial bleeding (8.6% v. 13.7%), suggesting that the deceleration energy necessary to produce intracranial bleeding in children is achieved before 15 feet.” **“The contact surface is likely a major factor** in predicting the extent of injury.” (citing Garretson et al., 1985). “Reichelderfer et al in a study of playground surfaces found that ... a 1-foot fall could produce a 475g impact.” (citing Reichelderfer et al., 1979). “Because of the proportionally greater cephalic mass in children, the likelihood of a cranial impact after a fall is increased.” (citing Kraus et al., 1986 and Durkin et al., 1998). “Nevertheless, child abuse must be suspected in all cases of severe head or bodily trauma from allegedly low-level falls.” (citing Chadwick, 1991). The authors state that in their low-level fall deaths a SCAN team workup was conducted and was negative for child abuse. Also that “In our study, cases of abuse were specifically excluded.”

Shows that GCS was a poor predictor of intracranial injury. Even with GCS 13-15, low-level falls had a significant likelihood of intracranial bleeding. A high index of suspicion and liberal use of CT. (See Adams et al., “Mandatory admission,” below.)

Coagulation testing in pediatric blunt trauma patients. Holmes JF, Goodwin HC, Land C, Kupperman N. Ped Emerg Care 2001; 17: 324-328. 830 ER pts with head or torso trauma. 28% had elevated coags.

This broke down into four subclasses:

- GCS less than 13
- Low BP
- Multiple fractures
- Large wounds

Mandatory admission after isolated mild closed head injury in children: is it necessary? Adams J, Frumiento C, Shatney-Leach L, Vane DW. J Pediatr Surg 2001 Jan; 36(1): 119-121. Usually these children are admitted even if there is no LOC and the CT is negative. We reviewed the National Pediatric Trauma Registry for a twelve year period, for age under 18 and GCS=15. 1,033 young people, average age 8y. Mostly falls, sports, and MVA. 386 had CT scans. None required neurosurgical intervention, and there were **no deaths**.

Playground injuries in children: a review and Pennsylvania Trauma Center experience. J Soc Pediatr Nurs 2001 Jan-Mar; 6(1): 11-20. Had 234 children aged 1 to 18 years with 421 injuries. Falls from playground equipment were 73% of all cases.

Upper extremity	117 injuries
Head	110 injuries

Recommends protective surfacing.

Current concepts: diffuse axonal injury-associated traumatic brain injury. Peduzzi D, Eleftherious E, Novack TA. Arch Phys Med Rehab 2001; 82: 1461-1471. See Mary Case's review in *Child Abuse Quarterly* for July 2002.

The potential and limitations of utilising head impact injury models to assess the likelihood of significant head injury in infants after a fall. Cory CZ, Jones MD, James DS, Leadbeatter S, Nokes LDM. (Cardiff) FSI 2001; 123: 89-106. Reviews published child biomechanical head injury models based on engineering principles, such as those of Duhaime et al. (J Neuros 1987; 66: 409) and Nokes et al. (FSI 1995; 79: 85) to calculate the severity of a head impact from a fall. Gives the state-of-the-art model called HIM (head injury model.) Addresses the basic engineering principles and mechanical factors to be considered. See Kent Hymel's review in the *Quarterly* for Jan 03: Contains a primer on the engineering principles. See the same authors' reconstruction of an alleged fall SDH in 2003, below.

Intracranial hemorrhage in children younger than 3 years: prediction of intent. Wells RG, Vetter C, Laud P. Arch Pediatr Adol Med 2002 Mar; 156(3): 252-257. From the abstract: "To determine whether certain CT imaging patterns ... help predict intentional compared with unintentional injuries." Retrospective case series over a 10 year period. 293 children under 3 with ICH. They somehow knew after the fact (presumably from final clinical or medicolegal evaluation) whether the injuries were or were not intentional. They found that they could achieve a SPEC of 83% (with a 95% confidence interval 74% to 89%) by using a combination of four CT variables:

- convexity SDH
- interhemispheric
- hygroma
- absence of a skull fracture

Wilbur Smith's review in *Child Abuse Quarterly* for July states that the authors found certain injury patterns were "highly sensitive and specific" (Dr. Smith's words) for diagnosing victims of abusive head injury. Dr Smith notes, however, several problems:

- a. The criteria for finally determining abuse were subjective
- b. There is interobserver variability in interpreting hemorrhage on CT scans
- c. MRI was not used, even though it is "much more sensitive."
- d. *The authors could not distinguish between accident and abuse in 12% of their cases.*

Neuropathology of inflicted head injury in children. Parulekar MV, Elston JS. Brain 2002 Mar; 125(Pt 3): 676-677, comment and authors' reply at 678. See also Clark BJ, Adams GG, Luthert PJ, Retinal haemorrhages in infant head injury. Brain 2002 Mar; 125 (Pt 3): 677-678.

Non-accidental head injury in children: gathering the evidence. Stoodley N. Lancet 2002 Jul 27; 360(9329): 271-272.

Unexplained subdural hematoma in young children: is it always child abuse? Fung EL, Sung RY, Nelson EA, Poon WS. Pediatrics International 2002 Feb; 44(1): 37-42. (Hong Kong). Asking, "To what extent is the proposed pathognomonic association between unexplained SDH/RH and child abuse a self-fulfilling prophecy?" Retrospective review of the hospital records of nine ch under 2 y adm w unexpl SDH. Four had no other injuries. Five had RH. One had multiple bruises. All survived but with poor neurological outcomes. **FOUND: in three cases, complete investigation failed to substantiate any abuse.** [The abstract does not say which three cases. -JKR] Does this mean that we missed the abuse? Or could trivial injury have caused these? "Despite a magnitude of opinion to the contrary, the issue of whether 'trivial'

head injury can cause SDH and/or RH is yet unresolved. Clearly much more information on this very sensitive and serious issue is required and these data shd be collected with an open mind.”

Some pitfalls of computer modelling. Thibault KL. Arch Pediatr Adol Med 2002 Mar; 156(3): 296-297.

Head injuries in infants: the risks of bouncy chairs and car seats. Wickham T and Abrahamson E. (London) Arch Dis Chil 2002; 86: 168-169. This British prospective study of 131 infants mean age 6 mos presenting to ER with a hx of head injury. 114 pts had skull series, not reported if any had CT scans. The histories were:

Fell off a surface	52
Fell out of a bouncy chair or car seat	17
Fell off a push chair	16
Fell over	10
Dropped	15

Categorized as to whether landed on a hard surface or not, whether the source was on an elevated surface or not. Apparently sx were rare, a few skull fxx, no serious injuries.

Retinal findings in children with intracranial hemorrhage. Schloff S, Mullaney PB, Armstrong DC, Simantirakis E, Humphreys RP, Myseros JS, Buncic JR, **Levin AV**. Hospital for Sick Children, Toronto. Ophth 2002 Aug; 109(8): 1472-1476. Includes **two cases of short falls causing intracranial hemorrhage** in small children -- one toddler fell out of a stroller, and one ?infant was dropped from mother's arms. No specifics on the type of intracranial hemorrhage in these cases, other than that no RH was present. See under RH -- Terson's.

Biomechanics and neuropathology of adult and paediatric head injury. (review) Ommaya AK, Goldsmith W, Thibault L. Br J Neuros 2002 Jun; 16(3): 220-242. Professor Goldsmith, a retired Berkeley physics professor, is Dr. Plunkett's biophysics mentor and *eminence grise*. In this article he has been lured out of retirement to defend Dr Plunkett's position regarding short falls. From the abstract: "Scaling of tolerances for skull failure and brain injuries in infants, children, and adults are developed.... Head injury tolerance levels at three age categories for cerebral concussion, skull fracture, and three grades of DAI are presented. Brain mass correlates inversely for TBI caused by angular head motions and locations of injurious stresses are predictable by centripetal theory.... Clinical similarities of human TBI patents [to experimental animals] do not necessarily predict equivalent biomechanics because such trauma can be produced in various ways. We recommend 'reverse engineering' for in-depth reconstruction of the TBI injury mechanism for qualitative diagnoses and reduction of outcome variability." [By JKR: basically what they're saying is that engineering reconstruction from the clinically observed primary injuries can exclude various mechanisms, or show that they are not excluded, based on presumed tissue tolerances obtained by scaling animal tissue-tolerance data.]

This art is unfavorably reviewed by Drs Christian and Duhaime in *Quarterly* for January 2003. According to their review, the paper concludes that

1. Falls, including trivial falls, are a leading cause of clinically significant TBI in infants and children.
2. Shaking probably cannot cause SDH.
3. Neck injuries would be expected from shaking. [Note, not just cervical cord injuries, but neck injuries --JKR]
4. Short falls and other impacts produce forces 3-4X greater than shaking.
5. Flexible infant skull causes large impact strains throughout the infant head and its contents.

6. Retinal hemorrhages are caused by rapid rise in ICP.
7. Rebleeds of previously innocuous chronic SDH's cause sudden decompensation.

The reviewers make the following debaters' points:

1. The authors' CDC data on falls actually show that the TBI mortality of pediatric falls was 0.4 per 100,000 (12 deaths), compared to an assault mortality of 1.5 per 100,000 (39 deaths). The reviewers cite this flaw to show that the authors are mistaken about the morbidity of falls in the young. But are they? Twelve fall deaths is not a trivial number. It's about one-third of the assault deaths, which could at least superficially be taken to imply that one-third of AHT fatalities alleged to be falls could actually be falls. In other words, while refuting the claim that falls were "a leading" cause of TBI, the reviewers have not succeeded in refuting the claim that falls were a *significant* cause of TBI. Also, I note that no less an authority than Jan Bays has said that falls are the third leading cause of traumatic death in children 1 to 4. (In Reece & Ludwig, 2d ed., p. 193).

2. The authors misquote the Duhaime shaking experiment as a six-month-old shaken only, instead of a one-month-old shaken with impact. (Odd, since they were involved in designing that experiment.) True enough, they do misquote the study in these respects. But does it matter? The point remains that the Duhaime study produced evidence that the shaking portion of the experiment did not develop enough angular acceleration to reach the monkey SDH threshold.

3. As to falls causing SDH, the authors misquote the 1994 study by Berney et al. (see above) on "low-energy" impacts as showing that low-height falls caused a significant number of SDH's. In actuality, the Berney study found only four SDH's in the 0-3 age group, and three of these were from injuries classified as "miscellaneous," which included child abuse.

4. The authors misquote a paper by Sargent et al. to the effect that 92% of abused children had soft tissue injuries (implying that if it's SDH alone, it probably isn't child abuse.) The reviewers point out that Sargent's paper showed nothing of the kind; it is a single case report that contains no data about soft tissue injuries one way or the other.

Comment by JKR: The reviewers have successfully shown that Ommaya et al. misquote previous studies. But as the reviewers acknowledge, the data on all of these questions are incomplete, and the questions "have been debated for years, largely and precisely because data remain inconclusive and controversial." Now *that* is an accurate assessment.

This article cited by Geddes et al., Reply to Punt, 2004 as one of two arts providing biomechanical support for the view that pediatric short falls can be fatal (the other one being Goldsmith & Plunkett, 2004).

Subdural haematoma and non-accidental head injury in children. (UK) Hoskote A, Richards P, Anslow P, McShane T. Child's Nervous System 2002 Jul; 18(6-7): 311-317. A retrospective series of 36 children referred to neurology or neurosurgery for SDH.

Witnessed trauma	9
Iatrogenic SDH	4
Suspected child abuse	23
After full workup of these 23 cases, found	
Definite child abuse*	14*
Found to be accidental	2
Found to be natural (lateral sinus thrombosis)	1
Still unexplained	6

* Confirmed by thorough medical and social assessments

The 14 definitely abused had these findings:

RH	85%
+ skeletal survey	61%
age 1 to 4 months	85%
bilateral SDH	78%
interh	43%
loss of gray-white diff	43%

Of the 6 unexplained SDH's, had:

Neonatal problems	5/6
Older than 5 mos	5/6
<u>Chronic</u> bilateral SDH	5/6 (emphasis added --JKR)
No RH	6/6
Negative skeletal survey	6/6
Noncontributory social services w/u	6/6

Identified the following risk factors for having abuse ruled in: age less than 12 weeks; inconsistent history; RH; positive skeletal survey; unexplained bruises. The predictive value of having these risk factors was:

One risk factor	82%
Two risk factors	93%
Three risk factors	100%

Concluded: SDH due to NAHI tends to present before 4 months of age with an inconsistent history; the pts are more seriously ill and have other findings, such as fractures and RH. A small subgroup of pts was identified who had isolated old SDH and in whom full investigation remained inconclusive..."

Infantile subdural hematomas due to traffic accidents. Vinchon M, Noizet O, Defoort-Dhellemmes S, Soto-Ares G, Dhellemmes P. Pediatric Neuros 2002; 245-253 (Lille). Had 18 infants with CT scans and 16 had ophth. Three had RH, all flame hemorrhages limited to the posterior pole. Neurol:

64%	SDH
44%	interh SDH
44%	cerebral contusion

Hyperdense blood lasted up to ten days. Mixed hyper- and hypodense blood was seen in some initial scans, contradicting the view that mixed hyper- and hypodense equals old and recent injury.

Incidence and description of high chair-related injuries to children. Powell EC, Jovtis E, Tanz RR. Ambulatory Pediatrics 2002 Jul-Aug; 2(4): 276-278. NEISS data for chil 3 and under 1994-98 sh 40,650 high-chair-related injuries tr in ER's. 94% resulted from a fall from the chair, with 44% head, 39% face. Injuries:

contusions or abrasions	36%
lacerations	25%
closed head injury	21%
fractures	8%

Two percent were admitted. Most injuries were in the first year of life.

Can the initial history predict whether a child with a head injury has been abused? Hettler J and Greenes DS. Pediatrics 2003 Mar; 111(3): 602-607. To determine the diagnostic utility of certain features of the presenting history. Retrospective review of 163 chil 0-3 admitted for intracranial injury (note: not head

trauma in general, but intracranial injury) over a seven year period. They excluded all known natural causes. 49 of them (30%) were definite abuse. **Having no history of trauma** had a high specificity (.97) and a high PPV (.92) for abuse. Another predictive historical element was blaming injuries on CPR: seen in 12% of definite abuse cases and 0% of not definite abuse cases. Changing history: 9% of definite abuse, 0% of not definite abuse.

<u>thing</u>	<u>SPEC</u>	<u>PVP</u>
no history of trauma	.97	.92
persistent neurol abn at discharge w hx of no or mild trauma	1.00	1.00
CPR offered as cause	1.00	
changing history	1.00	
RH		
moderate or high-specificity fractures		
high-specificity skin marks		
delayed presentation	low	low

Subdural hemorrhage sustained in a baby rocker? a biomechanical approach to causation. Jones MD, James DS, Cory CZ, Leadbeatter S, Nokes LDM. FSI 2003 Jan; 131(1): 14-21. (Cardiff) From the abstract: "An unconscious 8 weeks old infant was admitted to hospital and found to have bilateral subdural and retinal haemorrhages. He died the following day. The explanation for the subdural haemorrhage put forward by his carers was that the infant had been in a baby-rocker and that the carers had seen the rocker being rocked vigorously by their 14 months old daughter on two separate occasions." Kent Hymel's review in the *Quarterly* for July 03 relates that the authors used an engineering model to mathematically reconstruct an alleged accident. Conclude that the fatal SDH **could not have happened by the mechanism given in the history**. Favorably reviewed by Hymel bc it respects the limitations of engineering models here. Gives a good discussion with diagrams and formulae. See Cory CZ, 2001, above.

Injuries when children reportedly fall from a bed or couch. Hennrikus WL, Shaw BA, Gerardi JA. Clin Orthop 2003 Feb; 407: 148-151. Had 115 pts seen for orthopedic injuries following such a history. 113 fractures, 2 impalements. Overall, found that 95% of the injuries were explained by the history and 5% were not. But of the four children under 1 year of age, 50% were abusive. Conclusion: Orthopedic injuries attributed to a child falling from a bed or couch are usually accidental **unless the child is younger than 1 year**.

Unexplained subdural hematoma in young children. Lee AC. Pediatrics International 2003 Apr; 45(2): 220.

Anthropomorphic simulations of falls, shakes, and inflicted impacts in infants. Prange MT, Coats B, Duhaime A-C, Margulies SS. J Neurosurg 2003 Jul; 99(1): 143-150. Duhaime's group built a new dummy of a 1 ½ month old infant with a 1.13 kg head and a 9.2 cm radius of rotation to the cranial center of gravity at a 10.6 pound total body weight. For a complete summary, see under "Shaken." They shook the dummy, slammed it after shaking, and also dropped it 1 foot, 3 feet, and 5 feet onto three different surfaces (concrete, carpet pad, and foam mattress). They recorded rotational velocity (directly recorded), and calculated

maximum peak-to-peak change in angular velocity (delta theta max)
peak angular acceleration (dtheta/dt max)

Significant increases in both of these parameters were found with both higher height and harder surface. Inflicted impacts had significantly larger values than any falls. CONCLUSIONS: (From the abstract) "Vigorous shakes of this infant model produced rotational responses similar to those resulting from minor falls, but inflicted impacts produced responses that were significantly higher than even a 5 foot fall onto concrete. Because larger accelerations are associated with an increasing likelihood of injury, the findings

indicate that inflicted impacts against hard surfaces are more likely to be associated with inertial brain injuries than falls from a height less than 5 feet or from shaking.”

[Comments by JKR: (1) Their biomechanical experiment with dropping a model infant 3 feet and 5 feet onto hard surfaces achieved instantaneous head accelerations that are within the range for SDH and DAI found in earlier primate experiments. They conclude that the possibility of serious brain injury from such short falls onto hard surfaces is not excluded by their experiments. (2) In light of other articles and discussion on the possible rotational component occurring in vertical falls, it is of interest that these workers found significant rotational acceleration of the head in their drop model.]

Commented on in letter, J Neuros 2004 Mar; 100(3): 574-575.

Child head injuries: review of pattern from abusive and unintentional causes resulting in hospitalization.

Brown GW, Malone P. Alaska Med 2003 Jan-Mar; 45(1): 9-13. Reviewed the records of 85 children 0-4 years admitted in Vermont with head injuries. Mean age 19 mos. 73 accidentals and 12 abuse.

Mechanisms:

falls	53%
MVA	17%
AHT	14%

Had three deaths, all from MVA. Found that 42% of abuse suffered serious CNS injury compared to only 10% among the accidentals.

Childhood injuries due to falls from apartment balconies and windows. Istre GR et al. (Dallas) Had 98 victims aged 0-15 years. 40% required hospital admission. Most apartment falls occurred around lunch or dinner time. Many children slipped between balcony rails, because the rail spacing was found to be more than 10 cm (4 inches). The average spacing was 7 ½ inches (19 cm). Most of the windows were located less than 2 feet above the floor. These were older apartment buildings not subject to current building codes.

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. See under “Time of injury -- head,” below.” Case of a 9-month child who fell 30” off a bed onto a concrete fall and suffered a delayed death after a lucid interval of 64 hours. Had a linear parietal skull fracture with a small underlying fracture-related SDH and focal axonal injury (small tear) in the corpus callosum only, plus brain swelling. 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. Cited by Geddes et al., 2004, as one of the three arts showing that pediatric short falls can occasionally be fatal. (The other two being Kim, Wang, 2000 and Plunkett, 2001).

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:

falls

asphyxia
 strang
 hang
 window cords
 clothing drawstrings
 susp
 wedg
 facial occlusion
 overl
 falling TV's
 widened subarachnoid or subdural space
 GA-1
 AVM
 Dural sinus thrombosis
 Coagulopathies
 HDN
 Hemophilia
 VWD
 ITP
 Leukemia
 Brain tumor
 Prenatal trauma
 Perinatal trauma
 Encephalitis
 DPT
 Phenylpropanolamine-induced intracerebral hemorrhage
Caida de mollera

And here is the differential diagnosis of retinal hemorrhages:

Increased ICP
 NSVD
 Purtscher's syndrome/ CPR
 Seizures
 Meningitis

Now, as to the differential diagnosis of head injury, Dr Reece briefly summarizes the conclusion of the literature as to each diagnosis as follows:

Falls. The extreme rarity of fatal falls in small children is emphasized. Plunkett found ten fatal toddler falls (and no infants) out of 75,000 short playground falls in the CPSC National Injury Clearinghouse.

Asphyxia. Dr Reece notes that shaking and strangulation are intentional mechanisms of brain anoxia that place asphyxia in the differential diagnosis of inflicted head injury, particularly at the clinical level. He notes the following studies: Window cords: Rauchsvalbe & Mann, JAMA 1997 found 183 fatalities. Clothing drawstrings: Drago et al., 1997 found 8 fatalities. Suffocation: Drago & Dannenberg, 1999 found 2178 cases in 1995, with the following mechanisms: wedging 879, facial occlusion 512, overlaying 180, suspension 145, hanging 142. Byard et al., 1994 with similar findings.

Falling TV's. He notes Bernard et al., 1998 finding 73 injuries and 28 deaths, and DiScala et al., 2001 finding 183 injuries and 5 deaths.

Dr Reece's summary of the fall vs. inflicted literature. "...after excluding motor vehicle crashes and witnessed long falls, it is seen that most cases of serious and fatal childhood neurotrauma are abusive in origin. It is extremely rare for short falls to cause serious or life-threatening intracranial injuries in infants and young children based on the available peer-reviewed literature." (p. 19)

Other DDX under "Predisposing medical conditions:"

Widened subarachnoid or subdural space. Quoted Mark Dias to the general effect that there is nothing to this.

GA-1. An unremarkable early history followed by the development of a dystonic/dyskinetic syndrome in infancy, with or without macrocephaly. Frontotemporal atrophy, changes in the basal ganglia, white matter hypodensities, hydrocephalus, and sometimes subdural effusions. (citing Woelfle et al., 1996) SDH may be the initial presenting sign, and RH has been described. Cites Morris et al., 1999 and Hartley et al., 2001 to the effect that with innocent SDH due to GA-1, there will be no fractures or other associated injuries.

AVM's. Although rare in infants (1% to 2% of all reported AVM's), *they can bleed following trauma.*

Dural sinus thrombosis. This rarity usually occurs in sick neonates with systemic illness.

Coagulopathies.

HDN. Develops at 3 to 6 weeks. This can be caused by the following things: exclusive breastfeeding, maternal malnutrition, maternal drugs (anticonvulsants, antibiotics, or anticoagulants), chronic diarrhea in the infant, alpha-antitrypsin deficiency, hepatitis, cystic fibrosis, sprue, and other malabsorption. Up to 50% of the patients have intracranial hemorrhage. Mentions the case reports of ICH from Ryan & Gayle, 1992, Wetzel et al., 1995, Soylu et al., 1997, and Rutty et al., 1999.

Hemophilia and VWD. Dietrich et al. found 5 cases of intracranial bleeding in 49 hemophiliac children. Dr Reece observes that none of these cases would have been confused with inflicted head trauma, because (a) most of them had hemophilia A, (b) two had epidural hematomas, and (c) two others were stairway falls.

ITP, leukemia and brain tumors. Cites the review by Medeiros et al. of 300 cases of ITP children, finding that only two ever had intracranial hemorrhage from it and both recovered uneventfully. One case report exists of a leukemic who presented with SDH. And one case report by Kaplan, 1986, of a SDH due to a brain tumor that was initially misdiagnosed as abuse. [Kaplan JM. Pseudoabuse: the misdiagnosis of child abuse. JFS 1986; 31: 1420].

Intrauterine SDH. Usually trauma, but Akman & Cracco reporting 7 cases of spontaneous intrauterine SDH and 24 cases caused by medical risk factors such as liver disease, thrombocytopenia, and other coagulopathies.

Birth trauma. Vacuum extraction as well as forceps are known causes of subdural hematoma and intracerebral hemorrhage. Citing the reports of Towner et al., 1999, finding in a large retrospective review that up to 0.25% of vacuum or forceps deliveries result in SDH or ICH, the report of Doward & Sgouros, 2001, reporting two cases of SDH due to vacuum extraction, and Odita & Heibi reporting that 69% of their series of 16 newborns with ICH were due to vacuum extraction.

Encephalitis. Points out that Herpes simplex encephalitis can be associated with intracranial hemorrhage, but should not be confused with AHT.

DPT. No

Phenylpropanolamine. One case report.

Caida de mollera. Cites the report of Hansen, 1997. [Although this has never been replicated. -JKR]

Now, as to the DDX of RH, Dr Reece summarizes literature as follows:

Increased ICP. Cites the reports of Muller & Deck, 1974, Khan & Frenkel, 1975, Lambert, Johnson & Hoyt, 1986, Munger et al., 1993, McRae et al., 1994, and Emerson et al., 2001. Says that the problem with this line of argument is that RH are rare in nonabuse cases of increased ICP, and even when present are few and posterior. (citing Levin in Reece & Ludwig, 2001).

Birth hemorrhages. Up to 45% of vaginal deliveries, citing Levin and Emerson et al., 2001.

Purtscher's syndrome/ CPR. CPR does not cause RH. Citing Kanter, 1986, Gilliland & Luckenbach, 1993, and Odom et al., 1997.

Seizures. Citing Sandramouli, 1997 (33 children with sz, no RH) and Tyagi et al., 1998 (32 children with sz, no RH).

Meningitis. Case report by Fraser et al., 1995 of one case of a 12-year-old with meningococcal meningitis who was found to have a large subretinal hemorrhage with vitreous hemorrhage. The three responses are as follows:

Ken Feldman: Emphasizes the difference between impact injuries and impulsive injuries. Unfortunately, the clinical literature does not categorize injuries that way; it lumps all SDH together. SDH comes in two kinds: contact SDH and impulsive SDH. The kind most often seen in child abuse is the impulsive SDH.

“The literature on childhood falls quoted by Reece suggests that few children sustain significant head injury from short falls. However, cases of mostly clinically insignificant contact SDHs are now being reported. (citing Duhaime et al., Disappearing subdural hematomas in children, 1996, and Feldman et al., The cause of infant & toddler subdural hematomas, 2001.) Further, a few contact injuries from short falls may be fatal or have residual effects. (citing Feldman et al. and Plunkett, 2001.)” Points out that Plunkett’s data “does not document that impulsive SDH’s (associated with diffuse or multifocal brain injury) commonly seen in abuse are likely after minor falls.” [That sentence is carefully worded. Plunkett’s article is vague about what kind of SDH’s many of them were. Other scholars have said that most of his SDH’s appear to be space-taking lesions, but that is by inference. Hence Feldman’s qualified wording here. –JKR] As to the shaking mechanism, Feldman urges more biofidelic dolls, and also the need to document independently witnessed shaking events. As to time of injury, he describes his own retrospective chart review of MVA children, which was inconclusive in finding a pattern of instant LOC (concussion) in those with and without SDH.

Robert Sege: Points out serious weaknesses in the existing literature and research on AHT. (1) No control groups (of unintentionally head-injured children). (2) biased testing: patients with suspected abuse are more intensively imaged and eye-examined. Unintentionally injured children generally do not get eye examinations, bone surveys, MRI, etc. (3) Racial discrimination in evaluating for abuse; nonwhite children are more likely to get worked up for abuse. (4) Little organized evaluation of the sensitivity and specificity of specific diagnostic tests in pointing to the likelihood of inflicted neurotrauma being present. Says that because of these weaknesses, we don’t yet have the ability “to describe, with some certainty, the likelihood that a given constellation of physical findings is indicative of abuse.” (p. 41) Says that the diagnosis of inflicted childhood neurotrauma currently (and as used by Reece in his essay), “hinges critically on three findings: (1) **a history of confessed abuse, or, more often, a history that is inconsistent, developmentally inappropriate, or describes a mechanism insufficient to account for the injuries noted;** (2) **a constellation of clinical and roentgenographic head trauma findings characteristic of abuse;** and (3) **other associated physical findings (e.g. posterior rib fractures) that are typical of battered children.**” (p. 39).

Cindy Christian: Misdiagnosis is a significant problem in AHT, in both directions. Intentional neurotrauma is missed and unintentional neurotrauma is mistakenly attributed to abuse. We don’t know how often this happens. Discusses the natural diseases that are misdiagnosed as inflicted neurotrauma, especially congenital and acquired coagulopathies. Also discusses secondary coagulopathy that occurs *because of* intentional or unintentional head injury. We (hematologists) should develop recommendations for hematologic screening of apparently head-injured infants “based on the diseases known to cause spontaneous intracranial hemorrhage in infants.”

Another one is the literature summary by Robert A. Zimmerman, MD, entitled “Radiographic evaluation of inflicted childhood neurotrauma,” which is followed by responses from Mark Dias (pp 93 – 121), Paul Kleinman (pp 123-124), and Wilbur Smith (pp 125-126).

Dr Zimmerman starts out by saying that CT is good for SDH and SAH and adequate for fractures and “the hypodensity of parenchymal injury.” MRI in its recent speeded-up variants of turbo-spin-echo-T2-weighted and echoplanar diffusion-weighted, “has become the most sensitive modality to fully assess intracranial injury, including extraaxial collections and hemorrhages other than SAH...” As to CT, he says that the scanners are getting faster, with the next generation to be capable of 16 slices per second. Zimmerman discusses the radiographic incidence of various types of head injury in child abuse. As to skull fractures, he recites the incidence of Merten et al. 18.8%, Duhaime et al. 43%, Hobbs 33%, Leventhal et al. 27%. But he says that skull fracture correlates poorly with intracranial injury in the abuse setting. In his own study of 26 abused children with significant intracranial injury, 77% did not have a skull fracture. As to SDH, the most common intracranial bleeding in abuse, he recites the incidence of Dashti et al. 69%, Duhaime et al. 16%. His own work (1979) found interhemispheric in 65% of 26 AHT cases. EDH is rare in abuse, citing Ewing-Cobbs, Duhaime, and Dashti. As to SAH, it is almost always combined with SDH, which makes it hard to detect radiologically due to the mass effect or other imaging effects of the SDH. Duhaime et al found isolated SAH in only 3/100, while combined SAH/SDH in 16. As to ICH, he says that three things can cause ICH in AHT: contusion, DAI, and rupture of a vessel. The results of Ewing-Cobbs and Hymel indicate a low incidence of ICH from any mechanism: 3/31 and 1/39. As to swelling/edema/infarction as radiologically demonstrated, he breaks it out into CT reports and MRI reports. On CT, it is seen as low density. The incidence was Ewing-Cobbs 11/31, Dashti 5/32, Zimmerman 17/17

interhemispheric SDH cases, Johnson et al. 20/28 as shown on the first available CT(!) Johnson et al. found a strong association of diffuse hypodensity with early hypotension/hypoxia. Goes into the findings of Gilles & Nelson (1998) who found different findings in the younger and older age groups: the group with a mean age of 5 months had diffuse hypodensity, while the group with a mean age of 19 months had focal hypodensity. Infarction was present in all, either hemispheric necrosis or in arterial distributions (4 PCA, 4 callosal marginal branch of ACA, 4 border zone). As to MRI of edema, DWI was used in two studies: Suh et al (2001) and Biousse et al (2002). Suh found that DWI revealed more extensive brain injury than standard MRI. Biousse found that all 18 SBS infants had an abnormal DWI; in 13 of them DWI showed more extensive lesions than MRI, indicative of ischemia. Let's not get into MRS. Geddes et al by neuropathology found DAI in only 3/37 while hypoxic injury in 28. As to cervical spine imaging, he first refers to Geddes finding (2001) of cervical spine injury by pathology in 11 cases, "Thus, this may be a site of injury that produces central apnea and subsequent hypoxic/ischemic brain injury." (p. 87) [The "unified hypothesis" --JKR] Hadley et al (1989) found injuries of the cervicomedullary junction in 5/6 SBS cases by clinicopathologic study, consisting of SDHs, EDHs, or cord contusions. But Ken Feldman doing MRI (1997) found NO evidence of cervical cord injury or SDH in 12 SBS infants, but did find diffuse SAH of the cord in 1/5 fatal cases by autopsy. Zimmerman goes on to identify "areas of ambiguity in radiography of childhood neurotrauma." He identifies the following areas of ambiguity:

1. 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; Vinchon et al., J Neurosurg (Pediatr I) 2004; 101: 44, summarized in the *Quarterly* for January 2004 and Spring 2005, respectively. --JKR]

Dr Dias's response is a literature review that presents all the radiology articles in tabular form as well as summarizing them. On the issue of "Can we identify radiographic features that specifically differentiate intentional infant neurotrauma from unintentional trauma in this age group?", Dr Dias summarizes the following articles:

1. Billmire & Myers, 1985. Dr Dias summarizes that they described 84 infants -- 54 unintentional and 28 inflicted head injury. Skull fractures were more common in the unintentional group, while intracranial injuries were restricted to the inflicted group except for one automobile accident in the unintentional group.

2. Duhaime et al., Head injury in very young children, 1992. Dr Dias relates that these authors studied 100 consecutive head trauma admissions of children under 24 months, finding 24 definitely inflicted and 76 unintentional or suspicious cases. The falls they divided into greater than or less than 4 feet. All intracranial injuries from falls were over 4 feet. In the unintentional group, all EDH's were falls under 4 feet. All unintentional SDH and SAH and cerebral contusions and severe brain injuries were from automobile accidents. As to the definitely inflicted group, they had the following histories:

fall under 4 feet	8
no trauma	14
confession	2

Some of the injuries found in the inflicted group:

calvarial fracture without intracranial hemorrhage	7
basal skull fracture	2
SDH	13

3. Goldstein et al., 1993. Prospectively compared CT scans in 40 children of all ages with severe TBI. 29 unintentional and 11 inflicted. Again there was a notable age difference (7.3 years unintentional versus 1.6 years inflicted, $P < .001$). More common in the inflicted group were

SDH	12% of the unintentional, 57% of the inflicted
Interhemispheric SDH	$p < .002$

Of roughly equal frequency were EDH, SAH, ICH, and skull fracture.

6. Hymel et al., 1997. Compared CT scans in 39 unintentional with 39 inflicted. Again there was a marked age difference: 17 months in the unintentional versus 8 months in the inflicted. Found statistically significant differences in the frequency of the following lesions:

SDH	10%	accidental	versus	41%	inflicted
Interhemispheric	5%	“		44%	“
Large extraaxial fluid collections	0%	“		21%	“

No statistically significant difference in frequency was found for the following lesions:

basal ganglia edema
posterior fossa hemorrhage
shearing injury

4. Ewing-Cobbs et al., 1998. Compared 20 unintentional with 20 inflicted. The ages differed significantly (10.6 months versus 35.6 months at $P<.006$). Injuries more common in the respective groups included:

Inflicted group
SDH ($P<.005$)
atrophy
Unintentional group
EDH ($P<.04$)
shearing injury ($P<.04$)
ICH ($P<.04$)
Equally common
skull fractures
EDH
infarction/edema

5. Dashti et al., 1999. Retrospectively compared 367 unintentional with 38 inflicted ($n=405$). Found that SDH was more common in the inflicted group ($P<.001$), and that most of the SDH in the unintentional group were from automobile accidents.

6. Ewing-Cobbs et al., 2000. Had 60 children aged 1 month to 6 years with TBI -- 29 unintentional and 31 inflicted. Dr Dias notes that the ages of the two groups differed in a statistically significant way -- 35 months for the unintentional and 10 months for the inflicted ($P<.001$). Also the inflicted group had lower birth weights. Dr Dias notes that skull fractures were more common in the unintentional group but *occipital* skull fractures occurred only in the inflicted group. (p. 97) EDH was much more common in the unintentional, and SDH in the inflicted ($P<.004$). Also more common in the inflicted group were shearing injury and cerebral atrophy. More common in the unintentional group were intracerebral hematomas. MRI was reported on 48% of the unintentional and 77% of the inflicted. MRI is thought to be more sensitive for intracranial injury.

7. Reece & Sege, 2000. Retrospectively compared the charts of 233 unintentional with 54 inflicted. Again a marked age difference, 2.5 years unintentional versus 0.7 years inflicted. More common in the inflicted were SDH and SAH. Equally common were skull fractures and scalp contusions.

Dr Dias summarizes his findings as follows:

1. Skull fractures are at least as common in unintentional injury.
2. EDH is common in unintentional and uncommon in inflicted.
3. The preponderance of inflicted etiology for SDH and interhemispheric is consistently found.
4. Shearing injury does not have a consistent predominance in one group or the other.
5. Contusions seem more common in unintentional. (not clear what kind of contusions)
6. Intracerebral hematomas (ICH) show no consistent predominance.
7. Cerebral atrophy is more common in inflicted.

Dr Dias adds two incisive comments on the above studies.

- A. The studies suffered from methodological flaws, including
 1. Comparisons were not controlled for injury severity.
 2. The marked age difference is a “confounding variable” which may make the groups’ pathology non-comparable.
 3. Retrospective studies suffer from possible selection bias.

- B. "Finally, there exists some degree of circularity in reasoning; if one defines a particular injury or pattern of injuries *a priori* as inflicted, then by definition one will rarely if ever ascribe these injuries to (and therefore observe them following) an unintentional mechanism." (p. 100)

The response by Paul Kleinman.

Then there is a contribution by Kent Hymel on time of injury. See below under "Time of injury -- head."

Characteristics that distinguish accidental from abusive injury in hospitalized young children with head trauma. Bechtel K, Stoessel K, Leventhal JM, Ogle E, Teague B, Lavietes S, Banyas B, Allen K, Dziura J, Duncan C. Pediatrics 2004 Jul; 114(1): 165-168. Prospective clinical study of 82 children under two admitted for head trauma who had ophthalmology done -- 15 AHT and 72 accidental. The endpoint for separating the accidentals from the inflicted was the clinical classification scheme for accidental versus inflicted given by Reece & Sege, 2000 (*q.v.*). The accidentals were

67 accidentals:
 2 traffics
 65 witnessed falls
 47 under four feet
 9 five to twenty feet
 9 no height recorded

Found

	Abusive	Accidental
RH	60%	10% (7 chil)
Bilateral RH	40%	1.5%
Unilateral RH	20%	8% (6 chil)
Preretinal	30%	0%
Premacular	20%	0%
Extending to the periphery	27%	0%
SDH	80%	27%

Other findings given in the abstract:

Seizures	53%	6%
Abnormal mental status	53%	1%
Scalp hematoma	7%	49%

In her review of this article in the *Quarterly* for Autumn 2004, Dr Levitt comments that while the statistics found in this article and those given by Reece & Sege are "useful in making clinical decisions," for forensic and court purposes, "we need more studies with a much larger number." Because there are enough outliers in the accidental group to enable attorneys to raise doubts about the probative value of these diagnostic criteria. [Or, as Knight observes on page 471 of his *Forensic Pathology, Third Edition* (2004), if something has happened once, it can happen again. -JKR]

And indeed, when you look at these authors' results, you see that neither RH nor SDH was a good discriminant. What I think Dr Levitt is getting at in her request for a large multicenter study is that RH out to the periphery IS a good discriminant in these authors' results and in general clinical experience, and would become a probative discriminant if the numbers of subjects were large enough to reach statistical significance at a $P < .001$ level, which small single-center studies like this one can never achieve. -JKR
 Further comment by JKR: This article shows that intracranial hemorrhage and SDH do occur in falls. The article does not break down which accidentals that had SDH were falls, but in fact almost all the accidentals were falls, and at least 72% of those were short falls.

There was one confession of shaking in this article: Patient's father confessed to shaking him 48 hours before admission. No further detail.

This article cited by Hymel et al., 2007 for the proposition that abusive acts have been linked to subdural, subarachnoid, and retinal hemorrhaging.

Head injuries from falls in preschool children. Park SH, Cho BM, Oh SM. Yonsei Med J (Korea) 2004 Apr; 45(2): 229-232. From the abstract: "Falls are a leading cause of morbidity and mortality among children." Restrospective study of fall-rrelated head injury 1994-99. Had 68 cases. Divided into two age groups: group I (0-3 yrs) group II 4-6 yrs). Falls were characterised as low-level or high-level. (The abstract doesn't say what the levels were.) "Although more common in high-level falls, **significant intracranial injuries were also sustained form low-level falls.**" Height of fall did not influence clinical outcome. "We concluded that the height of fall should not limit the evaluation of patients and that aggressive management is mandatory to improve outcome even in patients with p[oor prognostic factors."

Influence of fall height and impact surface on biomechancis of feet-first free falls in children. Bertocci GE, Pierce MC, Deemer E, Aguel F, Janosky JE, Vogeley E. Injury 2004 Apr; 35(4): 417-424. This was an experiment with a dummy simulating a three year-old, using different impact surfaces and fall heights of 27 inches, 47 inches, and 64 inches. They measured the head acceleration, the femoral compressive load, and the head injury criteria (HIC). Dropped onto

foam
padded carpet
wood
linoleum

Found that both fall height and impact surface affected injury risk. BUT: these falls "were associated with a low risk of contact-type head injury, irrespective of impact surface type."

A biomechanical analysis of the causes of traumatic brain injury in infants and children. Goldsmith W, Plunkett J. Am J Forens Med P 2004 Jun; 25(2): 89-100. Cited by Geddes et al. in their Reply to Punt, 2004, as being one of the two arts that provide biomechanical support to the view that pediatric short falls can occasionally be fatal. (The other one being Ommaya, Goldsmith, Thibault, 2002.)

Children should wear helmets while ice-skating: a comparison of skating-related injuries. McGeehan J, Shields BJ, Smith GA. Pediatrics 2004; 114: 124-128. Saw 419 children for all types of injuries sustained while ice-skating, roller skating, and skateboarding. Age range 1-18. There were 41 head injuries, but all but eight of these were lacerations and other soft tissue head injuries. According to Ken Feldman, the reviewer in the Spring 2005 issue of the *Quarterly*, the eight non-superficial head injuries included four concussions. There were no cases of skull fracture or intracranial hemorrhage, and there were no neurological sequelae.

A population-based comparison of clinical and outsome characteristics of young children with serious inflicted and noninflicted traumatic brain injury. Keenan HT, Runyan DK, Marshall SW, Nocera MA, Merten DF. Pediatrics 2004 Sep; 114(3): 633-639. (Chapel Hill) From Dr Reece's summary in the Winter 05 *Quarterly*, which gives in tabular form the history, the presenting complaint, the types of bodily injuries found, and the outcomes in this prospective study identifying 152 children admitted for TBI over a two year period at UNC. Median age of inflicted was four months and noninflicted was 7 months. I will select some of the most interesting findings; for further details, see Dr Reece's review or the original article or my comments below.

	Inflicted	Noninflicted
History		
No explanation	64%	0%
Shake to revive	4%	0%

Shake or hit	2%	0%
Fall	15%	21%
Drop	7%	11%
Automobile-related	05	53%
Presenting complaint		
Stopped breathing	17%	0%
Respiratory distress	15%	0%
Seizure	12%	0%
Injuries found		
RH	76%	8%
Rib fracture	28%	6%
Long bone fracture	19%	7%
Skull fracture	18%	59%
Metaphyseal fracture	18%	3%
SDH	94%	61%
EDH	1%	18%
Cerebral edema	31%	14%
Anoxic encephalopathy	19%	11%

Important notes:

- 35% of the inflicted had no external evidence of injury.
- 38% of the inflicted had internal injuries outside the head.
- 10% of the inflicted had neither RH nor any fractures.

Dr Reece comments that this study “adds impressively” to the accumulating body of knowledge about inflicted versus noninflicted head injury, and is congruent with the other studies down to the level of which injuries and how frequent. He included this paper in his discussion of the ten top papers of the last ten years at the 2006 National Shaken Baby Conference.

A population-based study of inflicted traumatic brain injury in young children. Keenan HT, Runyan DK, Marshall SW, Nocera MA, Merten DF, Sinal SH. JAMA 2003 Aug; 290(5): 621-626.

The authors’ purpose was to determine the *incidence* of AHT. They start out by saying that the incidence of AHT is unknown. So they prospectively followed all North Carolina children aged 2 and under who were admitted to a PICU or died of TBI in 2000 and 2001. They found that there were 152 cases of serious or fatal TBI, of which 80 (53%) were inflicted TBI. Out of a state population of 230,000 children under 2, this yielded an incidence of 17 per 100,000 person-years. Relative to the general population, children who incurred an increased risk of inflicted TBI were born to mothers under 21, non-EuroAmerican, or were products of multiple births. See above under “Epidemiology” for further.

Analysis of pediatric head injury from falls. Kim KA, Wang MY, Griffith PM. Neurosurgery Focus 2000; No. 8. Supposedly available online at <http://www.aans.org/education/journal/neurosurgical/jan00/8-1-3.asp> Cited by Geddes et al. as one of three arts showing that short falls in infants can occasionally be fatal. (The other two being Plunkett, 2001 and Denton & Mileusnic, 2003).

Characteristics of inflicted TBI and noninflicted TBI. Keenan et al. Pediatrics 2004; 114: 633-639. See above under “Shaken.”

Characteristics that distinguish accidental from abusive injury in hospitalized young children with head trauma. Bechtel K, Stoessel K, Leventhal J, Ogle E, Teague B, Laviates S, Banyas B, Allen K, Dziura J, Duncan C. Pediatrics 2004 Jul; 114(1): 165-168. Prospective clinical study of 82 chil aged under 2 yr

admitted to Yale New Haven with head injury. 15 abusive and 72 accidental. all subjects had dilated ophthalmoscopy. Abusive was determined by “historical and physical examination findings and an extensive evaluation of the child’s psychosocial status,” similar to the classification scheme used by Duhaime et al., Very young children (1992) and Reece & Sege (2000). In other words, they used the history and investigation, not the head injury itself, to classify. The authors say this method “could be one limitation of our study.” Be that as it may, found:

	Abusive	Accidental
RH	60%	10%
Bilateral RH	40%	1%
Preretinal hemorrhages	30%	0%
Premacular RH	20%	0%
Extending to the periphery	27%	0%
Sz	53%	6%
AMS on presentation	53%	1%
Scalp hematoma	7%	49%

Conclusion: “Such characteristics [as the above] may be useful to distinguish accidental from abusive had trauma in children <24 months of age.” Dr Reece also included this paper in his listing of the top ten papers of the past ten years at the 2006 National Shaken Baby Conference.

Neuroradiological aspects of subdural hemorrhages. Datta S, Stoodley N, Jayawant S, Renowden S, Kemp A. Arch Dis Chil (UK) 2005; 90: 947-951. Retrospectively reviewed the head imaging of 74 children under 2 admitted with SDH between 1992 and 2001. They blindly re-reviewed all the images. 49 pts were non-accidental (NAHI), 3 were accidental (two road accidents and one fall down stairs), and 11 were miscellaneous. The following extrinsic (i.e. non-radiological) criteria used by the authors to retrospectively define non-accidental head injury:

confession (12)
conviction (8)
case conference (29)

Radiological features which were notably more common in non-accidental head injury and therefore found by the authors to be “highly suggestive of NAHI” were:

interhemispheric SDH
SDHs at multiple sites
SDHs of different densities

MRI was more sensitive than CT. CT missed small SDHs. Conclusions: MRI is also necessary in most cases. Head CT should be an integral part of the skeletal survey in all infants under 6 months who are referred for child protection investigation and in all children less than 2 years where child abuse is suspected and there are neurological signs, retinal haemorrhages, or fractures.

Additional notes from the article: Only one NAHI case had radiological evidence of DAI. Eleven SDH’s resulted from nontraumatic causes:

7 meningitis (all *S. pneumoniae*)*
2 postoperative rebleeds
1 hemorrhagic disease of the newborn
1 benign subdural effusion

* By “SDH” in this situation, the authors mean that there was a subdural fluid collection which was bloody by radiographic appearance and which in seven cases was tapped to reveal bloody fluid.

The clinical presentations are not given in detail in the article, but are summarized thus: “Presenting symptoms varied from drowsiness and malaise to fitting and coma.” Also, concerning multifocal SDH (which was seen not only in the abuse group but also in the other group(s)), there were 32/49 multifocals in the abuse group and 3/25 in the accidental/miscellaneous group; as to those three multifocals in the miscellaneous group, the authors have this to say: “In the miscellaneous group multifocal subdural collections were only seen in the infant with coagulopathy and the two children involved in road traffic accidents (one of whom had retinal haemorrhages.)” Also make the comment, concerning cerebral edema, that “Although CT is less sensitive in detecting cerebral oedema or ischaemia, particularly when performed early, diffusion-weighted MRI is exquisitely sensitive at showing these changes very early after injury...” As to methods of detecting DAI, the authors have this to say: “DAI was found in only one case, consistent with neuropathological evidence suggesting that DAI is less common following NAHI than previously thought [citing Geddes II]. CT may underestimate DAI, which is better seen on MRI, but this was only available in half of the NAHI cases.”

I think it would be worth copying the authors’ Table 2, because it sheds light on the sensitivity and specificity for NAHI of various imaging findings of SDH.

	NAHI (n=49)	ACC (n=3)	Misc (n=11)
Age 0-6m	39	0	5
Age 6-24m	10	3	6
Convexity collection	44	2	11
Interhemispheric	38	2	1
Posterior fossa	14	2	1
Middle fossa	9	2	0
Focal edema	9	2	2
Generalized edema	3	0	0
Parenchymal changes	12	0	3
Skull fracture	11	3	0
DAI	1	0	0
SDH of apparently different ages	26	0	0
SDH in multiple sites	32	2	1

As far as NAHI, the authors break out their cases that had definite evidence of impact, which were sixteen cases. In table 1 they categorize these cases as to if any fractures, where the fractures were located, and where the bruising was if any, and correlate this with where the SDH’s were. I have never seen anyone else do this.

As to rebleeds, the authors have this to say: “SDH’s of apparently different ages were found in 26 patients with NAHI. Legal questions often arise regarding the possibility of rebleeding into pre-existing SDH in these cases. In 20/26 cases there was extracranial evidence of previous abuse, supporting the interpretation that the SDHs were due to different episodes of NAHI. These children presented with either signs of raised intracranial pressure or encephalopathy on a background of having generally been unwell. It is accepted that rebleeding can occur following minimal trauma in the elderly population. The pattern of the more acute bleeding in cases of NAHI is different to that seen in adult rebleeds, being usually multifocal rather than unifocal, of small volumes, and in a distribution typical of that seen in the primary NAHHL. Therefore, this pattern of bleeding in the presence of subdural haematomas of apparently different ages is more likely to be due to further episodes of abuse rather than to spontaneous, synchronous multifocal rebleeds. Also, the volumes of blood associated with these possible rebleeds is rarely of such a volume as to cause either a significant acute rise in intracranial pressure or to be the direct cause of an acute encephalopathic illness. We did not come across any cases where further SDH arose spontaneously in post-meningitic effusions. Rebleeding occurred only in one patient after surgical evacuation.”

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. “Although case reports of

significant household accidents have been shown to cause posterior retinal hemorrhages (citing Cindy Christian et al., household falls, 1999), minor falls and household accidents do not cause the extensive retinal hemorrhages and diffuse brain injury seen in inflicted head trauma.” (p. 50) As to falls, she gives a full-page table summarizing the literature on falls versus abuse (p. 51), with emphasis on those studying reliably observed falls. Conclusion: “[S]imple household falls as an explanation for serious head injury should be considered inadequate.” [But note that that leaves out the non-simple household falls such as when a larger baby or child pitches itself out of the parent’s arms onto a hard surface or falls with a rotational component or when the adult also falls. As to those, see Minns, below.]

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. Unfortunately, the weakest part of this article is the part that deals with fall versus inflicted. Partly because fall versus inflicted is only a small subset of the universe of AHT cases. Why? Because most AHT cases have other injuries, such as bruises, burns, fractures, neglect, or social pathology, which take them out of the category where a short fall can plausibly arise as a differential diagnosis, even if one is reported by the caregiver. As Minns states, “A very contentious scenario is where children present with subdural and retinal haemorrhages only, with no history of trauma or a history of minor trauma only, e.g. from a short fall (less than three feet). In such circumstances, and after thorough investigation, the clinician can only state that these injuries are consistent with an injury of non-accidental origin.” I will quote in full the author’s treatment of fall versus inflicted.

“Short falls are often cited as an explanation or as a defence in SBS cases. They are regularly reported as falls from a bed, changing table, or the parent’s arms. We have seen babies of five weeks of age arch backwards, effectively diving from a parent’s shoulder and single-handed hold, falling 5 feet onto a tiled floor. The focal bruising, extensive skull fractures, and focal brain contusion, is not accompanied by a concussive element or other encephalopathy or delay in seeking treatment. Simple investigation of the circumstances therefore determines that such impact deceleration injuries are explicable.

“Toddlers fall very frequently out of harnesses and baby bouncers, and undoubtedly fatal and serious injury can occur from low height falls, but these are exceptionally rare. ... When an SDH is present this indicates a rotational injury, and a critical rotational velocity must have been attained for concussion, and for subdural to have occurred from very short falls. A ‘short impact time’ and a ‘high terminal velocity’ would be unusual with these minor falls, but with injuries that result during a fall when the head strikes an object causing acceleration rapidly in one direction followed immediately by rotation in the opposite direction, then sufficient rotational accelerations may be attained to induce haemorrhage.”

The author then goes on to discuss Plunkett’s witnessed short falls paper, but says that these were outside the infant age range and had no suspicious circumstantial or social-pathology features.

The author mentions that walker-stairs falls are “potentially very serious,” and bunk beds “may also rarely produce SDH’s.”

Accidental and nonaccidental head injuries in infants: a prospective study. Vinchon M, Defoort-Dhellemmes S, Desurmont M, Dhellemmes P. (Lille) J Neuros 2005; 102 (4 Suppl): 380-384. The incidence and causes of infantile SDH are poorly estimated. Prospective study of all infants admitted to Lille over a three year period, comprising 150 SDH patients, of which 57 were diagnosed as due to child abuse (38%). All had opth by a neuroophthalmologist. The SENS and SPEC of RH for child abuse were 75% and 93%. RH associated with accidental trauma were always mild. The SPEC of severe RH for abuse was 100% in this series.* Child abuse SDH was significantly correlated with the following factors:

perinatal illness
absence of signs of impact
seizures on presentation

* I hope the authors didn’t engage in circular reasoning by automatically diagnosing child abuse in every case of severe RH and then “discovering” that severe RH had a 100% correlation with child abuse. –JKR

Falls from highchairs. Schalamon J, Ainoedhofer H, Sacena AK et al. Eur J Pediatr 2006; 165: 732-733. From Dr Reece’s review in the Winter 07 *Quarterly*, had 34 chil.

head contusions	62%
concussions	14%
skull fractures	6%

21% required hospital admission. Mean time of hospitalization was 3 days. No deaths.

Comparison of accidental and nonaccidental traumatic head injury in children on noncontrast computed tomography. Tung GA, Kumar M, Richardson RC, Jenny C, Brown WD. Pediatrics 2006 Aug; 118(2): 626-633. From the abstract: “OBJECTIVE. Mixed-density convexity subdural hematoma and interhemispheric subdural hematoma suggest **nonaccidental** head injury. The purpose of this retrospective observational study is to investigate subdural hematoma on noncontrast computed tomography in infants with nonaccidental head injury and to compare these findings in infants with accidental head trauma for whom the date of injury was known.

PATIENTS AND METHODS. Two blinded, independent observers retrospectively reviewed computed tomography scans with subdural hematoma performed on the day of presentation on 9 infant victims of nonaccidental head injury (mean age: 6.8 months; range: 1–25 months) and on 38 infants (mean age: 4.8 months; range: newborn to 34 months) with accidental head trauma (birth-related: 19; short fall: 17; motor vehicle accident: 2).

RESULTS. Homogeneous hyperdense subdural hematoma was significantly more common in children with accidental head trauma (28 of 38 [74%]; nonaccidental head trauma: 3 of 9 [33%]), whereas mixed-density subdural hematoma was significantly more common in cases of nonaccidental head injury (6 of 9 [67%]; accidental head trauma: 7 of 38 [18%]). Twenty-two (79%) subdural hematomas were homogeneously hyperdense on noncontrast computed tomography performed within two days of accidental head trauma, one (4%) was homogeneous and isodense compared to brain tissue, one (4%) was homogeneous and hypodense, and four (14%) were mixed-density. There was no statistically significant difference in the proportion of interhemispheric subdural hematoma, epidural hematoma, calvarial fracture, brain contusion, or subarachnoid hemorrhage.

CONCLUSIONS. Homogeneous hyperdense subdural hematoma is more frequent in cases of accidental head trauma; mixed-density subdural hematoma is more frequent in cases of nonaccidental head injury but may be observed within 48 hours of accidental head trauma. Interhemispheric subdural hematoma is not specific for inflicted head injury.”

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.” In this prospective multicenter comparison study of 54 head injury admissions below age 36 months, they compared outcomes and mechanisms as between inflicted, noninflicted, and undetermined cases. Their tabulation of noninflicted injury mechanisms showed the following:

4 MVA's	
26 falls	
3	from heights over 10 feet
2	from heights between 6 and 10 feet
17	from heights less than 6 feet
4	stair falls

Had 7 falls that had SDH. (Not broken down as to height, except for saying that they had 3 short falls (3 to 6 feet) that had interhemispheric SDH. (see below) These 3 subjects had “superficial cortical brain injuries” and no impairment of consciousness.

The authors state, “A large body of literature supports a conclusion that short-distance pediatric falls are generally benign. Our results also support this conclusion. Six months after injury, our 26 subjects with noninflicted head injury attributed to a fall demonstrated normal mental development index and gross motor quotient scores ... On the other hand, our data revealed a few interesting exceptions.” They go on to discuss their fall cases. One was 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 probably would have shown up on a followup imaging or MRI.

The authors continue, “Four of our 26 subjects with noninflicted head injuries attributed to a fall manifested an impairment of consciousness. Seven of 26 revealed subdural hematoma... These 7 subjects included 3 with SDH that extended or originated from the interhemispheric region, indicative of a noncontact mechanism of injury. All 3 of these young children fell from a height of only 3 to 6 feet; manifested associated contact injuries; revealed only superficial, focal, cortical brain injuries; and experienced no alterations of consciousness. These cases seem to demonstrate that noncontact (i.e. interhemispheric) SDH of minimal clinical significance can result from a short-distance fall and that the injury thresholds or biomechanical requirements for noncontact SDH and acute concussion, also a noncontact injury, are different.” [Comment by JKR: That would be consistent with the primate work, in which the monkeys that had SDH and the monkeys that had concussion were not always the same monkeys.]

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 Neurol 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 hypoxic-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, thalami, 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., *N E 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 injuries.

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 head 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; Wilman, 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 Humpreys 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; Vanchon 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 shakers 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; Joffe & 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 1/4 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 1/4 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
12	abrasions
10	lacerations

"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 admitted 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 Duhaime 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 significance 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
 - 3 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.

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 evalutated ... 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 Sirotinak'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
- 11 bloody noses
- 4 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 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 ½ 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. Conners 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; Joffe & 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 treated 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, resulting 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 Pediatr 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/cpscpub/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 accidents 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 long periods of time. The challenge was to develop a product 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.

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."

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? Yamashita 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.

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:

1. What are the potential explanations for an enlarged subarachnoid space in infant?
2. Are they predisposed to subdural bleeding?
3. What is the DDX for SDH?
4. What is the pathophysiology of traumatic SDH?
5. What does serial imaging show?
6. What are the potential explanations for the heterogeneity in the appearance of a SDH on CT?
7. Under what circumstances do rebleeds occur?
8. What are the expected clinical consequences of a rebleed?

Presentation by Dr Mary Case at the October 2003 NAME Annual Meeting. Said that neomembranes only happen in the presence of underlying cerebral atrophy. Also said that there is no subdural space (I have heard this before from neurosurgeons.) Subdural bleeding, she says, is actually intradural bleeding from the dural attachments of the arachnoid vessels or bridging veins; it dissects within the inner layer of the dura mater. There is a dural boundary epithelium on the arachnoid side of an SDH. [It must be added that Dr Case's conclusions were regarded as highly controversial by most of the conference participants. It certainly doesn't square with conventional wisdom or with Maxeiner's purported demonstration of broken bridging veins in SDH. --JKR]

Subdural hematomas in infants with benign enlargement of the subarachnoid space are not pathognomonic for child abuse. McNeely PD, Atkinson JD, Saigal G, O'Gorman AM, Farmer J-P. Am J Neuror 2006; 27: 1725-1728. According to Dr Moles' review in the Winter 2007 *Quarterly*, retrospective case review of all children aged under 18 months at the Montreal Children's Hospital who had more than 5mm distance between the surface of the brain and the inner table of the skull. This is called benign enlargement of the subarachnoid space (BESS). Seven of these children also had a subdural hematoma. The authors excluded AHT patients and patients with a coagulopathy. Two of the seven pts had MVA and one had an own-height fall. Concl: "SDH may occur either spontaneously or as a result of a minor or unrecognized trauma in pts with BESS." Comment by JKR: Look, none of these children were medically normal pre-hematoma.

VACCINE SBS (DPT)

See APRI Update vol 13 no. 9, 2000, by David Chadwick and Rob Parrish. "DTP Vaccination or Shaken Baby Syndrome? The role of irresponsible medical expert testimony in creating a false causal connection." [The same article appears in SBS Quarterly, Fall 2000.] Here the authors review the literature both medical and legal. "The pathology described for SBS is due to mechanical injury, and there are very few possible exceptions. Somewhat similar pathology has been ascribed to spontaneous bleeding into the subdural space that may occur with certain congenital malformations or with blood coagulation problems. However, no medical papers exist which propose that this pathology could be related in any way to DTP immunization." See text for further.

Syncope after immunization. Braun MM. Arch Pediatr Adol Med 1997; 151: 255-259. From the VAERS there were 697 incidents of syncope with 6 skull fx or intracranial bleeds in vaccinees 12-28 years old.. Some had EDH and/or coup or contrecoup contusions, one SDH.

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-Barness, 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 childrn with nitally 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: retrop 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 duodenum initially missed. Presented 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: 56-61.

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 Pediatr 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 relatively 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%."

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.

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 prox 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 m girl with old fractures and FTT.

Ken Feldman comments that shaking can rupture the thoracic duct in ass w compr fx of vert which this child had.

Intestinal stricture following seat belt injury. Lynch JM, Albanese CT, Meza , 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 shd 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
13	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.

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, Metzenberg A. J Pediatr 1999 Feb; 134: 206-214. This disease causes peliosis hepatis, 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 of intraab 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 perms. 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 retroper injury.

ASCP Check Sample No. FP 03-9 (FP-290). Corey TC and Wetherston 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 perms. 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 (perf, 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 perms and 6 contusions or hematomas. The solid organ injuries were 6 spleens, two pancr, 2 K's, 7 livers. Ten children had combinations of injuries. 5/20 pts 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 Infec 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 inflicted, 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 art 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 $\frac{3}{4}$ of the hollow-viscus injuries and $\frac{2}{3}$ 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 not 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 diagnosis; we recommend a period of observation for all child who have sx after such an inj.”

Duodenal injuries in children: beware of child abuse. Gaines BA, Schultz BS, Morrison K, Ford HR. J Pediatr 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 perms than with UGI perms. 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 children 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 evidence 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. According 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 ¼ 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. Cited 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 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."

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 1. Kohler M. Pediatric Radiology 1998; 28: 582

Subdural hemorrhage as an initial sign of glutaric aciduria type a: 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 1. 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. Bilat 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 Sirotiak & 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 chronic 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 puruvate 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 wth limpness, myoclonus. The authors refer to two previous reports of ICH and state that condition cd 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 tomcat 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 dxd 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.

SEXUAL ABUSE

(see also “Parental Alienation Syndrome,” below)

Definition: [from AAP Clinical report: the evaluation of sexual abuse in children. Nancy Kellogg MD and the Committee on Child Abuse & Neglect, AAP. Pediatrics 2005 Aug; 116(2): 506-512] “Sexual abuse occurs when a child is engaged in sexual activities that he or she cannot comprehend, for which he or

she is developmentally unprepared and cannot give consent, and or that violate the law or social taboos of society. (citing Kempe, 1978). The sexual activities may include all forms of oral-genital, genital, or anal contact by or to the child or abuse that does not involve contact, such as exhibitionism, voyeurism, or using the child in the production of pornography.”

Calif. Penal Code Sec. 11165.1: “As used in this article, ‘sexual abuse’ means sexual assault or sexual exploitation as defined by the following:: ...” [See document in file.]

Calif. Penal Code Sec. 13823.5 et seq. requires the OCJP to promulgate protocols for the examination and reporting of sexual and domestic violence victims, including “child molestation.” See document in file.

CAPTA defines sexual abuse as

“Employment, use, persuasion, inducement, enticement, or coercion of any child to engage in, or assist any other person to engage in, any sexually explicit conduct or any simulation of such conduct for the purpose of producing any visual depiction of such conduct; or

“Rape, and in cases of caretaker or interfamilial relationships, statutory rape, molestation, prostitution, or other form of sexual exploitation of children, or incest with children...”

See Mignon SI et al., *Family Abuse: Consequences, Theories and Responses*. Boston: Allyn & Bacon, 2002, p. 19.

California Medical Protocol for Examination of Sexual Assault and Child Sexual Abuse Victims, July 2002. Governor’s Office of Criminal Justice Planning. <http://www.ojcp.ca.gov/>

Books:

Astrid Heger, S. Jean Emans, David Muram, *Evaluation of the Sexually Abused Child*, 2d ed. Oxford University Press, 2000 (book and CD-ROM).

Giardino AP, Finkel MA, Giardino ER, Seidl T, Ludwig S. *A Practical Guide to the Evaluation of Sexual Abuse in the Prepubertal Child*. Newbury Park: SAGE Publications, 1992. See second edition, 2002, below.

Jean M. Goodwin, ed. *Rediscovering Childhood Trauma: Historical Casebook and Clinical Applications*. Washington, DC: American Psychiatric Press, 1993.

Part Three: Sexual Abuse Cases. In: Brodeur AE and Monteleone JA, *Child Maltreatment: A Comprehensive Photographic Reference Identifying Potential Child Abuse*. St. Louis: G. W. Medical Publishing, 1994.

Section 4, Sexual Abuse. In: Hobbs CJ and Wynne J M, *Physical Signs of Child Abuse: A Colour Atlas*. London: W B Saunders Ltd., 1996. In the captions of the photographs of abused girls, this brings out the following features of the hymens and genitalia of abused patients:

- reddening
- swelling
- attenuation
- irregular opening
- recent tear
- friability of the posterior fourchette
- scarring of the posterior fourchette
- deep notch at 6:00
- midline tear

transection
labial fusion, especially posterior

Also includes photodocumentation of acute rape patients.

National Clearinghouse on Child Abuse and Neglect. www.calib.com/nccanch. 800-394-3366

Office of Criminal Justice Planning: California Medical Protocol for Examinations of Sexual Assault and Child Sexual Abuse Victims. (OCJP 923 and 925). As of early 2000, these forms and accompanying protocol are being revised. Indeed, as of December 2002 they have been revised. <http://www.ocjp.ca.gov/> See also Peterson & Durfee's *Guide*, 2003.

Evaluation of the Sexually Abused Child, 2d ed. Heger A, Emans JE, Muram D., eds. New York: Oxford University Press, 2000. A standard work. It has an annotated bibliography in the back.

Sexual Assault: The Medical-Legal Examination. Sharon R. Crowley, RN. Columbus, Ohio: McGraw-Hill, 2000. ISBN 0-8385-8533-7. 227 pp, 110 illustrations. I attended Miss Crowley's workshop at the AAFS Seattle Feb 2001 entitled, "Postmortem Genital Examinations with Colposcopy: SART to GO." ("To go" because she has improvised a portable photocolposcope for transport to outlying funeral homes and coroner's offices.) Some of the points she brought out included the relevance of the human sexual response cycle as per Masters & Johnson, and its absence, to the nature of the injuries re the creation of a "landing platform" for the male organ with respect to (a) lubrication and (b) pelvic tilt. "Prevention of injury requires participation." (But participation does not necessarily prevent injury.) (This would be in those females capable of sexual response.) Sexual assault injury is described as a "mounting" injury. States that lubrication occurs in 10 to 30 seconds. Also gave details on the use of the colposcope at 7.5x, 15x, and 30x, with mostly 15x being the most useful judicially for photocolposcopic evidence. She demonstrates microtears with and without methylene blue. Points out to use methylene blue extremely sparingly, just on the point of interest, otherwise it messes up the field. Points out that MB positivity has a DDX of 23 entities, including the fact that cervical mucus is MB+. Her book discusses the elderly and males also. She is engaged in doing her study of postmortem material at the San Luis Obispo County coroner's office. Her published material reports the findings in 311 living adult female victims of all ages plus some controls reporting consensual intercourse. Of the (adult) subjects seen within 24h of intercourse the following percentages had colposcopically demonstrable genital injury:

Sexual assault	80%
Consensual intercourse	11%

Overall, including both within-24h exams and later exams, total 311 victims and unk no. of controls,

Sexual assault	68%
Consensual intercourse	??%

Healing was 7 – 10 days in these adults. Pediatric wd be expected to be faster, she says.

Of those victims of assault reporting sodomy, 50%
had anal injury by anoscopy.

She also discusses offender typology re modus operandi. Advises the use of water technique for visualizing the posterior hymen. See also the printed handout from her talk, which I have, some of whose refs have been incorporated herein.

Mignon SI, Larson CJ, Holmes WM. *Family Abuse: Consequences, Theories, and Responses*. Boston: Allyn & Bacon, 2002. ISBN 0-205-29569-X. (have) Contains extensive discussion of sexual abuse from a psychology perspective.

Cynthia Crosson-Tower, *Understanding Child Abuse and neglect*. Boston: Allyn & Bacon, 2002. ISBN 0-205-33795-3. (have)

Medical Evaluation of Child Sexual Abuse: A Practical Guide. 2d edition. Finkel MA and Giardino AP, eds. Thousand Oaks: Sage Publications, 2002. Reviewed in Child Abuse & Neglect 2002 Oct; 26(10): 1109-1112.

Chapter by Angela Rosas, MD in MS Peterson and M Durfee, eds., *Child Abuse and Neglect: Guidelines for Identification, Assessment, and Case Management*. Volcano, Calif: Volcano Press, 2003. (have) Says that it is "estimated" that only 30% of sexually abused prepubertal females have physical findings. (p. 87) This estimate is much higher than the figure of less than 5 % recently found by Dr Heger. Gives a summary of the state OJCP protocols.

Toluidine blue

Use of toluidine blue for documentation of traumatic intercourse. Lauber AA and Souma ML. Ob Gyn 1982 Nov; 60(5): 644-648

Toluidine blue in the detection of perineal lacerations in pediatric and adolescent sexual abuse victims. Pediatrics 1986; 78: 1039-1043. Toluidine blue failed to distinguish consensual from nonconsensual intercourse in adolescents. See personal communication from Joyce Adams 1999.

Toluidine blue. Joyce Adams, personal communication, 1999. "I never used the dye in Fresno, and I am suspicious of its specificity after being a consultant for the past 2 years for a SART program at UCSD. It seems that much of the uptake in adult and adolescent women is of the diffuse variety, with accentuation of vulvar irritation which can have many causes. Also, tiny superficial abrasions in the fossa, posterior fourchette, and labia minora could be related to trauma, but how many young women have these findings who haven't had sex? Or had consensual sex? I don't think we have the answers to this. Is anyone looking?"

Toluidine blue in the detection at autopsy of perineal and anal lacerations in victims of sexual abuse. Bays J and Lewman LV. Arch Path Lab Med 1992; 116: 620-621 (have).

Reports four autopsy cases in which toluidine blue was used. Case 1 an abducted 4 yr old boy found dead in the woods. Two small perianal tears were noted grossly, and three lac at the anal verge by colposcopy of the excised anal specimen. Following toluidine blue application, repeat specimen colposcopy showed 15 additional lacerations of the anal verge. Case 2 a 3 month old infant found dead at home. ER noted blood around the anus. There was a healing fracture. Gross autopsy showed several tears around the anus. Toluidine blue showed a regular repeating pattern of tears which matched the flanges on a plastic nozzle. Convicted. Case 3 a 3 yr old boy with 100 injuries. A 1-cm lac of the anus and perineum. Toluidine blue revealed two more lacerations. Case 4 a 3 year old girl suffocated by older foster-brothers. Nothing seen anogenitally, and toluidine blue revealed nothing. So in summary, TB revealed new injuries in the three cases where perianal tears had already been seen grossly, and confirmed the negative exam in the one case where gross exam was negative.

Signs of genital trauma in adolescent rape victims examined acutely. Adams JA, Girardin B, Faugno D. (UCSD) J Pediatr Adol Gyn 2000 May; 13(2): 88. Joyce Adams group did a retrospective chart review of 214 females 14-19 years of age evaluated acutely by the SART team using magnification and dye for a complaint of sexual assault. 66% had + uptake of toluidine blue. See below.

Tanner staging.

Adolescent Health Update. Vol 1(2) Feb. 1989 AAP.

Assessment of Sexual Maturity Stages in Girls. AAP Publ. No. E-MA0089: photos of Tanner stages. Growth charts including percentiles for sexual maturation. Serono Laboratories, 800-283-8088. Charles F. Johnson, Teaching slides: Is it child pornography? AAP SCAN Newsletter, 1998 Mar; 10(1): 4.

Evaluating the child for sexual abuse. Lahoti SL, McClain N, Girardet RT, McNeese M, Cheung K. Am Fam Phys 2001 Mar; 63(5): 883-892. Symptoms enuresis etc, behavior changes including sexual acting out, regression, aggression, depression, eating disorders. Most vic physical findings are normal. Therefore the child's statements are extremely important. Examiner must be familiar with normal variants and nonspecific changes as well as diagnostic signs of CSA. Comment by Joyce Adams 843-844, 846.

Class correlation:

F. Brett Drake of Washington University of St. Louis (brettd@gwbmail.wustl.edu) has studied this. He indicates that the available epidemiologic evidence as of 1998 suggests that there is a class correlation of sexual abuse, but not as clearcut or as strong as the one for physical abuse. Citing Child Abuse & Neglect 20(11): 1003-1018; survey of physical abuse epidemiology by Drake, Am J Orthopsychiatry 68(2): 295-304.

Desmond Runyan of UNC cites a Gallup Poll from December 1995 entitled "Disciplining Children in America" (Princeton, NJ: Gallup Organization) showing a class correlation for sexual as well as physical abuse. He directs us to Straus M, Hamby S et al. Identification of child maltreatment with the parent-child conflict tactics scales: development and psychometric data for a national sample of American parents. Child Abuse & Neglect 1998; 22: 249-270, which uses the Gallup data.

Adolescent pregnancy and sexual abuse. M Jocelyn Elders and Alexa E Albert. JAMA 1998; 648-649. Up to 66% of pregnant teens report sexual abuse. The majority of pregnant girls under 15 had sex against their will. Over half of all infants born to women younger than 18 were fathered by adult men. But criminalizing this may deter the women from reporting, bc of fear of revealing the identity of their partner. Some of the rels are undoubtedly consensual. They do not seem to advocate routine CSA reporting in the care of these pts, but shd assure that they receive counselling.

Sexually transmitted organisms in children and child sexual abuse. Robinson AJ. Int J STD AIDS 1998; 9: 501-511. Bob Shapiro terms this an excellent, detailed review article with 82 refs.

Abuse history: is it really important in the medical encounter? Rubin NJ and Gardner J. Southern Medical Journal 1999; 92: 618-621.

Late effects

[Note that some of these papers are not specific to sexual abuse. JKR]

Long-term consequences of childhood physical and psychological maltreatment. Gross AB, Keller HR. Journal of Aggressive Behavior 1992; 18: 171-185. As described by Mignon et al. (2002a at 95) this paper studied low self-esteem, depression, and "maladaptive attributional style" in 260 college students who reported having been victims of childhood physical or psychological maltreatment. Found psychological rather than physical abuse to be the critical variable in these future psychopathologies.

The combined effects of physical, sexual, and emotional abuse during childhood: long-term health consequences for women. Moeller TP, Bachmann GA. Child Abuse & Neglect 1993; 17: 623-640. Mignon et al. (2002a at 96) describe this study as a questionnaire study from 668 adult gynecology patients. 53% reported having suf child abuse of some form, most commonly emotional abuse. Without separating the types of abuse as to effects, it found that as compared to nonabused, the abused females reported

significantly worse physical health with more hospitalizations, and more self-reported psychological problems.

Criminal consequences of childhood sexual victimization. Widom and Ames, Child Abuse & Neglect 1994; 18(4): 313. Says that there is a cycle of violence, i.e. the victims grow up to be sex criminals. These findings were later revised. It was based on clinical samples, which may overestimate late effects as compared to the population at large.

Relationships among child abuse, date abuse, and psychological problems. Sappington AA et al. J Clin Psychol 1997; 53: 319-329. As described by Mignon et al (2002a at 95), this paper studied 133 female undergraduates, found that early *verbal abuse* led to later victimization by date abuse and emotional problems.

Sexual contact between children and adults: a life-course perspective. Browning and Laumann, American Sociological Review 62(4): 540 (1997) Did not find an association with adult sexual offending, but did find with other outcomes, but these effects were mediated by other intervening events.

Health-related quality of life and symptom profiles of female survivors of sexual abuse. Dickinson LM, deGruy FV II, Dickinson WP, Candib LM. Arch Fam Med 1999; 8: 35-43. The study material was all female outpatients in a fam prac clinic who had unexplained physical symptoms. Of these, 38-46% reported a past history of sexual abuse. The abuse and the current psychiatric sx were correlated by severity.

A meta-analytic examination of assumed properties of child sexual abuse using college samples. Rind B, Tromovitch P, and Bauserman R. Psychological Bulletin 1998; 124(1): 22-53. This is a review of 59 previously published research studies that used college students as subjects. Finds that the negative social-adjustment effects attributed to child sexual abuse (CSA) were not separated from the effects of family environment; when the data was controlled for family environment, all of the negative social-adjustment effect attributed to CSA disappeared. Also, the subjective self-reports of negative effects from CSA showed effects that were not intense. Apparently there are comparable results from non-college-student studies. The authors point out that adult-child sex is common cross-culturally and “may fall in the ‘normal’ range of human sexual behaviors.” Also challenge the usage of the term “abuse” for conventionally immoral behavior that is not harmful. This article is commented on from a political point of view in Ondersma et al., 1999. Also in Lilienfeld, 2002.

Comments on the Rind et al. Meta-analysis controversy. Ondersma SJ, Chaffin M, Berliner L. APSAC Advisor 1999 Fall; 12(3): 2-5. Saying that Rind et al. stepped over the line when they went beyond data analysis and asked for reevaluation of the use of the term “abuse” for immoral but non-harmful behaviors. Argue that this type of statement is unethical under the APA code of ethics because it lends itself to being misused by outsiders. These commentators concede that Rind’s meta-analytical methods were valid and that his findings are reproducible and that they are consistent with the findings of others.

Long-term effects of CSA: objective and subjective characteristics of the abuse and psychopathology in later life. Lange A, DeBeurs E, Dolan C, Lachnit T, Sjollem A, Hanewald G. J Nerv Ment Dso 1999; 187: 150-158. A survey of 404 Dutch women who indicated willingness to be questioned about past CSA. They commonly indicated long-term effects, especially from severe abuse. But unknown is the number of abused females in the general population who experience no long term effects. Studies tend to pick up the positive cases.

The relation of child sexual abuse and depreression in young women: comparisons across four ethnic groups. Roosa MW, Reinholtz C, Angelini PJ. Journal of Abnormal Child Psychology 1999; 27(1): 65-76. CSA was a strong predictor of adult depression in all ethnic groups.

Research on resilience to child maltreatment: empirical considerations. Heller SS et al. Child Abuse & Neglect 1999; 23(4): 321-338. A literature review on resilience.

Childhood sexual abuse as a risk factor for depression in women: psychosocial and neurobiological correlates. Weiss EJ, Longhurst JG, Mazure CM. Am J Psychiatr 1999; 156: 816-828. Well-recognized association. The severity of the abuse correlates with the likelihood of depression.

Sexually abused teens show high-risk behaviors and mental problems. By Karla Harby. Reuters Medical News on Medscape, May 17, 2000. Both boys and girls. This work by Dr. Linda M. Barthauer of the University of Rochester's Children's Hospital at Strong, reported to the combined annual meeting of the Pediatric Academic Societies and the AAP. Sexual abuse stood out apart from financial status, physical abuse, or age. They say they are in fair to poor health and have missed medical care. 10% of girls and 4% of boys reported CSA on this questionnaire.

[Behavioral, educational, and medical problems increased in young children who have been sexually abused.] Archives of Disease in Childhood 2000 Aug; 83: 132-134. Dr. C. j. Hobbs of St. James University Hospital, Leeds, studied 140 sexually abused children with a control group of 83 non-abused age-mates in a retrospective chart review of hospital and school records. Followed them out for 8 years.

	abused	nonabused
Behavioral probs	60%	16%
Educational probs	24%	5%
Health probs	32%	1%

History of sexual abuse increases risky sexual behavior in adolescents. Reuters Medical News on Medscape, September 2000, citing Brown LK et al in Am J Psychiatry 2000 Sep; 157: 1413-1415 for a study of 116 sexually active adolescents of which 61 had been sexually abused earlier. Controlling for other risk factors such as psychopathology, disturbed families, and extent of dysfunction. Inconsistent condom use was 3x more, and also poor impulse control and rate of STD.

Widom, Child Abuse & Neglect 18(4). Well known article finding that in later life CSA victims were 4.7 times as likely to be arrested for sex crimes. Physical abuse was more strongly associated with arrest for rape or sodomy. Widom comments that, "The criminogenic effect associated with sexual offending may not result from sexual abuse uniquely, but rather may be associated with the trauma and stress of these early childhood experiences or society's response to the event." (313).

There was a later GAO report from a later set of data on the same cohort, which has also been commented on by Widom. Reportedly this study indicated that the independent effect of sexual abuse disappeared and the late effects could be accounted for by physical abuse. Those who experienced ONLY sexual abuse were at no increased risk for adult sex offending.

A meta-analytic examination of assumed properties of child sexual abuse using college samples. Rind B, Tromovich P, Bauserman R. Psych Bull 1998; 124(1): 22-53. (have) Showing that the literature does not indicate any separable effect on college women's psychological health from a history of CSA. See the response by the APA at <http://apa.org/releases/childsexabuse.html>. (have) Abstract: "Many lay persons and professionals believe that CSA causes intense harm, regardless of gender, pervasively in the general population. The authors examined this belief by reviewing 59 studies based on college samples. Meta-analyses revealed that students with CSA were, on average, slightly less well adjusted than controls. However, this poorer adjustment could not be attributed to CSA because family environment was consistently confounded with CSA. Family environment explained considerably more adjustment variance than CSA, and CSA-adjustment relations generally became nonsignificant when studies controlled for family environment. Self-reported reactions to and effects from CSA indicated that negative effects were neither pervasive nor typically intense, and that men reacted much less negatively than women. The college data were completely consistent with data from national samples. Basic beliefs about CSA in the general population were not supported."

The article includes a section called, "Child sexual abuse as a construct reconsidered." Points to "a history of conflating morality and law with science in the area of human sexuality by psychologists and others indicates a strong need for caution in scientific inquiries of sexual behaviors that remain taboo..." States that father-daughter incest has shown a larger effect size and more often been perceived negatively

by the women, as opposed to the other forms of CSA where effect size has tended to be small and subjective perceptions often neutral or positive. That the term “abuse” implies harm, and may beg the question where harm does not exist. “...the construct of CSA as commonly conceptualized by researchers, is of questionable scientific validity.” For a rebuttal to this article see Ondersma et al., 2001.

Adult sexual revictimization among black women sexually abused in childhood: a prospective examination of serious consequences of abuse. West CM, Williams LM, Siegel JA. Child Maltreatment 2000Feb; 5(1): 49-57. Which characteristics of CSA were predictive of revictimization? N=33 CSA victims were revictimized in adulthood, n=77 were not. Physical force 86% v. 53% (p less than .001). Penetration 94% v. 85%. Abused by family member 64% v 52%. At a level of P .001, physical force was the only characteristic of CSA that predicted subsequent victimization.

Preliminary evidence for aberrant cortical development in abused children: a quantitative EEG study. Ito Y. Teicher MH, Glod CA, Ackerman E. (Department of Psychiatry, Harvard Medical School.) J Neuropsychiatry Clin Neurosci 1998 Summer; 10(3): 298-307. Did EEG's on 15 hospitalized children with severe physical or sexual abuse and 15 normal children. Abused children had higher levels of left hemisphere coherence and a reversed asymmetry.

Child sexual abuse and subsequent psychopathology: results from the National Comorbidity Survey. Molnar BE, Buka SL, Kessler RC. Harvard School of Public Health. Am J Publ Health 2001 May; 91(5): 753-760. Conclusions: CSA usually occurs as part of a larger syndrome of childhood adversities. Nonetheless, CSA, whether alone or in a larger adversity cluster, is assoc with substantial increased risk of subsequent psychopathology.

Child sexual abuse and subsequent psychopathology: results from the National Comorbidity Survey. Molnar BE, Buka SL, Kessler RC. Am J Publ Health 2001 May; 91(5): 753-760. Multivariate analysis of 5877 subjects controlling for other childhood adversities. Significant associations were found between CSA and 14 mood, anxiety and substance use disorders among women and men.

Sex with children is abuse: comment on Rind, Tromovitch, and Bauserman (1998). Ondersma SJ, Chaffin M, Berliner L, Cordon I, Goodman GS, Barnett D. (Wayne State). Psychol Buoll 2001 Nov; 127(6): 707-714.. Rebut Rind and argue for the appropriateness of using the term “abuse” in research, because it is socially conventional.

Family Abuse: Consequences, Theories, and Responses. Mignon et al. 2002a at p. 73: “A history of child sexual abuse places victims at risk for a range of difficulties later in life, including emotional and behavioral problems, low self-esteem, sexual dysfunction, substance abuse, eating disorders, involvement in further abusive relationships, depression, and suicide.” (citing sources.) Borderline personality disorder, PTSD (DSM III-R), substance abuse. Referring also to Burgess AW and Holmstrom LL, *Rape: Crisis and Recovery* (1979), described as “a classic work in the field.”

Scars that won't heal: the neurobiology of child abuse. Martin H. Teicher. Scientific American 2002 Mar. pp 68-75. Reviewed by Dr. Reece in *Child Abuse Quarterly*. Boston psychiatrist did a questionnaire survey for symptoms of temporal lobe epilepsy of 253 adults, about half of whom reported a history of physical or sexual abuse as children. Found that the ones who had physical abuse had epilepsy scores 38% higher and the sexually abused had 49% higher, and if both had 113% higher. Also Dr Reece reports that these guys found by MRI increased activity in the vermis plus limbic irritability. Dr Reece goes on to review the MRI studies, one of which has shown reduced hippocampi, and EEG studies which were +.

When worlds collide: social science, politics, and the Rind et al (1998) child sexual abuse meta-analysis. Am Psychol 2002 Mar; 57(3): 176-188. The abstract says that this comments on political and social aspects of the AAP's response to the Rind article, including areas of academic freedom and authors' responsibilities in the reporting of politically controversial findings.

Childhood abuse and later medical disorders in women: an epidemiological study. Romans S et al. Psychotherapy Psychosomat 2002; 71: 141-150. From the review by Dr Berkowitz in the *Quarterly*:

Statistically significant associations were found in chronic fatigue, asthma, cardiovascular disease, and bladder problems. Interestingly, irritable bowel syndrome and pelvic pain, which had been previously associated, were not statistically significant.

The legacy of child maltreatment: long term health consequences for women. Ileana Arias, PhD. J Women's Health 2004; 13(5): 468-473. Abstracted on Medscape.

Incidence and prevalence:

A 1998 meta-analysis of 22 epidemiologic studies by Becky Bolen (bbolen@bu.edu) and Maria Scannapieco found a small increase in sexual abuse prevalence in recent years for females only. Found that the prevalence was 24-40% for females and 3-13% for males.

Epidemiology of sexual abuse of children: old problems, new directions. Leventhal JM. Child Abuse & Neglect 1998; 22(6): 481-491. A literature review by a well-known academic researcher.

The prevalence and circumstances of child sexual abuse: changes across a decade. Wyatt GE, Loeb TB, Solis J, Carmona JV. Child Abuse Negl 1999; 23: 45-59. (Los Angeles County). Prevalence has remained stable 1984-94. Rates of disclosure increased for AA women but decreased for W women.

Decrease in sexual abuse substantiations. 1999: Lisa M. Jones, PhD, Crimes Against Children Research Center, University of New Hampshire, 126 Horton SSCtr, Durham, NH 03824. Lmjones@cisunix.unh.edu David Finkelhor and I are currently exploring possible reasons for the decreasing trends in sexual abuse substantiations. Most states show a generally decreasing trend since about 1992, but a few states show a dramatic decrease over a 1 year period. It likely that for these states, the decrease is at least partly due to administrative changes, policy changes, or other statewide events... Reply by Anne E. Hollows A.E.Hollows@shu.ac.uk citing a 1999 review of research on CSA: Child Sexual Abuse -- Informing Practice from Research, by Jones DPH and Ramchandi P, HMSO 1999. Anne says that there has been a ratcheting down of prosecutions because social workers are increasingly concerned at the impact upon children of delays as well as adversarial practice within the prosecution of abusers. [JKR note: see under Epidemiol of child abuse, above, for decreasing incidence of all forms of reported abuse from the peak level of 1993. DHHS figures, not clear whether reports or substantiations.]

CSA cases have declined 31% since 1992. Dr. Finkelhor and Dr. Lisa Jones for the US DOJ. Substantiations declined 31%. US DOJ Office of Juvenile Justice and Delinquency Prevention. The Decline in Child Sexual Abuse Cases. [Bulletin] Washington, D.C.: Department of Justice, 2001. NCJ 184741.

Why is sexual abuse declining? A survey of state child protection administrators. Jones LM, Finkelhor D, Kopiec K. Child Abuse Negl 2001 Sep; 25(9): 1139-1158. Substantiations have declined 39% from 1992 to 1999. These guys did telephone interviews of child protection administrators in 43 states to ask why they thought this was occurring in their states. More than half of the officials were unaware that it was occurring. The state officials suggested the following possible reasons:

1. increased evidentiary requirements to substantiate sexual abuse
2. increased caseworker caution due to new legal rights for caregivers
3. new limits on the types of reports agencies accept for investigation
4. effective prevention programs
5. increased prosecution
6. increased public awareness

Commented on in: Re: Why is sexual abuse declining? A survey of state child protection administrators. (Jones, Finkelhor & Kopiec, 2001). Chadwick DL. Child Abuse & Neglect 2002 Sep; 26(9): 887-888, with authors' reply.

Putting together evidence on declining trends in sexual abuse: a complex puzzle. Jones LM, Finkelhor D. Child Abuse & Neglect 2003 Feb; 27(2): 133-135. [Finkelhor is the leading scholar of child abuse epidemiology in this country. JKR] See above and next below.

Is child sexual abuse declining? Evidence from a population-based survey of men and women in Australia. Dunne MP, Purdie DM, Cook MD, Boyle FM, Najman JM. Child Abuse & Neglect 2003; 27(2): 141-152. Yes it is. This was a self-report survey of men and women.

Interviewing:

The American Prosecutors' Research Institute (APRI) has published numerous short monographs on interviewing and sponsors annual seminars.

Practice parameters for the forensic evaluation of children and adolescents who may have been physically or sexually abused. American Academy of Child and Adolescent Psychiatry. J Am Acad Child Adolesc Psychiatry 1997; 36: 423-442. Cited as a standard reference on interviewing by the AAP, 1999. See also APSAC Guidelines, 1990.

Guidelines for Psychosocial Evaluation of Suspected Sexual Abuse in Young Children. Chicago: APSAC, 1990. Cited by AAP, 1999 as a standard reference.

Focused questions for interviewing children suspected of maltreatment and other traumatic experiences. Kathleen Coulborn Faller, PhD, ACSW. APSAC Advisor 1999 Spring; 12(1): 14-18.

Defending the investigative interview. Victor I. Vieth, Senior Attorney, APRI National Center for Prosecution of Child Abuse. APRI Update, vol. 12, no. 2 (1999).

False allegations of pregnancy resulting from incestuous rape and physician misconduct: proof positive. Jamieson MA, Walker M, Daicar AO, Reid RL. J Pediatr Adolesc Gyn 1998 Nov; 11(4): 181-184. This female said she had been raped by her gynecologist and got pregnant and had to have an abortion. It turned out she has uterovaginal agenesis.

Reliability and credibility of young children's reports: from research to policy and practice. Bruck M, Ceci SJ, and Hembrooke H. American Psychologist 1998; 53(2): 136-151. Reviews the research on suggestibility, and the effect of interviewing techniques.

The new wave in children's suggestibility research: a critique. Lyon TD. 84 Cornell L Rev 1004 (1999). Children are not as suggestible, or at least not as often suggested, as recent research would suggest. Even adults are suggestible.

On the assessment of children in suspected child sexual abuse in light of Daubert and Frye: limitations of profiles and interviews as scientifically grounded evidence. Bigelow BJ. J Forens Sci 2000 May; 45(3): 573-581.

Pedophile confessions closely match allegations of child victims. (Reuters Health News on Medscape May 18, 2000.) In children over 6 years of age, Amy A. Daso, a medical student at Case Western, compared the confessions of 31 pedophiles with the statements of 435 children. The 101 confessed acts of abuse closely

matched 68% of the victims' allegations. Author says, "Once a child reaches his or her 6th birthday, [other] studies suggest they are less suggestible by leading questions."

The decline in child sexual abuse cases. Jones L and Finkelhor D. Juvenile Justice Bulletin 2001 Jan; 1-12. Nationwide and broad 31% decrease in substantiated cases from 150,000 to 103,000 1992-1998. Physical abuse declined 16%. Considers it could be both a real decline and a decline in reporting.

Study: Fact and fiction cloudy for children. AbcNEWS.com, March 28, 2001. Reporting on work by Debra Anne Poole, professor of psychology at Central Michigan University in Mount Pleasant, co-author of the study in the Journal of Experimental Psychology. 114 children between 3 and 8 interacted with a man called "Mr. Science," who showed them science demonstrations. Three months later, the researchers gave the children's parents a book to read to the children about these demonstrations. The book contained a statement that "Mr. Science" had touched the children. When later interviewed by researchers, 35% of the children reported the touching as fact. "The results reinforce the concern of forensic experts about the difficulty children sometimes have in distinguishing real and suggested events, especially if they have been previously exposed to suggestions." To improve the reliability of older child witnesses, interviewers will have to be able to tease out whether a child has been exposed to several sources of information about an event. Younger children cannot distinguish between knowing and remembering.

Roundtable discussion: The medical evaluation of sexually abused children. Muram D. J Pediatr Adol Gyn 2003; 16: 5-14. See below under "General."

General

Sexual abuse, another hidden pediatric problem. The 1977 C. Anderson Aldrich Lecture. Kempe H. Pediatrics 1978; 62: 382-389.

Sexual abuse of children: a clinical spectrum. Summit R and Kryso J. Am J Orthopsychiatry 1978 Apr; 48(2): 237-251. See above under "Classic Articles."

Discusses twelve forms of incest and why it is harmful. Regarded as a classic in the field; summarizes Dr. Summit's own conclusions and those of others up to 1977.

Abstract: "This paper suggests that incest has been underestimated as a significant determinant of emotional disturbance, and that misuse of sexuality between parents and children can have detrimental consequences that parallel those resulting from other forms of child abuse. The spectrum of parent-child sexuality is classified into ten categories as a guide to the diagnosis, management, and prognosis of sexually abusive behavior."

The discussion is from a psychoanalytic point of view. The ten categories are as follows:

1. Incidental sexual contact. This is spontaneous sexual responses in the parent stimulated by incidental exposures in household life
2. Ideological sexual contact. This is where parents allow or encourage the exposure of a child to sexual activity or materials out of an ideological belief that it would be harmful to conceal it.
3. Psychotic intrusion. This is where the parent is psychotic, and imagines that the child is a participant in some kind of sexual process, and may actively involve the child in associated behavior.
4. Rustic environment.
5. True endogamous incest. (endogamous = within marriage). "The father is the key to the disturbed dynamics and is responsible for the choice to eroticize the relationship with the daughter." 242. Role distortion.
6. Misogynous incest. "The offender has a history of conflict with his own mother, and a tendency toward violence and punishment of women.... The daughter is seen as a possession, and possessing

her sexually is an assertion of his invulnerability to the control of women as well as an act of punitive defiance toward his wife." 245.

7. Imperious incest. "These men set themselves up as emperors in their household domain." 245
8. Pedophilic incest. "Some people have an erotic fascination with children. Males especially have the proclivity to retreat from castration fears and discomfort with peer relationships in search of a sex object they consider more innocent and less threatening." 246
9. Child rape. "Most pedophiles are gentle creatures. They cherish tenderness and innocence, and will back off from fear and resistance in their intended partner. The child rapist, confusing masculinity with power, can feel sexually adequate only by frightening and overpowering his victims. His need to punish, his attraction to violence, and his poor impulse control, all coupled with perverse guilt and fear of discovery, put the child in extreme physical danger." 246
10. Perverse incest. Pornographic. "kinky, unfettered lechery." "Forbidden fantasies."

The authors go on to discuss EFFECTS of sexual abuse. "Defining such intrusions as abusive requires that they bring harm to the child." 249. "We believe harm results from the perception by the child that the sexuality is socially inappropriate and that the relationship is exploitative." 249 "We are convinced from clinical and consulting experience that incest can do substantial harm..." (goes into detail). Discusses treatment through peer-group supervision as producing successful rehabilitation.

But, as to degree of harm from CSA, compare the 1998 meta-analysis by Rind et al., *Psychological Bulletin* 1998; 124(1): 22-53, showing a failure to establish effects on adult social adjustment attributable to CSA.

The child sexual abuse accommodation syndrome. Roland C. Summit. *Child Abuse & Neglect* 1983; 7:177. Identifying recantation as the standard course the child victims usually follow once the consequences of their disclosure become apparent to them. A copy of this article appears as an appendix to the two-volume ring-binder manual, *Investigation and Prosecution of Child Abuse, 2d ed.*, put out by APRI.

The traumatic impact of child sexual abuse: a conceptualization. Finkelhor D, Browne A. *Am J Orthopsychiatry* 1985 Oct; 55(4): Propounding **the traumagenic model**:

1. Traumatic sexualization means that the child's sexualization develops in an abnormal direction.
2. Sense of betrayal by a trusted older person
3. Victim starts to feel powerless because the older person has taken control
4. Stigma. Victim senses the shame of it and incorporates shame into their self-image.

Forensic evaluation of the sexually abused child. Enos WF, Conrath TB, and Byer JC. *Pediatrics* 1986 Sep; 78(3): 385-398

Buggery in childhood -- a common syndrome of child abuse. Hobbs C and Wynne J. *Lancet* 1986; 792-796. Describes the signs with color illustrations. For details of disclosure by these victims see:

Child sexual abuse: an increasing rate of diagnosis. Hobbs C and Wynne J. *Lancet* 1987; 837-841. See also for the same data in American journal:

Genital findings in sexually abused symptomatic and asymptomatic girls. Emans SJ, Woods ER, Flagg NT, Freeman A. *Pediatrics* 1987; 79: 778-785

Configuration and other anatomic details of the prepubertal hymen. Pokorny SF and Kozinetz CA. *Adolescent and Pediatric Gynecology*, 1988; 1: 97-103. Cited by McCann (1990), Kerns (1992), and Bays & Chadwick (1993) as a foundational study.

Sexual abuse of English boys and girls: the importance of anal examination. Hobbs C, Wynne J. *Child Abuse & Neglect* 1988 or 1989; 13: 195-210. Cited by Bruni, 2003, for the proposition that differences in examination position contribute to dispute over the anal findings in chronic abuse, and that there are differences of opinion over what constitutes normal.

Is it sexual abuse? Adams JA and Horton M. Clin Pediatr 1989; 28: 146-148

Anogenital trauma in sexually abused children. Finkel MA. Pediatrics 1989; 84: 317-322

Child sexual abuse: relationship between sexual acts and genital findings. Muram D. Child Abuse & Neglect 1989; 13: 211-216

Anal and perianal abnormalities in prepubertal victims of sexual abuse. Muram D. Am J Ob Gyn 1989 Aug; 161: 278-281.

	Abnormal	Normal
+Assault Hx	84%	16%
-Assault Hx	15%	85%

Describes the anal and perianal findings in 310 children aged 1 to 10 who were determined to be victims of sexual abuse. Overall, 66% of them were normal and 34% abnormal. Of the abnormalities, had

Anal gaping	61 cases
Skin tags	44 "
Rectal tears	33 "
Sphincter tears	15 "
HPV	4 "
Perineal scars	2 "
Bite marks	1 "

"Although we were able to detect a large number of children with anal and perianal abnormalities, it is difficult to conclude from this study which of these findings are diagnostic of anal abuse.... More controlled studies are required to identify the specific findings indicative of anal assault...." 281

"The findings of the medical examination must be correlated with other methods of validation such as a history from the child. To conclude from the findings of the medical examination alone that a child is a victim of abuse may cause enormous harm to the child and the family." 281.

Perianal findings in prepubertal children selected for nonabuse; a descriptive study. McCann J, Voris J, Simon M, Wells R. Child Abuse & Neglect 1989; 13: 179-193. Cited along with Berenson et al., 1992 and Heger et al., Atlas, 2000 by Bruni, 2003, for the proposition that midline tags can be congenital.

Genital findings in prepubertal girls selected for nonabuse: a descriptive study. McCann J, Wells R, Simon, Voris J. Pediatrics 1990 Sep; 86(3): 428-439.

114 normal subjects aged 10 months to 10 years. Some "abnormalities" were normal.

Introitus:

56%	erythema of the vestibule
50%	periurethral bands
39%	labial adhesions
34%	lymphoid follicles of the fossa navicularis
26%	avascular areas on the posterior fourchette

Hymen

Mounds & projections	33%
Midline tags	18%

Vaginal ridges

90%

This was to establish normative genital anatomy. In addition to the above common findings, unusual findings included posterior fourchette friability (4.7%), anterior hymenal clefts (1.2%), notches (6%). A vaginal discharge was detected in 2 and a foreign body in 1. The labial adhesions were very small, 2mm; examiner traction caused them to blanch or to dehisce, resulting in a small amount of bleeding. Periurethral bands discovered in half the children created a false pocket on either side of the meatus. "The same band-like structures were also noted in the posterior portion of the vestibule when asymmetry of the posterior vaginal fossa (fossa navicularis) was present." The different types of hymen found in this population are similar to those described by Pokorny (1987). Most were crescentic, i.e. with a bilateral attachment anteriorly and continuous attachment posteriorly. Configuration varied with age and method of examination. "The influence of the maternal hormones caused the hymen to remain thick and redundant up to 2 years of age and occasionally beyond. Similar changes began to reappear as the child approached puberty but before either breast tissue changes or the appearance of pubic hair. The redundant appearance of the hymen was altered through the application of traction on the labia or through the use of the knee-chest position." 437 There was good interobserver agreement on the more objective findings, less on the subjective ones such as the amount of erythema. "Even in our present state of knowledge, it is becoming increasingly evident that, as a consequence of naturally occurring physical changes, there will always be an overlap in findings between nonabused children and the victims of sexual misuse. The appreciation of this reality should serve as a constant reminder that the determination of sexual abuse can rarely rely on a physical examination alone and that consideration of all the components of the investigation -- especially the information obtained from the child -- is essential." 438

Cited by Bruni, 2003, for the proposition that anal scars outside the midline can be an indicator of remote abuse, especially if multiple.

Genital and anal conditions confused with child sexual abuse trauma. Bays J and Jenny C. Am J Dis Child 1990; 144: 1319-1322. See also LSA article by Wood & Bevan, BMJ 1999, below.

Perianal appearances associated with constipation. Agnarsson U, Warde C, McCarthy G, Evans N. Arch Dis Child 1990; 65(11): 1231-1234. Cited by Bruni, 2003, for the proposition that "It is agreed that constipation is a common cause of fissures, which can result in scars; in this case the most typical area is the anterior midline and the mean age is 2-3 years."

Normative sexual behavior in children. Friedrich WN, Grambsch P, Broughton D, Kuiper J, Beilke RL. Pediatrics 1991; 88: 456-464. See also their "behavior inventory" paper, next below (1992).

Listed as a "must read" article by McCann.

Behaviors were studied relative to class, age, etc. in 880 children aged 2 - 12 years. Using the Child Sexual Behavior Inventory.

Sexualization peaked at age 5.

Older children were less sexual than younger children.

0.8%	masturbates with object
1.1%	imitates intercourse
1.4%	sexual sounds
2.5%	French kisses
2.6%	undresses other people
2.7%	asks to watch explicit television
5.7%	talks about sexual acts
6.0%	touches others' sexual parts
8.1%	shows sexual parts to other children
8.8%	uses sexual words
15.3%	masturbates with hand
16.0%	shows sexual parts to adults
19.7%	touches sex parts in public

36.4% sits with crotch exposed
 38.7% shy about undressing
 64.5% shy with strange men

Sexuality was not related to socioeconomic variables.

Small bowel evisceration: unusual manifestation of child abuse. Press S, Grant P, Thompson VT, Milles KL. Pediatrics 1991 Oct; 88(4): 807-809. Evisceration of small intestine through a 3 cm tear in the anterior rectosigmoid produced when the 14 year old sister forcibly inserted the handle of a toilet plunger into the rectum of this 4 year old boy. The rectosigmoid was operatively repaired; child did well.

The hymen and Tanner staging of the breast. Yordan EE and Yordan YA. Adolescent and Pediatric Gynecology 1992; 5: 76-79

Genital injuries resulting from sexual abuse: a longitudinal study. McCann J, Voris J, Simon M. Pediatrics 1992; 89: 307-317

Three girls with clearcut hymenal or posterior fourchette tears were selected for longitudinal photographic followup. The article presents series of colposcopic photographs at intervals of followup for three years. They had to use a Q-tip to separate the tissues. Discusses healing by regeneration and repair. Found that the hymen heals only by regeneration; does not form scars. Even the posterior fourchette had only slight scarring (but to my view the scar is clearcut. JKR)

All hymeneal and superficial injuries healed within 1 week.

"In summary, the genital injuries of these children healed rapidly. Over time, the jagged, irregular margins of the hymen created by these acute injuries smoothed out and became difficult to detect without the use of a multimethod examination technique. The changes did remain stable while the children were in Tanner stage I. However, the narrow hymeneal rim at the point of the injury, which was a persistent findings, disappeared as the 9 year old entered puberty." 310

"Following these single isolated episodes of abuse, the hymeneal injuries healed with a minimum of scar tissue formation, and even the deep lacerations of the posterior fourchettes left little evidence of the trauma they had suffered. The difficulty encountered in the detection of the signs of injury after healing had occurred served as a reminder of the importance of the history, the subtlety of the changes, and the challenging nature of the medical evaluation of the sexually abused child." 310

Concave hymenal variations in suspected child sexual abuse victims. Kerns DL, Ritter ML, Thomas RG. Pediatrics 1992 Aug; 90(2): 265-272. Photocolposcopy was done on 1383 female child and adolescent suspected sexual abuse victims. From the photographs in the article it is apparent that by "concave hymenal variations," these authors mean what other authors term "notches." Concave hymenal variations were found in 12%. Of these, 35% were anterior, 57% were posterior or lateral.

Posterior or lateral notches were associated with a history of penile-vaginal contact ($P=.004$, penetration ($P=.006$), stranger as perpetrator ($P=.032$), hymenal rim narrowing ($P=.001$), and posterior fourchette abnormality ($P=.049$). Angular configuration was associated with the same things. Logistic regression by age indicated that these get more common with age, therefore consistent with an acquired, rather than a congenital, condition.

The colposcopic photographs depict as follows:

Fig. 1: an anterior curved concavity 12:00 to 2:00 in a 2 ½ y o with complains of genital pain and vague allegations of sexual contact with her father. Noted are normal vaginal ridges inserting on the hymen at 10:00 and 2:00

Fig. 2: Anterior angular smooth-edged concavity in a 3 y o w neglect and vague allegations of sexual contact

Fig. 3: Anterior 12:00 to 2:00 curved irregular hymenal concavity in a 6 y o w markedly sexualized behavior and anonspecific hx of sexual contact with a 13 y o male

Fig. 4: Lateral 2:00- 4:00 curved smooth cup-shaped concavity in a 5 y o w sexualized beh and unclear allegations

Fig. 5: Posterior smooth angular concavity with a hx of multiple painful ep of digital penetr

Fig 6: Posterior angular irregular concavity in a 12 ½ y o w hx of date rape with painful penile penetr. This is a frank acute transection with a hematoma of the vaginal floor. It provides an instructive comparison with the other, especially anterior, lesions.

Conclusions: "Multiple lines of evidence support the hypothesis that **posterior/lateral location, angular contour, and rim irregularity** of these concavities are associated with trauma." Emphasizing that "Reaching a comprehensive medical diagnostic impression requires integration of these findings with the rest of the general and anogenital physical examination...and most importantly, with detailed historical information, particularly the histories given by the children themselves." 271 Comment by JKR: This article is highly instructive because of the quality of the figures and the detailed breakdown of interpretations given by different blinded observers. It made a significant contribution to advancing the field.

Appearance of the hymen in prepubertal girls. Berenson AB, Heger AH, Hayes JM, Bailey RK, Emans SJ. Pediatrics 1992; 89: 387-394

211 normal girls 1 month to 7 years. Colposcopic photography.

Labial agglutination was common, mostly under 12 months.

Notches were generally anterior 11:00 - 1:00.

There were three shapes of hymens considered normal: annular, fimbriated, and crescentic. These were age-related. Fimbriated was common under 12 months (? Estrogen effect?). Most girls over 2 years had crescentic hymens (? Non-estrogenized ?).

Notches between 4:00 and 8:00 were never observed in normals.

Anterior notches were common in normals. (9:00 to 3:00)

Midline tags, either genital or anal, can be congenital. (Bruni, 2003)

The diagnosis of child sexual abuse. Dubowitz H, Black M, Harrington D. Am J Dis Child 1992; 146: 688-693

Interdisciplinary evaluations of alleged sexual abuse cases. Jaudes PK and Martone M. Pediatrics 1992; 89: 1164-1168.

Abuse of the child abuse accomodation syndrome. Roland C. Summit. J Child Sexual Ab 1992; 1(4): 153.

Child sexual behavior inventory: normative and clinical comparisons. Friedreich and Gramsbach, APA 1992; 4(3); 303-311. Oral-genital activity represented only 0.1% of all sexual behaviors observed.

A proposed system for the classification of anogenital findings in children with suspected sexual abuse.
Adams JA, Harper K, and Knudson S. *Adol Pediatr Gynecol* 1992; 5: 73-75 This proposed system consists of five categories:

- (1) Normal
- (2) Nonspecific
- (3) Suspicious
- (4) Suggestive
- (5) Clear

See below for later versions of this. Cited by Bruni, 2003, for the proposition that venous congestion is supportive of previous abuse, especially if it occurs during the first seconds of the examination. Agreeing with Hobbs, 1999 and disagreeing with McCann, Voris, 1989 about venous congestion in the chronic setting.

Medical diagnosis of the sexually abused child. Bays J and Chadwick D. *Child Abuse & Neglect* 1993; 17: 91-110. A short summary of this long review article can be found in the red paperback book by the Section on CAN, AAP, 2d ed., p 134.

A normal physical examination is common. Data the authors collected from 21 studies show that normal examinations were reported in 26% to 73% of girls (average 50%), and 17% to 82% of boys (average 53%). "Findings diagnostic of sexual abuse, for example, the presence of genital trauma, sexually transmitted disease, or sperm, were found only in 3% to 16% of the child victims [in the 21 studies]."

Reasons for lack of physical findings in sexually abused children:

- (a) "Delay in seeking a medical examination decreases the likelihood of positive findings. Rimza & Niggemann (1982) report that 36% of children examined within 24 hours of penetrating sexual assault had evidence of genital trauma, but only 13% had such evidence when seen after 24 hours." 94
- (b) Ejaculate unlikely to be found if ... washed ... or more than 72 hours.
- (c) "When injuries do occur as a result of sexual abuse, healing is rapid and often complete." 95
- (d) Many types of molestation...do not leave physical findings.
- (e) "Rape" means any penetration of the vulvar cleft, however slight. Therefore, "rape" can occur without ejaculation or damage to the hymen.
- (f) "Groth and Burgess (1977) reported that 34% of rapists of adult women had erectile or ejaculatory dysfunction." 94
- (g) "The anal sphincter allows routine passage of stools larger than the diameter of a penis without damage."
- (h) "Hymen tissue is elastic...and full penetration by a finger or penis, particularly in an older child, may cause no visible trauma or simply enlarge the hymenal opening."

DDX: "There are conditions that may mimic findings caused by sexual abuse." 95 "Examiners should be familiar with such conditions, as the consequences of a mistaken diagnosis of abuse can be serious for the child, family, and suspected perpetrator." 96

Vulvar

Lichen sclerosis*
Hemangioma
Streptococcal
Straddle
Prolapse
Anomalies

periurethral bands, intravaginal ridges, anterior midline perianal skin folds, perineal grooves, diastasis ani, or smooth, wedge-shaped areas in the midline of the anal verge.

Anal

Crohn's
HUS

Postmortem dil
 Neurogenic patulosity
 Lichen sclerosus
 Chronic constipation**

* "The most common dermatologic condition confused with trauma from sexual assault is lichen sclerosus. It manifests as alarming subepidermal hemorrhages of the genital or anal area following minor trauma such as wiping after toileting." 95

** "Fissures and anal dilation are found in constipated and also in sexually abused children. British studies indicate 6% to 26% of constipated children have anal fissures, and 15% to 18% have anal dilation." BUT "An American study of 171 children referred for gastrointestinal complaints, however, revealed anal or perianal abnormalities in only eight children (less than 5%). Only one of 30 children referred for constipation had an anal fissure. No child had anal dilation or distortion of the anal opening. The authors suggest that when perianal abnormalities are detected, further evaluation for possible sexual abuse is warranted. (Lazar & Muram, 1989).

Hymens vary. Pokorny and Kozinetz (1988) studied hymen configuration in 265 prepubertal girls... They propose three basic hymen types: fimbriated, ring, and crescentic. "A recent study indicates that as girls mature, the appearance of the hymen shifts from predominance of annular or concentric hymens in preschoolers to predominance of crescentic configurations in preadolescents." 96

"The appearance of the hymen can be altered by trauma or a variety of nontraumatic factors." 96 Different examining techniques and positions. Estrogen: "Maternal hormones render the hymen thick, pink-white in color, and redundant to 2 to 4 years of age. Hymens in latency-age girls are thinner, redder, and more sensitive to touch." 97

Accidents, masturbation, and use of tampons are very unlikely to cause injury to the hymen or internal genital structures.

Hymen: abuse injuries:

- Erythema
- Narrowed rim
- Notch
- Disruption
- Most injuries are found from 3:00 to 9:00.

Three findings that strongly indicate abuse, according to the National Child Sexual Abuse Summit Meeting in 1985:***

1. "Clearcut hymenal damage or disruption consisting of tears, fresh or old; scars, significant distortion of the normal shape, and/or hymenal bruising.
2. "Injuries in the region of the posterior fourchette consisting of lacerations or the scars of healed lacerations, bruises, and healing abraded areas often accompanied by a growth of new blood vessels called neovascularization.
3. "An anus that dilates to greater than 15 mm transverse diameter with very gentle buttock traction with the child in the knee-chest prone position, or an anus that demonstrates large scars..." 105

"Findings consistent with sexual abuse: history and other investigation may be important in diagnosing abuse:

1. Genital or anal Trichomoniasis, Chlamydia, Condyloma acuminata, or Herpes II not congenital.
2. Disruptions of hymen tissue including posterior or lateral angular concavities (also termed clefts or notches), transections, absence, decrease in amount, and scars.
3. Anal scars outside the midline
4. Anal tags outside the midline

5. Anal dilation over 15-20 mm without stool in the ampulla
6. Irregularity of the anal orifice after complete dilation
7. Marked dilation of the hymenal opening, persisting in different exam positions." 106

"Findings sometimes seen following sexual abuse but also other causes:

1. Bacterial vaginosis
2. Extensive labial adhesions
3. Posterior fourchette friability
4. Repeated anal dilation less than 15 mm
5. Edema of the perianal tissues
6. Shortening or eversion of the anal canal
7. Perianal fissures
8. Thickened perianal skin or reduction of skin folds
9. Penile erection maintained during examination in prepubertal boys." 106

"Findings unlikely to be due to abuse:

1. Candidal dermatitis
2. Small labial adhesions
3. Erythema of the vestibule
4. Periurethral bands
5. Lymphoid follicles of the fossa navicularis
6. Midline avascular areas of the fossa navicularis or posterior fourchette
7. Urethral dilation with labial traction
8. Small hymenal mounds, projections with otherwise normal hymen
9. Concavities of the hymen that are anterior and/or smooth, curved, and shallow
10. Intravaginal ridges behind a normal hymen
11. Imperforate hymen
12. Perianal erythema
13. Perianal increased pigmentation.
14. Perianal venous engorgement after 2 mins in knee-chest position
15. Skin tags/fold in the midline perianal
16. Smooth areas in the midline perianal
17. Anal dilation with stool in the ampulla
18. Flattening of the anal rugae during dilation.

"Above all, clinicians should remember that the physical exam is only a part of the evaluation of a child for possible abuse." 107

Normal versus abnormal genital findings in children: how well do examiners agree? Adams J & Wells, Child Abuse & Neglect 1993; 17: 663. Cannot diagnose sexual abuse without the history.

Medical evaluation of the sexually abused child. Carolyn J. Levitt. Peds Clin N Amer 1993 Jun; 20(2): 343-354

Perianal injuries resulting from sexual abuse: a longitudinal study. McCann J and Voris J. Pediatrics 1993; 91: 390-393.

To remind clinicians of the remarkable healing process that takes place. Four children aged 4 months to 12 years were followed photographically. On initial examination, all four had lacerations and erythema. 3/4 had edema and venous congestion and thickened or flattened anal folds. 2/4 had slight dilatation.

Healing by regeneration was complete in 72 hours.

Healing by repair took 60 days.

Acute signs had disappeared in 8 days.

Laceration healing times were estimated:

Superficial lacerations	1 to 11 days
Second degree lacerations	1 to 5 weeks with narrow scars
Third degree lacerations	12 to 14 months gradually fading

Hymenal findings in adolescent women: impact of tampon use and consensual sexual activity. S Jean Emans et al. Pediatrics 1994; 79: 778-785. Tampon use has no effect. This paper is attacked by Goodyear-Smith from New Zealand (1998) in a controversial diatribe, see below.

Colposcopic documentation: an objective approach to assessing sexual abuse of girls. Soderstrom RM. J Reprod Med 1994 Jan; 39(1): 6-8

Sexual abuse of the oral cavity in children. Heitzler GD, Cranin AN, and Gallo L. J Mich Dent Assoc 1994 Jul; 76(6): 28-30

The investigation of child sexual abuse: an interdisciplinary consensus statement. Lamb ME. Child Abuse & Neglect 1994; 18: 1021-1028 A group of twenty professionals, all but one of them psychologists (John McCann appears to be the only physician). The group's mandate was to evaluate existing knowledge regarding the ways in which child sexual abuse allegations could be investigated most productively. It concludes with a statement regarding the areas in which further research remains necessary before greater clarity can be achieved.

The problem: A rising rate combined with a high rate of nonsubstantiation. From 1985 to 1992, incidence of reported cases in US rose from 325,000 to 500,000.* "Approximately 40% of all child abuse reports are substantiated by social welfare authorities." (Reported incidence would be 0.7% of all the children in the USA as of 1992.) "...[T]he high rate of nonsubstantiation highlights the difficulty that may arise when attempting to validate allegations. These factors alone underscore the importance of developing reliable and valid means of investigating reports of child sexual maltreatment." 1022

* [Incidentally, since 1994 the report rate has been going down, as discussed by Dr. Finkelhor at the 1997 San Diego Conference. -- JKR]

Sexualized behavior is NOT a reliable indicator. It occurs in normals and we know too little about it. [Cf Faller, 1998, saying that sexualized behavior is considered a reliable criterion by many professionals but acknowledging that it overlaps with normals and needs further research. See below)

Interviewing child victims: can be extremely informative and accurate, but it is difficult. Most reliable if it occurs early. In an acute event description, look for "accounts that contain peripheral details and are logically embedded in a rich context." Use open-ended questions; but in children under 5, direct or focussing questions are needed; these must be nonsuggestive. "Repeated, highly leading, or suggestive questions asked in an accusatory manner are ill-advised because they are most likely to promote distortion on the part of the child and may introducedetails that are incorporated into and contaminate subsequent accounts." 1024

Using dolls and other props: ...

Medical examinations: "Over the last 15 years, there have been dramatic changes in the evaluation of medical evidence suggestive of child sexual abuse. Initial claims regarding the conclusiveness of medical exams were unfortunately overstated in light of professional ignorance regarding both normative anogenital development and the effects of trauma on tissues in the anogenital region. ... Contrary to popular belief, sexual abuse may leave few, if any, physical effects. Fondling, oral copulation, and other forms of sexual molestation that do not cause tissue damage may leave no signs other than a slight amount of redness that rapidly disappears. Likewise, gentle penetration of the rectum, even by relatively large objects, may cause no permanent tissue damage, especially when a lubricant is employed." 1025

"The size of the hymenal orifice is no longer viewed as a reliable indicator of abuse... [It] varies so widely from child to child..." 1026

"Penetration of a prepubertal girl's vagina by a penis or other large object usually results in a tear of her hymen and, at times, of the surrounding tissues as well. Recent hymenal tears are usually sharp-edged and V-shaped. They are most commonly found on the posterior rim of the hymen between the 3 and 9 o'clock positions, resulting in a narrow rim at the point of injury and exposure of the underlying intravaginal structures. Any bleeding or redness resulting from the abuse usually disappears within 48 hours. The tears themselves heal within 5 to 10 days and the sharp edges of the wounds smooth out and round off over a period of 3 to 6 months. Over time, these injuries become increasingly difficult to detect... As a result, the absence of physical signs does not prove that no abuse occurred. 1026

"If the child has been penetrated by a skillful perpetrator who has carefully avoided hurting the child or injuring the tissues by gradually stretching the hymen, an examiner may observe only a smooth-edged hymen with a narrow rim... Such deformations of the posterior hymenal rim do not occur naturally and are unlikely to be caused by any other known trauma, including straddle injuries." 1026

"Perianal injuries caused by sexual abuse occur relatively infrequently and are more difficult to detect. Although severe physical injuries can result from a violent act of sodomy, it is less clear what tissue changes should be expected following ongoing, relatively gentle penetration of the anal orifice when a lubricant is used. As a result, considerable controversy persists concerning the evaluation of perianal signs (including anal dilation reflexes) in children who may have experienced anal penetration. Other perianal soft tissue damage is similarly difficult to interpret when the history is unclear or the child is very young. As with genital trauma, perianal injuries heal rapidly, leaving little evidence of previous tissue damage." 1027

"Within the medical community, it is now widely recognized that physical examinations may not yield conclusive evidence that sexual abuse either did or did not occur. Further research is needed on the immediate physical consequences of specific sexual acts as well as on the developmental changes in these physical signs in the succeeding years. Such research will complement a growing but still inadequate understanding of normative anogenital development and variability, particularly in diverse ethnic and racial subgroups." 1028

Cited by Bruni, 2003, for the proposition that anal physical findings and their interpretation continue to be discussed.

Sexual homicide of children. (monograph). SSA Kenneth V. Lanning, FBI Academy, Quantico, Va. The APSAC Advisor 1994 Winter: 7(4)

Examination findings in legally confirmed child sexual abuse: it's normal to be normal. Adams JA Harper K, Knudson S, Revilla J. Pediatrics 1994 Sep; 94(3): 310-317

236 victims of sexual assault, aged 8 mos to 18 years. The assault in all cases was confirmed by judicial conviction of the perpetrator. Colposcopic photos were reviewed blindly.

Findings (vulvovaginal)		
	Normal	28%*
	Nonspecific	49%*
	Suspicious	9%
	Abnormal	14%
Findings (anal)		
	Normal	99%
	Abnormal	1%

* Note that only 10% of the patients were examined within three days of the molest. Cf the Consensus Statement (1994) (infra) that, "...medical examinations should be performed...as soon as possible after the alleged incidents." 1026

The two history factors that most strongly correlated statistically with an "abnormal" exam were (a) short time since the attack, and (b) history of blood.

Conclusions of the authors: "This study provides additional data that the majority of children with legally confirmed sexual abuse will have normal or nonspecific genital findings. Abnormal anal findings are very rarely found. The best predictors of abnormal genital findings in female victims are the time since the assault and a history that blood was reported or observed... A history of vaginal penetration given by the child did not significantly correlate with abnormal genital findings." 316

"This study also reaffirms that the history of the molest provided by the child is probably the most important evidence of sexual abuse. While widely accepted in the medical field, this fact is still not universally accepted in the legal arena... Health professionals who examine children must be as diligent in obtaining and recording the details of the child's statement as we are in recording the appearance of the hymen, and not be pressured to make a 'diagnosis' of sexual abuse based on medical findings alone." 316-317

Criminal investigation of sexual victimization of children. SSA Kenneth V. Lanning, FBI Academy, Quantico, Va. Published as Chapter 14, Section I in The APSAC Handbook on Child Maltreatment. Newbury Park: SAGE Publications, 1996.

Pediatric genitourinary trauma. McAleer IM and Kaplan GW. Urol Clin N Amer 1995 Feb; 22(1): 177-188. Deals with renal injuries; the kidney is the urogenital organ most frequently injured. Renal injuries are classified on a scale of I to V. Only grades IV and V need operative treatment. CT is the main diagnostic modality, with cystography and ureterography used for bladder and ureter injuries.

Child abuse and neglect. Lawrence S. Wissow, MD, MPH. (Depts. of Pediatrics and Psychiatry, Johns Hopkins) N E J Med 1995 May; 332(21): 1425-1431. This short general review deals with both physical abuse and sexual abuse.

He gives a definition of sexual abuse: "Sexual abuse includes the inappropriate exposure of a child to sexual acts or materials, the passive use of children as sexual stimuli for adults, and actual sexual contact between children and older people." 1425.

He lists the required elements of a basic psychosocial history (table 1).

On the issue of child credibility: Citing Paradise, 1988 and Everson, 1989 for the proposition that less than 10% of spontaneous disclosures to physicians by children were found to be false. "Very young children (of preschool and early school age) seem more vulnerable to distortions of memory than do older children or adults..." "Children, probably even more than adults, sense the answers an interviewer seems to be looking for. In an effort to please, a child may accept an interviewer's statements as fact even when they contradict what the child has witnessed. Children frequently interpret repeated questions as a sign that their prior answers have been wrong." 1426-7. Discusses also the typical content and progress of a disclosure. As many as 25% of children recant their disclosures; Wissow briefly discusses why this happens and how to prevent. (This applies to both physical and sexual abuse).

Controversies in the diagnosis of sexual abuse: "Initially, an enlarged hymenal opening...was considered a good indicator of trauma. Subsequent studies, however, have found considerable overlap in hymenal diameter between children who have been abused and those who have not, as well as variation in the diameter according to examination technique and the state of relaxation." 1429 "It may have some bumps and irregularities, but disruptions in the posterior aspect are rarely found in children who have not been abused and warrant further investigation for possible trauma. Many sexually abused children have no definitive lesions, because they are examined long after healing has occurred or because maltreatment did not result in genital or rectal injury." 1429.

He discusses the controversy over STD's, saying that it is inconclusive.

Table 2 is a list in tabular form of "signs and symptoms that should arouse concern about child abuse or neglect." (meaning physical abuse).

A longitudinal study of hymeneal morphology in the first three years of life. Berenson AB. Pediatrics 1995 Apr; 95(4): 490-496 To document the effects of growth and development. 134 subjects photographed. Changes in hymeneal morphology were observed in 65% of the subjects, usually from

annular or fimbriated to crescentic. Anterior notches decreased. There were no posterior notches. Intravaginal ridges increased. Nine tags formed. Four subjects initially had superior notches; superior notches evolved into crescentic hymens. A crescentic hymen is one with NO TISSUE ANTERIORLY. "Although not present at birth, the crescentic configuration has been documented to develop by 1 year of age in approximately one fourth of female children. This study demonstrates that this trend continues in years 2 and 3. Furthermore, the hymen of 75% of the subjects shifted from thickened and redundant to nonredundant with sharp edges between birth and three years. Studies on older subjects have reported that the hymen thickens and becomes redundant again with overlapping folds as the child enters puberty, confirming that hymenal morphology is dependent on estrogen status." 494-5

The push for disclosure: ideology and adaptation in the care of young molestation survivors. Harper G and Walzer S. Harv Rev Psychiatr 1995 May; 3(1): 50-51

Photographic documentation of child abuse. APSAC. Chicago, IL., 1995. Cited by Muram, 2003, as the standard of care today.

Twenty years in the evaluation of the sexually abused child: has medicine helped or hurt the child and the family? Heger AH. Child Abuse & Neglect 1996; 20: 893-897

Gives a history of the medical response to the problem. Hymeneal diameters failed bc they turned out to be traction- and position-dependent. In 1985 colposcopic photography became the standard of care and the basis of most research. "In parallel with all the research into the medical findings of abuse grew a very clear understanding of what was normal." 894 (citing Berenson & Heger, 1991, 1992; McCann, 1989, 1990). However, there was a downside to the success of colposcopic photography which was not apparent at the time. "[The courts] were relieved that they did not need to rely solely on a child's testimony. Requests to have the child examined with 'the magic machine' were common. The system began to expect medical abnormalities in order to pursue a case, and the history from the child became less important." 895 From this it went to using exam alone where there was NO history of abuse from the child and where the child actually denied any abuse, only a symptom, or else a history of abuse from an adult. "Inappropriate interventions might result." "This fueled a backlash and negatively impacted the ability of many to appropriately protect children who had been abused." 895. "History from the child continues to be the most important part of any evaluation." 895.

"Medical evaluators have come to an agreement on clinical findings which are diagnostic of sexual abuse as well as what is normal. (Gardner, 1992; Heger & Emans, 1992; McCann, 1993)" 895

"We have helped when we practice as part of a team, promoting a conservative and appropriate diagnosis and treatment... However, the medical diagnosis should not replace the child's history and be used to define abuse when it may not have occurred. This misuse of the medical diagnosis hurts the child and the family and results in the separation of children from families, and creates a sense of backlash and fear in the community, forcing the denial of child sexual abuse as a reality." 896.

Postmortem perianal findings in children. McCann J, Reay D, Seibert J, Stephens B, and Wirtz S. Am J Forens Med Path 1996 Dec; 17(4): 289-298. Studied autopsy anuses in 65 decedents selected for nonabuse. Dilatation and exposed pectinate line common. Thus the opinion of Hobbs & Wynne (1989) that postmortem "gross anal dilatation" and "irregular anal margin, smooth perianal skin" indicate abuse is not supported, as these findings were common in this study.

Don't confuse the exposed pectinate line with lacerations.

Lacerations and fissures were NOT found in normals in this study.

"A study dealing specifically with the autopsy findings of children known to have been sodomized prior to death is needed to help identify the soft-tissue changes that occur as a result of sexual abuse. However, based upon the results of this preliminary report, it is suggested that postmortem perianal findings must be interpreted with caution. Standards of normal are not yet firmly established." 297

The role of photo documentation of genital findings in medical evaluations of suspected child sexual abuse. Joyce Adams. Child Maltreatment 1997 Nov; 2(4): 341-347

Clinician agreement on physical findings in child sexual abuse cases. Sinal SH, Lawless MR, Rainey DY et al. Arch Pediatr Adol Med 1997 May; 151: 497-501 This study used a larger sample (139 sets) of colposcopic photographs of Tanner 1, 2 girls referred to a general pediatric clinic for evaluation of possible sexual abuse. Its purpose was to use a larger sample than previous studies which used small samples. (Joyce Adams in 1992 used 16 photographs; Muram in 1991 used 6 photographs.) They used the 1992 Adams system of classification of anogenital findings (see Adams, Harper, Knudson, above). So using 139 sets of colposcopic photographs, they submitted these to two groups of experienced clinicians (four clinicians in group A and three clinicians in group B). Group A was blinded to the history; group B knew the history. The examiners were paired on each set of photographs. Agreement on the assigned score was recorded for each pair of examiners as to each of the 139 photo sets. Perfect agreement occurred for 29% of the physician pairs from group B (knowing the history) and 39% from group A (blinded); in other words, agreement was higher without the history (!) The authors computed a kappa score of degree of agreement based on their Adams scale of 1 - 6 (6 was "uninterpretable"). They report that "The kappa scores indicated poor agreement among clinicians. The overall kappa score was .20." (p. 499)

Assessments of girls' genital findings and the likelihood of sexual abuse: agreement among physicians self-rated as skilled. Paradise JE, Finkel MA, Beiser AS, Berenson AB et al. Arch Pediatr Adol Med 1997 Sep; 151: 883-891. They mailed seven simulated cases, including one photograph of each case, to a sample of pediatricians and received usable responses from 414 pediatricians, of whom 206 rated themselves as "skilled" in assessing children for sexual abuse and 208 did not. The "skilled" group was subdivided by level of experience as very experienced and less experienced. The resulting diagnoses were compared with a consensus standard developed by the authors. Of the self-rated "skilled" physicians, 73% conformed in their diagnostic interpretation to the consensus standard, although only 45% selected descriptions of the findings that conformed to the consensus descriptions. The authors conclude, "Our data suggest that physicians who consider themselves skilled...not only conform variably with consensus standards, but also often differ among themselves in describing and interpreting girls' genital findings. ... Taken together, the variability we observed in physicians' assessments and the paucity of scientific information about the predictive value of individual genital findings for specific sexually abusive physical contacts suggest that conclusions about the likelihood of sexual abuse based largely or solely on the appearance of girls' external genitalia should be formulated with caution." (p. 890) My editorial: In other words, even if the skilled physicians had agreed completely on the findings, the meaning of those findings is not scientifically established and therefore the diagnostic interpretation re sexual abusive acts would be questionable even if agreement were complete.

Pediatrics and child sexual abuse: where we've been and where we're going. (editorial) Carol Jenny. Pediatr Ann 1997 May; 26(5): 284-286. Need to build a knowledge base and train young doctors.

Examination for sexual abuse in prepubertal children: an update. Botash AS. Pediatr Ann 1997 May; 26(5): 312-320. Intended for the general pediatrician this advocates including genital exam in every complete physical and early referral to a center in abuse cases.

Robbed of innocence: signs and symptoms of pediatric sexual abuse may be subtle. Gausche M. Emerg Med Serv 1997 Mar; 26(3): 39-42

Ethical issues in professionals' response to child maltreatment. Howard Dubowitz. Child Maltreatment 1997 Nov; 2(4): 348-355. See below under "Unclassifiable."

Common errors in forensic pediatric pathology. Sturmer WQ. Am J Forens Med Path 1998; 19(4): 317-320 at 318: "Failure to appreciate postmortem rectal changes resulting in erroneous diagnoses of sexual abuse is, unfortunately, not uncommon, and utmost care must be exercised in making these interpretations."

Can tampon use cause hymen changes in girls who have not had sexual intercourse? A review of the literature. Goodyear-Smith F and Laidlaw TM. FSI 1998; 94: 147-153. This takes up a dispute over whether posterior complete clefts (as distinct from partial clefts -- comment by Dr. Finkel in CAQ) are diagnostic of traumatic penetration. Study by Emans 1994 J Peds (above) says yes, these authors say no. Dr. Finkel comments that, "Complete clefts found between the 4:00 and 8:00 position of the hymenal edge

are considered by most clinicians to represent a transection.” (CAQ Apr 99) He says this article presents no real evidence to dispute that, but agrees that one should be objective and realize that investigation is sometimes limited as far as the actual cause of lesions. See next below.

What is an “intact” hymen? A critique of the literature. Goodyear-Smith F and Laidlaw TM. Med Sci Law 1998; 38: 289-300. Once more taking up the cudgels against Emans’s 1994 paper. Apparently saying that this is a difficult question. [Goodyear-Smith’s work is controversial. –JKR]

A comparative survey of beliefs about “normal” childhood sexual behaviors. Heiman ML, Leiblum S, Esquilin SC, and Pallito LM. Child Abuse Negl 1998; 22: 289-304. Surveyed professionals, facilitators, medical students, as to their beliefs. Professionals were more puritanical, as were females. Shows that standards are quite subjective. Dr. Ricci comments that, “Perhaps the most important comment in this study was at the end where the authors note, ‘Until we have a better sense of the sexual development of children, it is critical that professionals in the field of sexual abuse and human sexuality are aware of the influence of their role and gender in forming their beliefs about children’s sexual behavior.’”

Genital warts in children. Lori Frasier MD. The APSAC “Advisor,” summer 1998, vol 11 no 2. Congenital vs acquired. Congenital can take months or years to appear (latent virus). “There is some evidence that HPV is transmitted on small water droplets called fomites. Individuals who bathe in public showers have a much higher rate of plantar warts than those who don’t.” Viral typing does not differentiate congenital from acquired. Mostly 6, 11. Location does not differentiate. The differentiation essentially comes down to the interview. Must look for other venereal diseases, examine mouth.

Children with anogenital symptoms and signs referred for sexual abuse evaluations. Kellogg ND Parra JM and Menard S. Arch Ped Adol Med 1998 Jul; 152: 634-641

157 pts. Importantly, none of these pts presented with a history of abuse; in other words, there was no disclosure. These pts were referred because someone else felt there was possible abuse. 25 pts (15%) had findings that were “suggestive,” “probable,” or “definite” sexual abuse. 70 pts had nonspecific findings or a dx other than abuse. Here are the findings that correlated with a presenting symptom and are suspicious for abuse:

- hymeneal notch
- hymeneal attenuation to less than 1mm
- HSV 1 or 2 with notch or attenuation
- Hymeneal hemorrhages
- GC with notch or attenuation

Symptoms:

Anal or genital bleeding or bruising was the most common symptom; of 32 pts found objectively to have this finding, 8 had suggestive of abuse; the others had Group A strep, Candidiasis, LSA, accidental trauma, fusion defect, hemangioma, anal fissure, varicosities. Of those presenting with irritation, 95% had no ev of ab; otherw diaper rash, LSA, prominent vascularity of vestibule. Malformations were never abuse: anal tags, septate hymen, fusion defect, anal dil from encopresis, and nonspecific enlargement of vaginal opening. Vaginal discharge: 79% negative for abuse: of 27 pts with discharge who had no findings sugg of ab, 18 cultured who had no findings sugg of ab, 6 cultured pos (2 H. flu, 1 Shig, 1 grp B, 2 usual flora.) Of the 7 pts with a discharge who did hv findings sugg of ab, 4 cult +: (3 GC, 1 HSV + chlam), and 3 were culture-negative vaginitis. The presenting symptom that most often led to a finding of “sugg of ab” was “lesion:” 12 pts, of whom 7 (58%) had findings sugg of ab.

These results (only 15%+ for abuse) are contrasted with the results of Adams et al. (1994) supra, where all the patients had legally determined sexual abuse; of those, 96% had findings suggestive or better for abuse.

“Many, if not most, examinations of sexually abused children elicit no abnormalities. ... When tissue damage does occur, injuries may heal quickly and completely. Clearly, a child’s disclosure is key to the detection and diagnosis of sexual abuse.” (639, citing Abbey Berenson et al, The appearance of the hymen in prepubertal girls, 1992.)

“Physiologic changes associated with a reduction in the influence of maternal hormones occur around age 2 years and include increased vascular pattern, thinning, and decreased redundancy in the

hymeneal membrane. These changes cause the hymen to appear pinker, with the opening becoming more apparent." 639

The appearance of acute, healing, and healed anogenital trauma. John McCann. Child Abuse & Neglect 1998; 22(6): 605-615. With response by David Muram, and discussion. No pictures. He begins by stating the state of affairs: these injuries heal rapidly, except that hymeneal tears undergo a slower evolution. **Most child molestation does not cause tissue injury**, unlike adult rape. "Even the anatomical changes that have resulted from verified accidental injuries of a child's anal-genital region are few in number. Due to this lack of knowledge, medical examiners have been handicapped in their efforts to interpret the physical findings encountered during an evaluation of a child suspected of having been sexually abused." (606).

Normal anogenital anatomy. Abbey B. Berenson. Child Abuse & Neglect 1998; 22(6): 589-596, with response by Larry Ricci and discussion.

Evaluation for possible physical or sexual abuse. Kini N and Lazoritz S. Peds Clin N Amer 1998 Feb; 45(1): 205-219. The final determination is up to the legal system. The clinician only determines reasonable suspicion.

Female genital mutilation. AAP Committee on Bioethics Statement, approved by the Council on Child and Adolescent Health. Pediatrics 1998; 102: 153-156. Describe four major types: Type I = excision of the skin surrounding the clitoris with or without excision of part or all of the clitoris; Type II = removal of the entire clitoris and part or all of the labia minora; Type III=infibulation: the clitoris and labia minora are excised and the labia majora are sutured; Type IV=different practices of variable severity including pricking, piercing or incision of the clitoris and/or labia, stretching of the clitoris or labia, cauterization of the clitoris, and scraping or introduction of caustic substances into the vagina.

Pediatric male rectal and genital trauma: accidental and nonaccidental injuries. Kadish HA, Schunk JE and Britton H. (Utah) Pediatr Emerg Care 1998 Apr; 14(2): 95-98. 44 accident patients aged six months to 17 years are compared with 44 assault patients. The accidental patients usually had scrotal injury; the assault patients only had rectal injury. The most common accidental mechanism was a fall onto an object. The most common accidental injuries were laceration of the scrotum (36%) and laceration of the penis (25%). None of the accidental patients had any isolated anorectal laceration. The sexual assault group was aged 7 months to 18 years. they all had rectal lesions. Two of them had penile lesions.

As far as evidence of anal trauma, the authors considered the following to be evidence of sexual abuse: scars, tags, localized venous engorgement, dilation, asymmetrically thickened folds, tears, abrasions, warts.

Commenting on this article in Child Abuse Quarterly Oct. 1998, John McCann emphasizes the difference in location between accidental and abusive injury: scrotal versus anal. And indeed, that dichotomy is dramatically present in these authors' data. --JKR

Vaginal injury from a water slide in a premenarcheal patient. Kunkel NC. Pediatr Emerg Care 1998; 14: 210-211. 10 year old girl hydroplaned. Bled immediately. Found to have a posterior introital superficial laceration AND a deep 4 x 3 cm laceration of the upper vagina which required operative repair. The authors review 16 other cases of vaginal damage from high-pressure water injury in the literature.

The assessment of child sexual abuse allegations: using research to guide clinical decision making. Dammeyer MD. Behavioral Sciences and the Law 1998; 16(1): 21-34. Which sources of information are reliable? Medical examination and the child's report.

The parental alienation syndrome: what is it and what data support it? Faller KC. Child Maltreatment 1998 May; 3(2): 100-115. This article attacks the work of Richard A. Gardner, who has contended that many juvenile allegations of sexual molestation by a parent are the results of prompting by a hostile parent or allied system interventions. Faller reviews briefly the systematic criteria that are used by experts (psychologists) in evaluating a child's allegation of sexual abuse by a parent. 109. Criteria used include (a)

the fact that the child describes sexual abuse, (b) detail about the abuse, (c) advanced sexual knowledge, (d) sexualized behavior as reported by others. She discusses the probable validity of each of these criteria, (some needing further research).

The medical evaluation of the sexually abused child: lessons from a decade of research. Atabaki S and Paradise JE. Pediatrics 1999; 104: 178-186. Review article. Says more research is needed on both technique and interpretation. Cautions to be alert to the anatomic variations of both genital and anal findings in non-abused chil.

Appearance of the hymen in adolescents is not well documented. (letter). Curtis E, San Lázaro C. BMJ 1999 Feb; 318(7183): 605

Influence of the history on physicians' interpretations of girls' genital findings. Paradise JE, Winter MR, Finkel MA, Berenson AB, and Beiser A. Pediatrics 1999 May; 103(5): 980-986. Mailed photographs to AAP members, then sent back the same photographs again with a different history. The diagnoses changed when the history changed -- 5/6 for the inexperienced physicians, and 2/6 for the experienced. Questionnaires with genital photographs were got back from 604 pediatricians. Then they sent the same photographs again with a different history. This changed the diagnosis of the photographs 5% of the time for experienced physicians and 27% of the time for inexperienced. Conclusion: diagnostic expectation appears likely to influence physicians' interpretations of girls' genital findings. Physicians should be alert to the possibility of diagnostic expectation bias and its potentially serious social and legal consequences.

Medical assessment and legal outcome in child sexual abuse. Palusci V, Cox EO, Cyrus TA et al. Arch Pediatr Adol Med 1999 Apr; 153(4): 388-392. He found that when it comes to criminal adjudication, as opposed to medical treatment and CPS intervention, positive physical findings were associated with a judicial finding of guilt. Behavior symptoms and disclosure were less associated. Therefore, "Medical assessment plays an important role in the overall community response to child sexual abuse."

Evolution of a classification scale: medical evaluation of suspected child sexual abuse. Joyce A. Adams, MD, Associate Professor of Pediatrics, UCSD. Unpublished manuscript, 1999. This is a revision of Dr. Adams' earlier scale. It is now a two-part scale. The first part is the anogenital findings. The second part is the overall likelihood of abuse. See also Abbey Berenson's 2000 article, below, re partial and complete transections or deep notches.

The anogenital findings scale:

- Class I normal
- Class II nonspecific
 - Includes introital erythema, anal fissure, flattening, congestion
- Class III concerning
 - Includes immediate anal dil, posterior hymenal cleft, fresh labial abrasions or lacerations, scar or laceration of the posterior fourchette, perianal scar.
- Class IV clear evidence of abuse
 - Acute laceration of the hymen.
 - Bruising of the hymen.
 - Deep perianal lacerations entering the sphincter ani
 - Absence of hymenal tissue

The overall scale

- Class I No indic of abuse
 - Injuries consistent with accident
- Class II Possible abuse
 - Class I or II anatomic findings plus signif behav changes
 - Condyloma or HSV I
 - Ambiguous disclosure
 - Class III findings
- Class III Probable abuse
 - Clear disclosure

Chlamydia culture over 2 years of age
 HSV II cult
 Trich
 Class IV Definite abuse
 Class IV physical findings with no history of accident
 Sperm
 Pregnancy
 GC, Syph if perinatal ruled out
 Documented/witnessed abuse
 HIV if perinatal or transf ruled out

AAP Guidelines for the evaluation of sexual abuse of children: subject review. Pediatrics 1999 Jan; 103(1): 186-191 (have)

Definition: "Sexual abuse occurs when a child is engaged in sexual activities that the child cannot comprehend, for which the child is developmentally unprepared and cannot give consent, and/or that violate the law or social taboos of society."

In 1996 you had, as far as substantiated cases,

Physical abuse	23%
Sexual abuse	9%
Neglect	60%
Emotional	4%
Other	5%

When the exam is within 72 hours, or there is bleeding or acute injury, should follow state protocol (have) for collecting trace evidence.

Findings that are concerning but not diagnostic include:

1. Abrasions or bruises of the inner thighs and genitalia
2. Scarring or tears of the labia minora
3. Enlargement of the hymenal opening

Findings that are more concerning include:

1. Scarring, tears, or distortion of the hymen
2. A decreased amount of or absent hymenal tissue
3. Scarring of the fossa navicularis
4. Injury to or scarring of the posterior fourchette
5. Anal lacerations

Diagnostic even without a history:

1. Semen, sperm, acid phosphatase, +GC cult, +syph serol, +HIV if not congenital

In the presence of a concerning history or concerning findings, physician may elect to observe the child for changes in behavior.

The differentiation of accidental injury "may be difficult and may require a careful history and multidisciplinary approach."

Also discusses divorce context.

“In both criminal and civil proceedings, physicians must testify to their findings ‘to a reasonable degree of medical certainty.’ ” Citing McCann, Voris, 1993.

Woods lamp utility in the identification of semen. Santucci KA et al. Pediatrics 1999; 104: 1342-1344. Woods lamp DOES NOT work for identifying semen. These authors illuminated 19 fresh semen samples with it and none fluoresced. 41 doctors using it were unable to differentiate semen from common household products.

Lesson of the week: child sexual abuse enquiries and unrecognized vulval lichen sclerosus et atrophicus. Wood PL and Bevan T. BMJ 1999; 319: 899-900

Child sexual abuse enquiries and unrecognized vulval lichen sclerosus et atrophicus. Wood PL and Bevan T. BMJ 1999; 31: 899-900. Pale thin skin around the introitus with submucosal hemorrhages. Symptoms may include itching, rubbing, scratching, reported masturbation, soreness, blistering, bleeding, bruising, discharge. Jan Bays comments (Child Abuse Quarterly, July 2000) that LSA can also be perianal in either boys or girls, and also penile.

Accidental hymenal injury mimicking sexual trauma. Boos SC. Pediatrics 1999; 103: 1287-1289. This girl slipped & fell on a toy, had abr of R labium minus, fossa nav, and a 6:00 complete transection. He reviews the past literature on accidental genital injuries in childhood and finds that only 6/161 (3.7%) of accidental involved hymenal trauma.

Adolescent girls investigated for sexual abuse: history, physical findings and legal outcome. FSI 1999; 104: 1-15. Cited by Bruni, 2003, for the proposition that anal physical findings and their interpretation “remain an area of controversy with variable significance attributed to them.”

Do physicians have adequate knowledge of child sexual abuse? The results of two surveys of practising physicians, 1986 and 1996. Lentsch KA and Johnson CF. Child Maltr 2000; 5: 72-78. Only 46% of physicians check the size of the vaginal orifice, although over 70% do a genital exam most of the time. 62% were able to identify the hymen on a photograph. 13% of physicians thought a horizontal hymenal diameter of 12 mm was normal.

Do physicians have adequate knowledge of child sexual abuse? The results of two surveys of practicing physicians, 1986 and 1996. Lentsch KA and Johnson CF. Child Maltreatment 2000 Feb; 5(1): 72-78. 62% of physicians could identify the hymen in a photograph. Shown a photograph of a normal prepubertal hymen, 36% of physicians identified it as normal, 62% would have reported it as possible sexual abuse. Shown a photograph of an anal fissure with a history denying constipation, 99% correctly identified it as an anal fissure and 56% said they would report it as possible abuse.

Signs of genital trauma in adolescent rape victims examined acutely. Adams JA, Girardin B, Faugno D. (UCSD) J Pediatr Adol Gyn 2000 May; 13(2): 88. Joyce Adams group did a retrospective chart review of 214 females 14-19 years of age evaluated acutely by the SART team using magnification and dye for a complaint of sexual assault. 23 reported anal penetration.

posterior fourchette tear	36%
posterior fourchette or fossa	40%
hymenal tears in virgins	19%
hymenal tears in nonvirgins	3%
erythema	32%
swelling of hymen	19%
Toluidine blue +	66%
no findings	21%
anal tears	14/23 = 61%

A case-control study of anatomic changes resulting from sexual abuse. Berenson AB, Chacko MR, Wiemann CM, Mishaw CO, Friedrich WN, Grady JJ. Am J Ob Gyn 2000 Apr; 182(4): 820-834. (have) Compares 200 non-abused girls aged 3-8 with 192 abused girls. No nonabused had any deep notches or any transections. But then very few of the abused had them: only 2 deep notches and 1 transection. Joyce Adams comments that this study supports the inclusion of complete transection in the "definite evidence" category of her classification scale, and notches "near to the base of the hymen" in the "concerning" category. (SIGCA-MD-L, April 25, 2000). David L. Kerns MD of the Santa Clara Valley Medical Center in San Jose, however, criticizes Adams's use of this Berenson work (SIGCA Oct 11 2000), saying that Adams selectively ignores a Berenson statement that deep posterior notches (over 50% but not complete transections) "may be considered as a definitive sign of sexual abuse or other trauma," in other words, can be more than "concerning" as in Adams's classification scale. He propounds several other criticisms of Adams' classification scale, calling it a "dumbing down" of diagnosis. He emphasises the importance of a good history. He fears that the cookbook approach will be exploited by defense attorneys to minimize the diagnostic value of all but the most extreme lesions. Martin O. Finkel shares many of these concerns, and says that even after 20 years of hymenal examinations it is difficult to appreciate the variability of findings of healed injuries. He points out that over 90% of victims have no lesion, and the protection of them depends on a nuanced history. Dr. McCann comments on healing. Sharp acute V-shaped tears rapidly round off, but do leave a membrane residual at the base. Dating them is unknown: he is studying that. Adams replies that this scale is a work in progress, and that the actual number of positives is very small, 2 complete transections seen out of hundreds of patients, so she advocates being conservative in diagnostic interpretation.

Forensic interpretation of blood in the anus. (letter). Boyd G. Stephens. Am J Forens Med Path 2000 Jan; 21(1): 93-94. Reports two cases of erroneous clinical diagnosis of sodomy. Believes the blood in the anus got there by drainage from femoral sticks. Cautions that "Because of the legal ramifications that can follow an accusation of unsubstantiated sexual penetration, the forensic interpretation of blood in the vagina or anus requires careful consideration."

Case 1 a brain dead child abuse-head injury case, hospital found blood in the anus and anal dil. Man was charged with sodomy as a result. At autopsy the anus was negative.

Case 2 similar infant, clinicians reported a rectal tear. Autopsy was negative.

ASCP Check Sample No. FP 00-8. Kim Collins, MD. A case of alleged sexual assault in a 22 year old female. 2000 Colposcopy has proven useful... 20%-45% of assault victims have no injuries. Recent reports have described mild or severe injuries by other mechanisms, e.g. consensual intercourse, tampons, jetskis, water skiing, water slides. "Some investigators even report that vaginal lacerations after coitus are relatively common in young women." Time to loss of motility in adult vagina is 2 – 3 hours; in pediatric vagina probably shorter bc of lack of mucus. FISH is fluorescence in-situ hybridization, which detects Y chromosomal material in air-dried vaginal extracts. Nuclear fast red picroindigocarmine (Christmas tree stain) stains heads red, tails yellow-green. Head meas 4-5 micr by 2-3 micr tail 40-55. The first sign of sperm degeneration (after loss of motility) is tail loss. Tail is lost after 16 hours in the vagina, 6 hours in the rectum. Spermatozoa identifiable in living patients: In vagina after 72h 50% will no longer have, after 10d 100% will no longer have. In rectum 24h will no longer have, although reports of 65h. In oral cavity 6h. PAP detectable for 24h, not detectable after 48h. P30 is more stable and more sensitive.

A case-control study of anatomic changes resulting from sexual abuse. Berenson AB, Chacko MR, Weimann CM, Mishaw CO, Friedrich WN, Grady JJ. Am J Ob Gyn 2000 Apr; 182(4): 820-831. Examined 192 children with a history of penetration and 200 children who denied abuse. Statistical tests done. Four abused children had a transection or deep notch, no non-abused had these. There was a statistically significant difference in the frequency of a vaginal discharge $P=.01$. Otherwise, there was no statistical difference in the frequency of finding labial agglutination, vaginal ridge, superficial hymenal notch, hymenal bumps, increased vascularity, friability of tissues. Therefore: **the genital examination rarely discriminates an abused child from a non-abused child.** "Legal experts should focus on the child's history as the primary evidence of abuse."

Medical examination of sexually abused children: medico-legal value. Lauritsen AK, Meldgaard K, Charles AV. (Aarhus) J Forens Sci 2000 Jan; 45(1): 115-117. 34 cases with a positive history of abuse. Normal findings in 23. Nonspecific findings such as erythema in 13. Ruptured hymen in 1. Conclusion: The medical examination seldom provides any proof. "The most important is the story told by the child." Cited by Bruni, 2003, for the proposition that "Anal physical findings, and their interpretation, continue to be discussed and remain an area of controversy with variable significance attributed to them."

Forensic evidence findings in prepubertal victims of sexual assault. Christian CW, LavelleJM, DeJong AR, Loiselle kj Brenner L Joffe M. Pediatrics 2000 Jul; 106(1 Pt 1): 100-104. Timing of the examination was critical for recovery of any evidence from the child's body. Overall 24% had ev, but 64% of it was found on the clothing, not on the body. After 9 hours, no body swabs were ever positive for sperm. The clothing is very important, particularly if there has been time-lapse. Apparently children's bodies retain physical evidence more briefly than do adults'. -JKR

Child sexual behavior inventory: normative, psychiatric, and sexual abuse comparisons. Friedrich WN, Fisher JL, Dittner CA et al., Child Maltr 2001; 6: 37-49. Reviewed in Child Abuse Quarterly for July 2001. This is the third version of the child sexual behavior inventory. 1114 children, normal and mentally ill and sexually abused. Parent questionnaires. Mostly caucasian. Sexually abused children scored significantly higher.

Challenging case: behavioral changes -- Erica: a question of sexual abuse. Stein M, Adams J, Wells R. Pediatrics 2001; 107 Suppl: 845-849. Reviewed by Larry Ricci in Child Abuse Quarterly July 2001. Case of a 20 month old toddler who started pointing to her vagina and saying "play ... Daddy." Dr Ricci notes that his own recent research has shown that sexual behavior in children had no correlation with sexual abuse in 247 children. He comments that the use of anatomically correct dolls is rarely indicated and only in the hands of an expert, contrary to what these authors say. Similarly the taking of forensic samples is rarely indicated, contrary to what these authors say. See next below.

The diagnostic utility of sexual behavior problems in diagnosing sexual abuse in a forensic child abuse evaluation clinic. Drach KM, Wientzen J, Ricci LR. Child Abuse & Neglect 2001; 25: 489-503. Sexual behavior did not correlate w abuse. See "Erica" article, 2001, above.

Evolution of a classification scale: medical evaluation of suspected child sexual abuse. Joyce Adams. Child Maltr 2001; 6: 31-36. Earlier versions were 1992, 1996, 1997. No longer makes use of hymenal measurements. Does classify a 50% posterior notch as significant for abuse. But does it classify a deep cleft of the posterior area as "clear evidence of abuse" as discussed by other authorities? (see Berenson, Case-control study, below.) Cited by Bruni, 2003, for the proposition that "Anal physical findings, and their interpretation, continue to be discussed and remain an area of controversy with variable significance attributed to them."

Evaluating Children for possible sexual abuse. Adams J. Am Fam Phys 2001 Mar; 1; 63(5): 843-844, 846.

Detection of child sexual abuse. Topley J, Thomas A, Hobbs C, Wynne J. Am J Ob Gyn 2001 Apr; 184(5): 1043-1045.

Evaluating the child for sexual abuse. Lahoti SL McClain N et al. Am Fam Phys 2001 Mar; 63(5): 883-892. Practical advice for the general gynecologist.

AAP Policy Statement: Care of the adolescent sexual assault victim. AAP Committee on Adolescence. Pediatrics 2001; 107: 1476-1479.

The medical evaluation in cases of child sexual abuse. Muram D. J Pediatr Adol Gyn 2001 May; 14(2): 55-64.

The relationship between the transverse hymenal orifice diameter by the separation technique and other possible markers of sexual abuse. Ingram DM, Everett VD, Ingram DL. Child Abuse & Neglect 2001; 21: 317-322. They compared almost 2,000 girls in three groups: a penetration group, a sexual contact and maybe penetrated group, and a control or probably no contact group. They found that there was a statistically significant increase in transverse hymenal opening diameter in the groups with findings, but that it was not helpful as an independent discriminant. Whenever there was an increased diameter that might be used as diagnostic evidence of penetration, there were always other, more definite findings of penetration such as tears and clefts. "The findings of hymenal trauma were obvious and that the [transverse opening diameter] is redundant and provided no new or useful clinical information."

But note that these researchers did not say that the diameter cannot be reliably measured. They only said that it need not be. At the same time, their logic clearly implies that the diameter by itself is not a useful diagnostic finding, because there was overlap between the abused groups and the nonabused group. Therefore the diameter is not a discriminant between abused and nonabused.

Association of a posterior fourchette finding with other ev of sexual abuse in prepubertal females. Legano, McHugh, Vince Palusci and others. J Pediatr Adol Gynecol 2001 Aug; 14(3): 142-143. Thinning of the posterior fourchette was statistically associated with sexual abuse.

Adolescent sexual assault: documentation of acute injuries using photo-colposcopy. Adams JA, Girardin B, Fuagno D. (UCSD) J Pediatr Adol Gyn 2001 Nov; 14(4): 174-180. (See 2000 paper by the same authors on the same material from Poway). Had 214 adolescent rape victims examined within 72 hours. "Hymenal tears were uncommon, even in self-described virginal girls. Timely examination of adolescent victims is important to document injuries; however, many victims will still not have signs of bruising, abrasions, or tears."

Why is sexual abuse declining? A survey of state child protection administrators. Jones LM, Finkelhor D, Kopiec K. Child Abuse & Neglect 2001; 25(9): 1139-1158. A telephone survey of 43 state child protection administrators. They offered the following possible reasons: (1) increased evidentiary requirements to substantiate cases; (2) increased caseworker caution due to new legal rights for caregivers; (3) increasing limitations on the types of cases that agencies accept for investigation; (4) effective prevention due to increased public awareness, increased prosecution, and prevention programs. But only half of the officials cited any of these factors, leaving the researchers to conclude that a portion of the decline may result from a real decline in occurrence. (Dr Finkelhor gave the keynote address at the San Diego conference on this same topic several years ago. --JKR)

Genital injuries in prepubertal girls from inline skating accidents. Herrmann B, Crawford J. Pediatrics 2002 Aug; 110(2 Pt 1): e16. Case report of two cases of a "rapid-splits" mechanism in eight year old girls. From Dr McCann's review in the *Quarterly*: These were midline perineal lacerations, one involving the posterior fourchette and the other into the anal verge. Neither one had any hymeneal injury. Dr McCann comments that "Once again, the importance of a clear history was considered essential in arriving at [the conclusion that the findings were consistent with an accidental mechanism.]" Dr McCann seems to be implying that if the history had been vague or unclear, it would not have been possible for an examining physician to establish that the injuries were consistent with an accidental mechanism. --JKR

Genital examinations for alleged sexual abuse of prepubertal girls: findings by pediatric emergency medicine physicians compared with child abuse-trained physicians. Makaroff KL, Brauley JL, Brandner AM, Myers PA, Shapiro RA. (Cincinnati). Child Abuse & Neglect 2002 Dec; 26(12): 1235-1242. They reexamined 46 girls in whom the ER physician had made a diagnosis of sexual abuse. Found that only eight of these children showed clear anatomic evidence of abuse (17%). Found that 70% had normal genital findings, 9% had nonspecific findings, and 4% (two children) had findings that are more commonly seen in abused than in nonabuse but are still non-diagnostic. Conclusion: poor agreement. More training needed. Followup exams needed.

National estimates of the timing of sexual maturation and racial differences among US children. Sun SS, Schubert CM, Chumlea WC, Roche AF, Kulin HE, Lee PA, Himes JH, Ryan AS. Pediatrics 2002 Nov;

110(5): 911-919. This article is used in the evaluation of child pornography. See also Carole Jenny's 2007 lecture, below.

Children referred for possible sexual abuse: medical findings in 2384 children. Heger A, Ticson L, Velasquez O, Bernier R. Child Abuse & Neglect 2002; 26: 645-659. Found high rates of normal exams:

Referrals seen	normal	abnormal
Overall	96.3 %	3.7 %
Children who had disclosed (N = 1652)	95.6 %	4.4 %
severe abuse (penetration) (N = 1134)	94.5 %	5.5 %
girls (N = 957)	93.6 %	6.4 %
boys (N = 177)	98.9 %	1.1 %
nonsevere abuse (N = 518)	98.3 %	1.7 %
Children who showed concerning behaviors	99.8 %	0.2 %
Children referred for eval of a medical condition	92.0 %	8.0 %

Most of the children were referred within seven days of the latest abusive event, unlike previous studies where many subjects were seen weeks or months after the event.

Also gives a comprehensive chart of comparable findings* reported in thirteen past articles going from 1979 to 2000 (p. 647). As far as vaginal findings, these thirteen articles reported the following percentages:

Normal/nonsp: 65%, 85%, 39%, 16%, 70%, 42%, 62%, 77%, 85%, --%, --%, 65%, 97% = avg 64%
 Susp/suggstv: 55%, --%, 16%, 84%, --%, --%, 10%, 9%, 12%, --%, --%, --%, --% = avg 31%
 Definitive: 35%, 15%, 48%, --%, 30%, 58%, 28%, 14%, 3%, --%, --%, --%, --% = avg 29%

* The authors point out that the older findings are not really comparable, bc of different knowledge base and examination techniques and lack of peer review and photodocumentation, which began in 1989. (p. 654) The authors also state that "The early studies included a wider ramnge of anatomical variations or findings as diagnostic or suggestive of abuse..." (see below)

88 of the present authors' total cases were found to have physical findings diagnostic of sexual abuse (3.7 %). Of these 88, 15 (17 %) were chil who had been referred for eval of a medical condition, not for suspicion of abuse. The diagnostic findings in these 15 consisted of either venereal disease or genital injuries. There were 182 chil in the group referred for eval of a medical condition., and the 15 found to have findings diagnostic of abuse comprised 8% of that group. In other words, the chil referred for eval of a "medical condition," and not for suspicion of abuse, had by far the highest rate of anatomic diagnosis of sexual abuse, almost twice as high as the children who had disclosed. The authors (p. 653) comment that this finding is in line with that of a previous study by Bowen & Aldous (1999), who found a much higher rate of abuse in general referrals than in specific referrals for abuse.

Other items of interest:

- a. Of the 957 girls who disclosed penetration, 6% had abnormal examinations.

(By JKR: Why is this? Did they only think they were penetrated? Were they imagining it? Did they misunderstand the interviewers' questions? Was penetration atraumatic 94% of the time? The authors comment on this. They say that it is probably the first three. "Perhaps the best explanation is the child's unsophisticated understanding of hwat happened and the misinterpretation by the child of simulated intercourse as either penile-vaginal or penile-anal penetration." (p. 653)

- b. Of the 177 boys who disclosed penetration, 1% had abnormal examinations.

(By JKR: McCann's studies have already shown that most anal penetration causes no injury, and that even when it does, the injuries heal quickly.)

c. The results of this work cannot be compared with published work that predates the advent of photodocumentation and evaluation scales and standardization of terminology and procedures which have taken place since 1989.

d. “[M]any of the anatomical findings that were reported as abnormal in the early studies are now considered by clinicians and researchers to be nonspecific genital variations. These nonspecific anatomical variations include enlarged hymenal diameter, narrowing of the hymenal edge, partial notching or clefts of the posterior hymenal rim, erythema or swelling, bumps or irregularities, and changes in tone or rugal patterns of the anus.” (p. 648)

e. Negative exams play a valuable role in providing safety and reassurance for the child and the family. But “unfortunately,” positive *and* negative exams play a dominant role in the legal and foster-care systems, which do not focus on the child but on other priorities and can harm the child and family. (p. 653, 655) Medical evidence is the deciding factor in many legal cases and [is] the most significant factor in the progress of a case through the legal system. But legal-social is not the main purpose, as the authors want to see it, of the medical examination. It is to them an ancillary purpose and, by clear implication, one that interferes with the main, child-centered, purposes of reassurance and safety. Even though they are paid by the government in a clinic that is funded and maintained by the government largely for its legal-social role, the authors prefer to see themselves as servants of the patient, not the government. They leave unstated, but one can clearly appreciate between the lines, the fact that they are concerned by the counterproductive effects on children and families that they have observed in many cases, perhaps including cases with positive medical findings as well as cases with negative medical findings. –JKR

My thanks to Stephanie Erlich, M.D. for getting me this paper.

California Medical Protocol for Examination of Sexual Assault and Child Sexual Abuse Victims. OJCP 2002. <http://www.ojcp.ca.gov/> Summarized in Peterson MS and Durfee M, eds. Child Abuse and Neglect: Guidelines for Identification, Assessment, and Case Management. Volcano, CA: Volcano Press, 2003. (have).

Ten-year research update review: child sexual abuse. Putnam FW. (Cincinnati Children’s). J Am Acad Child Adol Psychiatr 2003 Mar; 42(3): 269-278. This is a literature review. Found that CSA was 10% of all substantiated child abuse. About 88,000 cases per year in all the English-language literature. Adjusted prevalence of 17% and 8% for women and men. The best-documented outcomes are depression in adults and sexualized behavior in children. Cognitive-behavioral therapy of the child and nonoffending parent is the best treatment. Prevention is child education and home visits.

Anogenital injuries in child pedestrians run over by low-speed motor vehicles: four cases with findings that mimic child sexual abuse. Boos SC, Rosas AJ, Boyle C, McCann J. Pediatrics 2003 Jul; 112 (1 Pt 1): e77-e84. From the abstract: “Differentiating between child sexual abuse and nonintentional causes of anogenital injury can be challenging, and a misdiagnosis can have a profound impact on the child and family. This case series documents an important nonintentional mechanism of anogenital injury that mimics the physical findings of CSA.” Two children had perianal lacerations, 2 had hymenal lacerations. One child followed for 4 weeks developed findings identical to those seen in healed sexual abuse. Authors advise to document anogenital exam in such traffic victims to avoid later misinterpretations.

Roundtable discussion: The medical evaluation of sexually abused children. Muram D. J Pediatr Adol Gyn 2003 Feb; 16(1): 5-14. (have) This is a roundtable moderated by Dr Muram between Astrid Heger, Martin A Finkel, Karen J Simmons, and JM Whitworth. Topics:

1. History taking
2. Genital findings
3. Colposcopy and photodocumentation
4. Interpreting the findings to laymen
5. Anal findings

- 6. STD's
- 7. Family services
- 8. Preparing for court
- 9. Telemedicine

1. History taking

H: history is essential. bond with the child. interview every ch over 5. direct, nonleading....

2. Genital findings

H: More than 94% of pts have normal examinations. “[W]e have determined that the only finding that is diagnostic of a penetrating injury to the hymen is a transection of the hymen that extends to the base of the hymen. Although partial tears of the hymen do occur, they heal completely and may have only a slight residual notch that is indistinguishable from nonspecific findings. Significant lacerations to the posterior fourchette may heal with midline scarring and are equally common in cases of accidental trauma and sexual assault....” p. 6.

F: Injuries are rare bc the perp does not intend to harm the child. When chil [do] incur genital or anal injuries, these are often minor. These heal by regeneration within 96 hours and leave no residual or diagnostic findings.

3. Photodocumentation

H: Photodocumentation has revolutionized the field of CSA. Colposc is preferred. But “Perhaps the most tragic downside to photodocumentation is the overemphasis on the medical findings and a focus on minor changes in genital anatomy as significant indicators of abuse.” p. 7.

M: Photodocumentation is now the standard of care. See the APSAC guidelines (1995).

4. Interpreting the findings to laymen

W: [We] avoid terms like “suspicious” or “may be seen in.” We use strictly medical terms: diagnostic of or consistent with. Remember that a normal examination is consistent with CSA.

Diagnostic of CSA (findings that establish sexual contact even in the absence of a Hx)
(Note that CSA does not necessarily involve penetration. As to penetration, see below.)

- Pregnancy
- Syphilis and gonorrhea
- Photographically documented sexual acts
- Semen or sperm

Diagnostic of penetration: (note that penetration is not the same thing as sexual abuse. Penetration can be due to accident.

- A hymeneal scar extending to or beyond the vaginal wall
- A missing section of the hymen in the posterior rim
- An anal scar extending through the anal sphincter

Consistent with CSA

- A whole succession of findings, including
 - Microlacerations
 - Abrasions
 - Healed fissures
 - STD's which can be nonsexually transmitted

Normal findings do not necessarily mean that nothing happened. We provide attorneys with a printed list of reasons why there may be no findings.

[Comment by JKR: That's fine, but what's going to happen in court is that the defense attorney is going to take you through that list item by item, and force you to admit that you have been provided no evidence that any of those reasons actually occurred in the present case.]

M: For interpreting the medical terminology to laymen I proposed an interpretive classification:

Normal examination
 Nonspecific abnormalities
 Abnormal findings suggestive of abuse
 Abnormal findings indicative of abuse

[Comment by JKR: M goes on to say that his classification is substantially the same as W's, but in my opinion there is a sharp divergence between the two. Beginning with the fact that his is four layers instead of two. Plus the fact that W explicitly rejects question-begging or vague terms like "suggestive of" or "suspicious for." W basically is saying that these are advocacy terms that are designed to force the accused to disprove abuse.]

M: How can a child have a normal exam after being penetrated in a sexual assault?

W: Reasons:

1. The genital anatomy is designed to be penetrated. Hymen has a hole, is stretchable.
2. There can be superficial (labial-only) "penetration." The hymen is recessed within the vagina and may not be reached. Thus the child's history of penetration is accurate; that is what she perceived. She has no frame of reference to distinguish one level of penetration from another.
3. Lubricants/ lubrication. Can include sexual arousal on the part of the victim.
4. Perpetrator usually gentle. These are by seduction, not force.
5. Rapid healing. Mucus membranes (e.g. the lining of the oral cavity) heal rapidly and completely. [other than hymenal transections, I assume he is saying. -JKR]
6. Pubertal transition can mask old injury. The tissues become estrogenized and thicker. [This amounts to a statement that in the chronic case the genital examination has low sensitivity -- that old injuries are easily missed. That is probably true, but note that this statement raises medicolegal issues. The cross examination would go something like this:

Q. Doctor, are you saying that you might have missed an old injury to the victim?

A. No, I'm simply saying that old injuries in this age group can be difficult to detect.

Q. Did you detect an old injury?

A. No.

Q. Are you confident that there was no old injury?

A. []

Q. Doctor, are you confident of your findings in this case?

-- JKR]

S: Says that research (in part Muram's research: Muram et al., 1999) shows that there is an element of subjectivity in the interpretation of findings. The examiner's interpretation is biased by the history; if he has received a history of abuse, he tends to interpret equivocal findings as evidence of abuse, even though someone else coming in blind would not. This is an "unfortunate" aspect of the dependence of sexual abuse medicine on the child's history.

5. Anal findings

H: I have only seen a handful of children with findings diagnostic of anal trauma. Anal trauma heals quickly and completely.

F: "There has probably been more controversy around the interpretation of anal findings than any other. I believe the root of this difficulty stems from the early reliance on literature that was a reflection of a few clinicians' experiences. The British literature from the early 70's and 80's reflected a myriad of physical findings that were considered diagnostic of anal penetration. (citing Paul, 1986, Hobbs & Wynne, 1986, 1989) It wasn't until the late 80's and early 90's when clinicians in the United States who had amassed significant clinical experience over time in child sexual abuse began to question the previously published observations. (citing McCann et al., 1989; Muram, 1989) Early publications alluded to findings such as funneling, hypertrophied rugae, anal wink, and buttocks separation test. The reality is that the anus is designed for contraction and dilatation. However, children who have encopresis and pass mammoth bowel movements do not generally have changes in the sphincter other than some decrease in tone. The only published paper by a clinician who looked at the anus of adults engaging in consensual anal intercourse could not tell by observation the difference between an anus that was penetrated repeatedly and one that was not." (citing Feigen, 1974) Goes on with a discussion of why definite anal trauma is rarely found. Same reasons as above under normal genital exam. "A very few" child sodomy victims will have superficial lacerations. (p. 9) He has never seen a definitely traumatic anal tag. "My sense is that a prominent median raphe and congenital abnormalities have been misinterpreted as posttraumatic findings..."

6. STD's

The discussants say that the diagnostic yield of cultures has been low. Their use should be adjusted to the history. But positive cultures are still the gold standard of diagnosis.

7. Family services

8. Preparing for court

H: Five things:

1. Know your records
2. Tell the truth
3. Don't exaggerate or get defensive
4. Don't volunteer anything
5. Never go to court without having met with the DA

F: Gives a lengthy discussion, of which the signal point is: Go over the records line by line with the DA.

W. Three steps to preparation:

1. Reading

- a. Re-read the literature
- b. Read your previous testimony
- c. Scan your own publications

2. Demand a meeting with counsel prior to testifying

3. Anticipate cross-examination

- a. Know all of the literature which in any way suggests a conclusion different from yours.

S: Refers to Heger's book (Heger, Emans, Muram, *Evaluation of the Sexually Abused Child*, 2d ed., 2000).

9. Telemedicine

Disorders of sexual preference and medicolegal issues thereof. Sharma BR. AM J Forens Med P 2003 Sep; 24(3): 277-282. Gives technical names and definitions for a large number of deviations including pedophilia.

Acute trananal evisceration of the small bowel: report of a case and review of the literature. Morris AM, Setty SP, Standage BA, Hansene PD. Dis Col Rect 2003 Sep; 46(9): 1280-1283. Case report of a surgical patient with chronic constipation and postoperative adhesions leading to transanal herniation of the intestine. There are 52 previously reported cases in the world literature. This is only the third case to implicate chronic constipation as a contributing factor in this rare lesion.

Healing patterns in anogenital injuries: a longitudinal study of injuries associated with sexual abuse, accidental injuries or genital surgery in the preadolescent child. Heptinstall-Heger A, Mcconnell G, Ticson L. Pediatrics 2003; 112: 829-837. Acc to Dr Reece's review in the Jan 04 *Quarterly*, a 10-year prospective study of 94 children (48 sexually abused, 19 anal trauma, 27 girls with trauma or surgery). 23/48 cases had anatomic findings diagnostic of sexual assault. All healed, a few with scarring or adhesions. Conclusion: "anogenital trauma heals quickly, often without residua."

Anal findings in sexual abuse of children (a descriptive study).* Bruni M. JFS 2003 Nov; 48(6): 1343-1346. Studied 50 children (age range 2-14 years, average 8) where the abuser had confessed to repeated abuse and been convicted in court. (So these were repeated abuse cases, not one-time attacks.) The exams were carried out 4 weeks to 14 months after the incident. In his more recent examinations, the author preferred the left lateral position; otherwise, the supine and knee-chest positions were employed. In all patients, a history of constipation, previous surgery, chemotherapy, or Crohn's disease was ruled out. Penetration was digital in 44 cases and penile in 6 cases. 32% had anal scars and tags. Other findings in 33% included reflex dilatation and venous congestion. From the abstract: "The results confirm earlier reports that physical signs, including scars, tags, reflex anal dilatation, funnelled anus, and extensive venous congestion, are often present in abused children, singly or in combination, and that anal examination should be undertaken even months after known or suspected sexual assault." But for legal purposes these signs "do not *per se* provide proof of abuse." Says that anal findings in abuse "remain an area of controversy." (Citing Lamb, 1994; Edgardh et al., 1999; Lauritsen et al., 2000; Adams, 2001.) Takes note that scars, tags, and fissures can come from nontraumatic causes. Also takes note of the fact that midline anal tags can be congenital. (Citing Berenson et al., Appearance of the hymen in prepubertal girls, 1992; McCann, Voris, 1989; Heger et al., Atlas, 2000.) Scars and tags following abuse can usually be differentiated from causes other than abuse ... by their site and number, and by a carefully taken clinical history." (1346)

The physical signs looked for were

anal or perianal scar	42 cases
tag	16
reflex dilatation	17
funnel	8
perianal venous congestion	18
normal	3

Perianal venous congestion is supportive of previous abuse, especially if it occurs during the first seconds of the examination. Agreeing with Hobbs, 1999 and disagreeing with McCann, Voris, 1989 about venous congestion in the chronic setting. Perianal venous congestion was defined as purple blue to black discoloration appearing within the first 30 seconds of the examination. McCann Voris found that “venous congestion per se should be of no significance in the recognition of abuse, but the present author agrees with Hobbs et al.’s observations on the subject and in particular the affirmation that its presence, when associated with other signs, is supportive of previous abuse, especially if it occurs in the first seconds of the examination.”

Reflex anal dilatation was described as a dynamic finding (not fixed dilatation) if there was the visibly relaxed sphincter to greater than 1 cm in the absence of stool in the ampulla. “The pathogenesis and significance of RAD is debated. It appears with similar frequency to venous congestion, but RAD is an ‘uncommon physical sign’ in nonabused children, and its presence is likely to be significant.” (1346)

Funnelling was defined as “a fixed funnel shape” of the anus (Hobbs & Wynne). On funnelling, our sample size was insufficient to be conclusive.

Fissures were defined as “a discontinuity in the lining of the anal canal.” (Hobbs & Wynne) Constipation is a common cause of fissures. Fissures heal and leave scars, usually in the anterior midline in 2-3 year-olds. (citing Agnarsson et al., 1990.) “A single fissure, and the subsequent scar even if single and meridian may indicate previous abuse, if other pathogenic modalities, such as documented severe constipation, Crohn’s disease, previous chemotherapy, or local surgery are excluded. The experience here, in agreement with Hobbs et al., is that the significance of a scar as an indicator of possible remote abuse increases if it is multiple, in a site outside of the midline, or extending away from the anus, and if other signs are present.” (1346). “Over the past few years there has been an intense debate in Italy regarding the significance of scars and mainly the single scar. It is agreed that constipation is a common cause...” (1346)

* [It should be noted that Dr Bruni is a disciple of Dr C J Hobbs (Hobbs & Wynne, *q.v.*), who “made important suggestions and reviewed the manuscript... Without [his] help this paper could not have been written.” It appears to me that this work was, in effect, sponsored by Dr Hobbs with a view toward rehabilitating his earlier work whose validity has been called into question by McCann, Voris and others. The author comments that “The greater frequency of physical signs seen in this study, compared with other studies, (citing Hobbs & Wynne, English boys, 1988), may be due to the careful selection of cases.” By which the author apparently means the fact that all of his cases were cases in which a confession and a judicial conviction had been obtained, unlike the earlier work of Hobbs that found a much lower frequency; however, I wonder whether there mayn’t be circular reasoning here: these were cases in which Dr Bruni examined for the prosecution, and in which it is reasonable to assume that Dr Bruni’s report was a factor in bringing about the confession. In other words, my findings are from confessed cases, but the confessions resulted from my findings. Also that there are no controls; all 50 cases are abused children. Dr Bruni seems to acknowledge these problems, along with the small sample size to which he refers, by means of a concluding sentence that “A comparative or case-control study with carefully selected ‘abused’ and ‘nonabused’ children is required to assess the importance of physical signs.” He fails to acknowledge that that study has already been done -- by McCann Voris. --JKR]

Genital anatomy in non-abused pre-school girls. Myhre AK, Berntzen K, Bratlid D. Acta Paediatrica 2003; 92: 1453-1462. (Norway) Studied 195 subjects selected for nonabuse with photocolposcopy, including hymenal measurements. Found that 36 had a gaping hymenal orifice, 3 had a discharge, 15 had labial adhesions. Specifically addressed the posterior hymen: found that in the supine position 1 had a

posterior notch, 0 had a deep notch or a transection. In the knee-chest position 0 had a superficial notch and 0 had a deep notch or a transection.

Normal studies are essential for objective medical evaluations of children who may have been sexually abused. Adams J. *Acta Paediatrica* 2003; 92: 1378-1380. Comment on Myhre et al., above. Joyce Adams comments that measurements are "difficult and...at best, estimates," and that "clinicians should focus instead on the continuity of the hymenal rim. If there is a clear rim of hymenal tissue in the posterior aspect of the orifice, and the free edge of the hymen can be followed visually at least from the 3:00 to the 9:00 positions when the patient is supine, this is likely to be a normal finding."

Anogenital warts in children: sexual abuse or not? Hornor G. *Journal of Pediatric Health Care* 2004; 18(4): In children under 3, these are not diagnostic of sexual abuse bc these children's anogenital areas are routinely touched by adults who may carry HPV on their fingers. In this group, warts should trigger a psychosocial history and a search for other evidence of abuse, but need not be reported. In the over-3 age group, they must be reported as giving rise to a reasonable suspicion of sexual abuse. Abstracted on Medscape.

Anal fissures and anal scars in anal abuse -- are they significant? Pierce AM. *Pediatric Surgery International* 2004; 20: 334-338. A British specialist in sexual abuse examined 214 children referred to her. According to John McCann's summary in the *Quarterly* for Winter 2005,

Group A	81 children with no history of abuse. 2 (2%) had anal fissures.
Group B	83 children who alleged sexual abuse but denied anal abuse. 9 (11%) had anal fissures or anal scars.
Group C	50 children who gave a strong history for anal abuse. 41 (84%) had anal fissures or anal scars.

Dr McCann comments that these numbers are much higher than any other investigator has reported, and points out that the definitive clinical diagnosis of scar tissue in the anus is fraught with difficulty. He doubts the validity of the results.

Correlation of colposcopic anogenital findings and overall assessment of child sexual abuse: a prospective study. Cheung PC, Ko CH, Lee HY, Ho LM, To WW Ip PL. *Hong Kong Med J* 2004 Dec; 10(6): 378-383. Examined 77 children prospectively (age range 6 mos to 16 years), including 16 confirmed cases of abuse. Found the following anogenital findings:

normal	45%
nonspecific	29%
concerning	13%
abnormal	13%

Of the 16 cases of confirmed abuse, 7 had normal or nonspecific colposcopy. The SENS and SPEC of definitely abnormal colposcopic findings for diagnosing abuse were

SENS	56%
SPEC	98%

The abstract does not say how abuse was confirmed.

Vulvar and genital trauma in pediatric and adolescent gynecology. Merritt DF. *Curr Opin Ob Gyn* 2004 Oct; 16(5): 371-381. From the abstract: "Many recently published articles have noted that the history as given by the child is the most important factor in determining the etiology of genital injuries as abuse or accidental. The history is more important than any findings on physical examination. Photographic

documentation of injuries by photocolposcopy has allowed a new level of peer review, improving the understanding of what are normal and nonspecific findings, many of which were considered to be abnormal in the past.

Skeletal injuries associated with sexual abuse. Johnson K, Chapman S, Hall CM. (UK) Pediatric Radiology 2004 Aug; 34(8): 620-623. From the abstract: Sexual abuse is often associated with physical abuse. Usually soft tissue injuries, but fractures occur in 5% of sexually abused children. We report three sexually abused children who had fractures:

A 3 year old girl with extensive soft-tissue injuries to the arms, legs, and perineum sustained fractures of both pubic rami and the right sacroiliac joint.

A 5 month old girl with an introital tear had an undisplaced left femoral shaft fracture.

A 5 year old girl presented with an acute abdomen and pneumoperitoneum due to a ruptured rectum due to sexual abuse. She had old healed fractures of both pubic rami with disruption of the symphysis pubis.

Child sexual abuse. Johnson CF. Lancet 2004 Jul 31; 364(9432): 462-470. From the abstract: Child sexual abuse is a worldwide concern. It affects 2-62% of women and 3-16% of men. Psychological and medical consequences can persist through adulthood. Associated sexually transmitted diseases and suicide attempts can be fatal. All physicians should become familiar with anogenital examination of children and should perform it routinely, with awareness of the potentially fatal long term sequelae of child sexual abuse. Because as many as 96% of children assessed for suspected sexual abuse will have normal genital and anal examinations, a forensic interview by a trained professional must be relied on to document suspicion of abuse.

Sexual Assault: Victimization Across the Life Span: 2 volume set. Angelo P. Giardino, Barbara Girardin, Elizabeth Datner, Janice Asher, Diana Faugno, Mary Spencer. Saint Louis: G.W. Medical Publishing, Inc., 2004.

Update on childhood sexual abuse. Sapp MV, Vandeven AM. (Harvard) Curr Opin Pediatr 2005 Apr; 17(2): 258-264. (See also Update on child maltreatment with special focus on shaken baby syndrome, in the same issue.) CSA is a problem of epidemic proportions. Reviewed the literature. The late effects of CSA can be permanent and life-threatening. Clinicians should become familiar with the clinical and behavioral indicators that signal early CSA, because children do not usually disclose it. *Child advocacy centers* are intended to decrease stress and provide adequate protection and services for the child [as the first priority, it appears --JKR], while still optimizing the chances for a successful legal outcome. Improved awareness for communities and professionals is required to assure quality care for all children who are sexually abused.

Anal fissures and anal scars in anal abuse -- are they significant? Pierce AM. (U.K.) Pediatr Surg Int 2004; 20: 334-338. According to Dr McCann's summary in the 05 Winter *Quarterly*, this British researcher reviewed seven years of her experience examining 214 children referred to her because of possible sexual abuse. She divided them into three groups:

Group A	81 chil with no hx or ev of sexual abuse	2 had anal fissures
Group B	83 chil + hx sexual but denied anal abuse	9 had fissures or scars
Group C	50 chil + hx anal abuse	41 had fissures or scars

Fissures were defined as "breaks in the mucosa extending to the perianal skin." No photographs were taken. Dr McCann points out the identifying scar tissue in this area is very difficult. Also that the number of positives is much higher than most studies have found. Also that the lack of photographs makes it impossible to peer review the work.

The aetiology of paediatric inflammatory vulvovaginitis. Cuadros J, Mazon A, Martinez R et al. (Madrid) Eur J Paediatr 2004; 163: 105-107. Autoinoculation from the upper respiratory tract. Cultured 74 subjects mean age 5.8 years. The main risk factor was a history of recent URI. 47 Strep pyogenes, 12 Hemophilus influenzae, some polymicrobial., Corynebacterium, Staph epi, Gardnerella vaginalis, E coli, Pseudo.

Do pediatric chief residents recognize details of prepubertal female genital anatomy? A national survey. Dubow SR, Giardino AP, Christian CW, Johnson CF. Child Abuse & Neglect 2005; 29: 195-205. Used the same two black-and-white photographs previously used. Found that out of 139 respondents, 50% considered their training to be insufficient. As far as correctly identifying the parts, had

clitoris	94%
posterior commissure	87%
urethra	63%
labia majora	80%
labia minora	90%
hymen	64%

Dr Reece comments that this level of training is “inadequate.”

Correlation of colposcopic anogenital findings and overall assessment of child sexual abuse: prospective study. Cheung PC, Ko CH, Lee HY, Ho LM, To WW, Ip PL. Hong Kong Med J 2004; 10(6): 378-383. Conclusions: Anogenital findings are often normal or non-specific in sexual abuse. In general, colposcopy examination findings do not directly reflect the final diagnosis. A category-4 finding on colposcopy is very helpful in confirming definite abuse, whereas other findings do not rule out the diagnosis. The SENS of abnormal findings was 56% and the SPEC was 98%. Colposcopy showed a fair correlation ($\kappa=0.245$) with the overall medicolegal assessment of abuse. Seven of the 16 confirmed cases of abuse had normal or nonspecific findings. The diagnostic impact of normal, nonspecific, concerning, and definite findings was (expressed as likelihood ratios) 0.23, 1.12, 0.00, and 34.3 respectively.

AAP Clinical report: the evaluation of sexual abuse in children. Nancy Kellogg MD and the Committee on Child Abuse & Neglect, AAP. Pediatrics 2005 Aug; 116(2): 506-512. Updates the 1991 and 1991 reports. Each year appr 1% of children experience some form of sexual abuse, resulting in the sexual victimization of 12% - 25% of girls and 8% - 10% of boys. Definition: “Sexual abuse occurs when a child is engaged in sexual activities that he or she cannot comprehend, for which he or she is developmentally unprepared and cannot give consent, and or that violate the law or social taboos of society. (citing Kempe, 1978). The sexual activities may include all forms of oral-genital, genital, or anal contact by or to the child or abuse that does not involve contact, such as exhibitionism, voyeurism, or using the child in the production of pornography.”

Physical examination. The examiner has to be a health care provider who is licensed to make medical diagnoses and recommend treatment. (This is new). Any abnormalities should be interpreted appropriately with regard to the specificity of the finding to trauma (e.g. nonspecific, suggestive, or indicative of trauma). ...Measurements of the hymenal orifice alone are not helpful. Discusses STD testing.

Diagnostic considerations. “The diagnosis of child sexual abuse often can be made on the basis of a child’s history. Sexual abuse is rarely diagnosed on the basis of only physical examination or laboratory findings. Physical findings are often absent even when the perpetrator admits to penetration of the child’s genitalia. (citing Muram, 1989; Kerns & Ritter, 1992; Heger, Ticson, Velasquez, Bernier, 2002) ... Findings that are concerning include: (1) abrasions or bruising of the genitalia; (2) an acute or healed tear in the posterior aspect of the hymen that extends to or nearly to the base of the hymen; (3) a markedly decreased amount of hymenal tissue or absent hymenal tissue in the posterior aspect; (4) injury to or scarring of the posterior fourchette, fossa navicularis, or hymen; and (5) anal bruising or lacerations. The interpretation of physical findings continues to evolve as evidence-based research becomes available. (citing Adams, The evolution of a classification scale, 2001) ...Table 2 provides suggested guidelines for making the decision to report based on currently available information. [Table 2 lists blocks for “data available” and “response.” For example, under “history” it may be “clear statement” or “none or vague.”

Then there are “behavioral symptoms,” “physical examination,” and “diagnostic tests.” Under “response” there are “level of concern about sexual abuse” and “report decision.” Under “level of concern,” there can be “high” or “intermediate.” Under “report decision,” there can be “report,” “refer when possible,” etc. The physical examination findings that are associated with a “high” level of concern range from normal to nonspecific to concerning. The level of concern depends upon the other columns. The diagnostic tests that are associated with high level of concern are C trachomatis, T vaginalis, HIV, syphilis, herpes, but under herpes the asterisk says “If nonsexual transmission is unlikely or excluded.”] The differential diagnosis of genital trauma also includes accidental injury and physical (i.e. non-sexual) abuse. This differentiation may be difficult and may require a careful history and multidisciplinary approach. Because many normal anatomic variations, congenital malformations and infections, or other medical conditions may be confused with abuse, familiarity with these other causes is important. (citing Bays & Jenny, 1990; AAAP, AAP, ACOG, Protecting adolescents, 2004).

Legal issues. Discusses the unlikelihood of prosecution for failure to report, particularly in doubtful symptoms. Detailed records and drawings needed. Discusses divorce proceedings.

The Anatomy of Child and Adolescent Sexual Abuse: A CD-ROM Atlas/Reference. John J. McCann, MD, FAAP and David L. Kerns, MD, FAAP. InterCorp, Inc., 11469 Olive Blvd Suite 225, St. Louis, MO 63141. (2006) <http://www.intercorpinc.com> 1200 photographs, 600 cases, 400 references, 150 topics, 2 dozen morphs. Excellent photographs presented by the leading expert. But substantially the same material already appears in print, in refs mentioned above.

Anogenital and respiratory tract human papillomavirus infections among children: age, gender, and potential transmission through sexual abuse. Sinclair KA, Woods CR, Kirse DJ, Sinal SH. Pediatrics 2005 Oct; 116(4): 815-825. To see how HPV presents in children under 13 and its association with CSA. Had 124 chil with disease -- 10 with oral, 40 with laryngeal, 67 with anogenital, and 7 with both. The authors observed that 73% of the patients with anogenital lesions got referred to a CSA clinic, while none of the oral-respiratory ones did. There was an association with CSA. HPV of any type had a 70% PPV for possible sexual abuse in chil over 8, only 36% for chil under 8.

Diagnostic findings in alleged sexual abuse: symptoms have no predictive value. Kelly P, Koh J, Thompson JM. J Pediatr Child Health (NZ) 2006; 42: 12-117. According to Dr Krugman’s review in the summer 2006 *Quarterly*, summarizes descriptive data on 2134 suspected CSA patients from a Maori clinic. Found that 69% had normal genital exams, 20% had nonspecific findings, and 5% had findings diagnostic of penetration. Symptoms were not predictive of positive findings. Dr Krugman comments that this is what was already well known.

When is a child not a child? Lecture by Carole Jenny on sexual maturation, San Diego Conference, January 2007. http://www.chadwickcenter.org/CD/Thursday/ThursdayDocs/L3_jenny.pdf PowerPoint lecture. Useful for gauging sexual maturity for purposes of evaluating child pornography and other forensic applications. See also Sun et al., 2002, above. See also the limited discussion in *Nelson’s*.

The healing of hymenal injuries in prepubertal and adolescent females: a descriptive study. McCann J, Miyamoto S, Boyle C, Rogers K. Pediatrics Electronic Pages 2007 May; 119(5): e1094. A large multicenter study from 25 centers that retrospectively examined the photographs and documentation of hymenal exams in 239 hymenally injured girls ranging in age from 4 months to 18 years. The purpose was to study the time-course and possible dating of hymenal injuries. (Note that all the subjects had injuries.)

Had 113 prepubertal and 126 pubertal. 87% were examined within 48 hours of injury; all the rest were examined within 72 hours. Followup imaging. Presents a chart showing the time of disappearance of various injuries:

<u>Injury</u>	<u>Group</u>	<u>Severity</u>	<u>Last detected</u>	<u>Earliest disapp</u>	<u>Gone</u>
Abrasion	Pre		1d	3d	3-22d
	Pub		4d	11d	11d
Blood blister	Pre		7d	30d	
	Pub		34d	9d	

Hematoma	Pre		1d	
	Pub	1d	3d	3-20d
Petechiae	Pre	1d	1d	2-211d
	Pub	2d	2d	3-109d
Submucosal hemorrhage	Pre			
		mild	2d	3d
		mod	8d	5d
		marked	15d	5d
				3-942d
				10-304d
				16-730d
	Pub			
		mild	7d	4d
		mod	12d	3d
		marked	11d	3d
				8-29d
				14-36d
				12-97d

Conclusion: “The hymenal injuries healed rapidly and except for the more extensive lacerations left no evidence of a previous injury. There were no significant differences in the healing process and the outcome of the hymenal injuries in the 2 groups of girls [pubertal and prepubertal].” [Notice that: “*no evidence* of a previous injury.” Once the injury has healed, which is soon, you can’t see anything. What about all those past legal cases where medical experts testified in court that there was evidence of a previous injury? What becomes of those convictions now? See Dr Dubowitz’s carefully worded editorial next below. --JKR]

Healing of hymenal injuries: implications for child health care professionals. Dubowitz H. Pediatrics 2007 May; 119(5): 997-999. Editorial comment on the above article by McCann et al. Dr Dubowitz notes that it took a huge multicenter study to come up with 239 hymenal injuries – enough to provide some scientific basis for drawing conclusions, showing how uncommon it is for the CSA examination to be abnormal. He questions the recommendation that the exam should “always” include both the supine and the knee-chest (prone) position, in order to not miss anterior injuries; he says that acute injuries are not missed in the supine position, and the prone position is embarrassing. As far as dating of injuries, which was the primary goal of the study, the study showed that dating of injuries “was difficult and of limited forensic value.” The finding of complete healing in numerous subjects “confirms the need for considerable constraint in interpreting the physical findings beyond the acute period.” Since there is rapid and complete healing and liquid evidence disappears, examinations need to be conducted soon, within a day -- but not in the emergency room in the middle of the night with inexperienced personnel. Comments on the need for uniform terminology -- avoid duplicative terms like “notch/cleft” and “tear/transection.” Dr Dubowitz comments on the fact that some past diagnostic criteria have turned out to be erroneous, and the fact that this implicates medicolegal consequences. The study “indirectly shows where earlier thinking turned out to be erroneous.” [advertises to the issue of hymenal diameters] “It is the nature of science that knowledge evolves. However, there is an ethical concern here if such previous knowledge contributed to a finding of sexual abuse, a child being removed from the home, and someone being incarcerated. [citing Dubowitz, Ethical issues, 1997] In general, these decisions were based on more than 1 physical finding. Nevertheless, there may be a need to ‘set the record straight’ despite the logistic challenges of whom to contact, how to reach them, and over what period of time.” Comment by JKR: I have examined the “Ethical issues” article of 1997 that Dr Dubowitz refers to. It does not deal with obsolete or faulty diagnostic criteria, nor with incarceration. The closest it comes is in a discussion of “The ethics of screening,” where it discusses the fact that pediatricians fail to report potential abuse to the CPS because of concerns about poor quality service by the CPS and the worry that a report may do the child more harm than good.

FEMALE CIRCUMCISION

(FGM)

California Penal Code Sec. 273.4-(a). “If the act constituting a felony violation of subdivision (a) of section 273a was female genital mutilation, as defined in subdivision (b), the defendant shall be punished

by an additional term of imprisonment in the state prison for one year, in addition and consecutive to the punishment prescribed by section 273a.

(b) “‘Female genital mutilation’ means the excision or infibulation of the labia majora, labia minora, clitoris, or vulva, performed for nonmedical purposes.

(c) “Nothing in this section shall preclude prosecution under Section 203, 205, 206, or any other provision of law.”

Female circumcision as a public health issue. Nahid Toubia, MD. NEJM 1994; 331(11): 712-716.

Female Genital Mutilation: A Call for Global Action, second edition. Nahid Toubia, MD. 1995. ISBN 1-893136-04-3.

Training kit for health professionals and educators. Nahid Toubia, MD (1995). \$150. See web site, above. 26 color slides with speaker’s notes.

Female genital mutilation: the penultimate gender abuse. Donald G. Barstow. Child Abuse & Neglect 1999; 23: 501-510. Carole Jenny notes in her review in Child Abuse Quarterly that a federal statute, 18 U.S.C. Sec. 11601/06/97 outlawed FGM throughout the US in 1997. This article is an extensive review.

Female genital mutilation. AAP Policy Statement RE9749. Committee on Bioethics. Pediatrics 1998 Jul; 102(1): 153-156. <http://aappolicy.aappublications.org/cgi/content/full/pediatrics%3b102/1/153> Opposes the practice.

Visual Diagnosis of Child Sexual Abuse. Carole Jenny and Deborah Lowen. AAP, 1998. Slide program includes two photos of FGM (slides # 58, 59)

The RAINBO organization dedicated to the issue of female circumcision worldwide.
<http://www.rainbo.org>

Female Genital Mutilation. WHO Fact Sheet, June 2000.
<http://www.who.int/mediacentre/factsheets/fs241/en/>

Nurses and request for female genital mutilation: cultural rights versus human rights. Sala R, Manara D. Nursing Ethics: An International Journal for Health Care Professionals 2001; 8(3): 247-258. Discusses the situation where nurses are asked by a patient’s family to arrange or assist in female circumcision.

Preventing ethical dilemmas: understanding islamic health care practices. Ott BB, Al-Khadhuri J, Al-Junaibi S. Pediatric Nursing 2003; 29(3): 227-230. Discusses Islam as a way of life, involving health-related issues and behavior issues relevant to nursing care. The authors are two nursing graduate students from Muscat. They emphasize that the cultural practices of Muslims differ considerably from place to place, according to local custom, just as do those of members of other religions. As far as FGM, the brief discussion immediately follows a paragraph on male circumcision. The brief discussion is noncommittal, referring to the AAP statement of 1998 and to a JAMA article, September 2002. The authors do not state that FGM is required or even encouraged by Islam; they state, “Female children in some Muslim cultures sometimes experience female circumcision. For some Muslim women, female circumcision is a source of pride and belonging, for others, it is experienced as genital mutilation. Nurses have been torn between supporting a respected cultural practice and abhorring torture of the female species...” (citing Sala & Manara, 2001).

Female Genital Cutting. US DHHS National Women’s Health Center publication, February 2005. Available at <http://www.4woman.gov/faq/fgc.htm> See also WHO fact sheet, “FGM,” 2000, above.

Razor's Edge -- The Controversy of Female Genital Mutilation: When Culture Harms the Girls -- The Globalization of Female Genital Mutilation. (March 2005).
<http://www.irinnews.org/webspecials/FGM/default.asp>

Female genital mutilation: knowledge, attitude, and practice among nurses. Onuh SO, Igberase GO, Joanness OU, et al. J Nat Med Assoc 2006; 98: 409-414. (Benin) According to the review by Deborah Pullin RN in the Autumn 2006 Quarterly, only 4.4% of these 182 Nigerian nurses said they would have their own daughters circumcised, only 6.6% routinely perform FGM, and 86% said it was not a good practice. The purpose of the study was to examine the mechanism of cultural change.

Prosecution of female genital mutilation in the United States. Rena Marie Justice. American Prosecutors Research Institute *Update*, vol 19, no. 7, 2006. Exists in 28 African countries as well as places in Asia and the Middle East, and increasingly among immigrant groups in Europe and North America. Eighteen African countries have prevalence rates of 50% or higher. There are several forms (citing US DHHS National Women's Health Center publication, *Female Genital Cutting*, above as well as the WHO fact sheet and *Razor's Edge*, above). There's clitoridectomy, excision (which includes the labia minora), and infibulation, which involves removal of most or all of the external genitalia with stitching together of the sides of the vagina. A host of complications, including infections, HIV, incontinence, dyspareunia, dysuria, dysmenorrhea, difficulty in childbirth, depression and other psychological effects. The cultural beliefs that support it include curbing promiscuity, ensuring marriageability, and initiation. Discusses FGM in the United States. Somali communities. 228,000 females in the U.S. either have it or are at risk for it. In 1996 Congress enacted the Federal Prohibition of Female Genital Mutilation Act, 18 U.S.C. Sec. 116. There have been no published prosecutions under this act. State legislation: 18 states have statutes. (For California, see PC 273.4(a), above). Discusses possible defenses. As I read this report, apparently there has never been a prosecution under any of these statutes.

Female genital mutilation and obstetric outcome: WHO collaborative prospective study in six African countries. Banks E, Meirik O, Farley T et al. Lancet 2006; 367: 2835-2841. Found an elevated complication rate. Gives a classification of FGM:

FGM-I	removal of prepuce and clitoris
FGM-II	removal of clitoris and labia minora
FGM-III	removal of part or all of the external genitalia with stitching or narrowing of the vaginal opening

Accompanying editorial from a Nigerian medical school says UTI is a frequent complication. Says there can be no justification.

COCAINE BABIES AND METHAMPHETAMINE

see also "Drug Abuse" and "SIDS Biology" and "Domestic Violence"

Cocaine intoxication in a breast-fed infant. Chasnoff IJ, Lewis D, Squires L. Pediatrics 1987; 80: 836-838

Drug-addicted mothers, their infants, and SIDS. Rosen TS and Johnson HL. Ann NY Acad Sci 1988; 5 - 10X increased risk of SIDS. Higher rate of prematurity, fetal distress, VD, perinatal asphyxia, in these polydrug abusers. Studied 111 pregnant women in a drug abuse clinic. See under "SIDS Biology," above.

Neonatal body proportionality and body composition after in-utero exposure to cocaine and marijuana. Frank DA, Bauchner H, Parker S et al. J Pediatr 1990; 117: 622-626.

Cocaine and fetal death. Morild I, Stajic M. FSI 1990; 47: 181-189.

Detection of fetal cocaine exposure by analysis of amniotic fluid. Jain L, Meyer W, Moore C, Tebbett I et al. Ob Gyn 1993 May; 81(5 part 1): 787-790

Risk of intracranial hemorrhage and other adverse outcomes after cocaine exposure in a cohort of 323 very low birth weight infants. Dusick AM et al. J Peds 1993; 122(3): 438-445.

Cocaine and alcohol use in pregnancy. Moroney JT and Allen MH. In: *Neurological Complications of Pregnancy*. New York: Raven Press, 1994: 231-242.

Prenatal Cocaine Exposure. Konkol RJ and Olsen GD. Boca Raton: CRC Press, 1996. ISBN 0-8493-9465-1 Ch. 1 Methodological issues and neurological correlates. Ch 2 And the fetomaternal unit. Ch 3 Obstetrical pathophysiology. Ch 4 Neonatal strokes? Ch 5 Vascular complications Ch 6 Behavioral and convulsive effects. Ch 7 Neonatal cocaine withdrawal. Ch 8 Eye. Ch 9 Behavioral consequences. Ch 10 Rodent model. Ch 11 PET scans. This book is in the Carpenter Library.

Retinal hemorrhages associated with in utero exposure to cocaine: experimental and clinical findings. Silva-Araujo AL, Tavares MA, Patacao MH, Carolino RM. Retina 1996; 16(5): 411-418. (have) See under "RH -- other."

Mortality within the first 2 years in infants exposed to cocaine, opiate, or cannabinoid during gestation. Ostrea EM Jr., Ostrea AM, Simpson PM. (Detroit) Pediatrics 1997 Jul; 100(1): 79-83. Prospective study of almost 3,000 infants from birth through 2 years. At birth, 44% of the infants tested positive for drugs (!?), incl 30% +coc. The drug + infants were born with smaller weight, smaller heads, and had single, multigravid, multiparous mothers w no PNC. Within the first 2 yrs, 44 total infants died (both drug+ and drug-), for an overall crude mortality of about 15/1000 LB. Eleven of these were closed as SIDS. Positivity for both coc and op at birth was associated with higher mortality for LBW infants (odds ratio of 5.9) but not for normal BW infants. "We conclude that prenatal drug exposure in infants, although associated with a high perinatal morbidity, is **not** associated with an overall increase in their mortality rate or incidence of SIDS during the first 2 years of life. However, a significantly higher mortality rate was observed among low birth weight infants who were + for both coc and op."

Maltreatment of children born to women who used cocaine during pregnancy: a population-based study. John M. Leventhal, Brian WC Forsyth, Keqin Qi, et al. Pediatrics 1997; 100(2). E7. Gives complete internet ref in text; this is an electronically published article only abstracted in print. (have abstract). Increased risk of maltreatmt.

Maltreatment of children born to women who used cocaine during pregnancy: a population-based study. Leventhal JM, Brown WC, Forsyth MB et al. Pediatrics electronic pages 1997; 100(2): 258 (abstract). Text at pediatrics.or/cgi/content/full/100/2/e7. Reviewed the medical records of 139 cocaine-exposed infants versus a control group of non-cocaine-exposed infants from the same social class, followed for the first 2 years of life. Found that 9% of the exposed infants versus 1% of the nonexposed infants had either been maltreated or had been removed from the home. Relative risk =6.5 BUT, after controlling for covariables in baseline clinical status and social factors, the adjusted odds ratios were no longer statistically significant; ratios decreased to 4 for maltr and 1.6 for placement. 80% of the exposed and control children were Black, 96% of the mothers were Medicaid. Conclusion: "In this study, children born to women who used cocaine during pregnancy were at a substantially increased risk of maltreatment or placement outside the home compared with a sociodemographically similar comparison group. Differences in baseline variables between the two groups, however, partially accounted for this increased risk. Therefore a mother's use of cocaine is more likely a marker of increased risk rather than a single explanatory variable."

Incidence of passive exposure to crack/cocaine and clinical findings in infants seen in an outpatient service. Lustbader AS, Mayes LC, McGee BA, Jatlow P, Roberts WL. Pediatrics 1998; 102(1). URL: <http://www.pediatrics.org/cgi/content/full/102/1/e5>. Significant numbers of young children are passively exposed to such levels that it shows up in the urine. It is associated with respiratory symptoms.

Summarized in Child Abuse Quarterly, Oct. 98: Article reviewed by Ira Chasnoff in Pediatrics 1998; 102: 145-147. Chasnoff opines that exposing children to drugs is child abuse.

Prenatal exposure to cocaine and other drugs: outcome at four to six years. Ann N Y Acad Sci 1998. Chasnoff IJ, Anson A, Stenson H et al. Chasnoff later cites this article for the proposition that "prenatally exposed children showing significantly high rates of depression, anxiety, aggressive behavior, thought problems, impulsivity, and distractability." see Chasnoff, Pediatrics 1998.

Birth outcome from a prospective, matched study of prenatal crack/cocaine use: I. Interactive and dose effects on health and growth. Eyler FD, Behnke M, Conlon M, Woods NS et al. Pediatrics 1998; 101: 229-237

Prenatal cocaine exposure and school-age intelligence. Wasserman GA, Kline JK, Bateman DA et al. Drug Alcohol Depend 1998 May 1; 50(3): 203-210. They tested IQ at age 6-9 in 88 children born with prenatal cocaine exposure. They tried to control for the effects of socioeconomic cofactors, including caregiver IQ and home environment. They found that IQ scores DID NOT DIFFER between exposed children and non-exposed children.

Study: prenatal cocaine not so bad on a child. ABCNews.com, March 28, 2001. No convincing evidence that prenatal cocaine exposure is associated with negative developmental effects in children 6 years and younger. Poverty, not cocaine to blame. A meta-analysis in JAMA by lead author Dr. Deborah Frank, associate professor of pediatrics at BU School of Medicine, reviewing 36 studies published since 1984.

Growth, development, and behavior in early childhood following prenatal cocaine exposure. A systematic review. Frank DA, Augustyn M, Knight WG, Pell T, Zuckerman B. JAMA 2001; 285: 1613-1625. Reviewed by Howard Dubowitz in Child Abuse Quarterly for July 2001. Literature review of 36 articles. No unique developmental effects.

Developing language skills of cocaine-exposed infants. Singer LT, Arendt R, Minnes S et al. Pediatrics 2001; 107: 1057-1064. Measured meconium BE levels as well as maternal history in 245 infants and stratified them by level as heavily exposed, lightly exposed, or not exposed. At one year gave them a language test and found a dose-response curve for poor auditory comprehension.

The search for congenital malformations in newborns with fetal cocaine exposure. Behnke M, Eyler FD, Garvan CW, Wobie K. Pediatrics 2001 May; 107(5): e74. A prospective longitudinal cohort study of 272 offspring of 154 prenatal crack abusers and 154 nonusing controls matched for race, parity, PNC, and class. Using anthropomorphic measures. Results: there were no differences on major risk variables between the included and excluded infants. There were significantly more premature infants in the cocaine group. Cocaine infants were significantly smaller in birth weight, length, and HC, but did not differ on other anthropomorphics.

Behind the drug: the child victims of meth labs. APRI Update vol 15 no 2, 2002. Mark Ellis JD, Barbara Sturgis PhD, Gregg Wright MD. By an assistant professor at the Univ of Nebraska, a clinical psychologist, and a pediatrician, all at the Univ of Nebraska Center on Children, Family, and the Law. Describes the meth acute effects (rush lasting for minutes, euphoria and increased activity lasting for several hours, sleeplessness and irritability lasting for several days), preoccupation with maintaining the high, "tweaking" (unpleasant state at the end of a binge, alleviated by taking other drugs such as alcohol), "crash" with sleeping for several days. Tolerance is marked; more and more drug required to attain and maintain a high. Users are completely focused on preventing the crash. Insomnia, hallucinations, paranoia, delusions, and rages. Exposure to criminals. Users "overhear" conversations saying that their children will be harmed or taken away. Manufacture: fumes of phosphine, ammonia, organic solvents; explosions, burns. Iodine or lithium batteries. Toxic chemicals and toxic adults. Does not say anything about passive exposure of children to drug use. "Each level of meth use has an impact on the user's ability to care for children. Low-intensity abusers can continue to function, but experience mood swings. When high, abusers feel good, are active, and can get a lot accomplished. They are also irritable and impatient, increasing the possibility of abusive behavior. When the effects of the drug wear off, even low-level abusers are

likely to feel an increased need to sleep, which can interfere with caregiving. Binging user ... irritability increases and the abuser can become argumentative, assaultive, and threatening. Children are often left to fend for themselves. ...exposure to weapons...

[Cocaine exposure in utero tied to cognitive, developmental problems in early life.] Singer LT. JAMA 2002 Apr 17; 287: 1952-1960, with an accompanying editorial by Dr. Barry Zuckerman at 1990-1991. Reuters Medical News on Medscape. Developmental delays during the first 2 years of life. Did Bayley scales prospectively on 415 infants from high-risk lower-class Black families. 218 cocaine-exposed, 197 not exposed. Found that at 24 months the exposed had a 6-point deficit compared to the not-exposed, consistent with a mental score of 80 with significant developmental delay. Continued deficits at 4 years. The editorial argues that "[T]hese findings should not be used to promote stigmatization of cocaine-exposed children by clinicians and the media with potential resurgence of uniquely punitive (and clinically harmful) legal measures directed at women who use cocaine during pregnancy." [Note by JKR: Cocaine-exposed newborns are usually taken away from their parents at birth and only restored later, if at all. To what extent could these deficits be due to foster care and multiple custody?]

Drug-exposed infants and developmental outcomes: effects of home intervention and ongoing maternal drug use. Schuler ME, Nair P, Kettinger L. Arch Ped Adol Med 2003; 157: 133-138. A randomized controlled trial of home intervention, with Bayley Scales as an endpoint, 108 subject dyads. Drug-exposed infants had higher Bayley scores than control infants(!) Home intervention improved outcomes, mainly by ending continued maternal drug use.

Conviction of stillborn baby's mother upheld. South Carolina case involved woman who used cocaine and a new statute extending abuse to include unborn children. By David E. Savage, Times staff writer. The Los Angeles Times, October 6, 2003. Defendant Regina McKnight, mildly retarded 26 year old gave birth to a stillborn 35-weeker that was cocaine-positive, as was Regina. Convicted of "homicide by child abuse," under a "newly created crime." US Supreme Court denial of certiorari. *McKnight v. South Carolina*, Oct. 6, 2003, 540 U.S. No. 02-1741. The Times story says this was the first in the nation to be convicted of homicide for using cocaine during pregnancy causing stillbirth. The statute applied was "homicide by child abuse," which carries a maximum sentence of 20 years. The issue in dispute was the South Carolina Supreme Court's interpretation of this statute to apply to the cocaine-fetus situation. A separate South Carolina statute makes it a crime to intentionally kill a fetus, leading to a two-year term.

Fetal bilateral renal agenesis, phocomelia, and single umbilical artery associated with cocaine abuse in early pregnancy. Kashiwagi M, Chaoui R, Stallmach T, Hurlimann S, Lauber U, Hebisch G (Zurich). Birth Defects Research Part A. Clinical Molecular Teratology 2003 Nov; 67(11): 951-952. From the abstract: "Maternal cocaine abuse in pregnancy is associated with complications such as IUGR, abruptio placentae, and preterm delivery. We report what is, to our knowledge, the first published observation of fetal bilateral renal agenesis associated with a vascular disruption syndrome comprising upper limb reduction defect and a single umbilical artery following maternal cocaine abuse in early pregnancy. Conclusion: This constellation in a fetus aborted at 18 weeks extends the spectrum of complications possibly associated with cocaine abuse during pregnancy."

Cognitive outcomes of preschool children with prenatal cocaine exposure. Singer LT, Minnes S, Short E, Arendt RE et al. JAMA 2004 May; 291(20): 2448-2456. 190 exposed and 186 nonexposed. Tested at age 4. Was not associated with lower overall IQ or verbal performance scales, but WAS associated with small but significant deficits on some subscales. BUT, if they had a good home environment, their IQ's were the same as nonexposed children.

Children prenatally exposed to cocaine: developmental outcomes and environmental risks at seven years of age. Arendt RE, Short EJ, Singer LT, Minnes S et al. J Dev Behav Ped 2004 Apr; 25(2): 83-90. Compared 101 exposed children to 103 unexposed as to WAIS-IQ, visual-motor, and motor at age 7. Exposed children scored significantly lower. However, home environment was a stronger predictor of these deficits. "Although prenatal cocaine exposure may confer some degree of developmental disadvantage in the visual motor domain, it frequently occurs in the context of an inadequate rearing

environment, which may be a stronger determinant than prenatal cocaine exposure of children's outcome."

The maternal lifestyle study: cognitive, motor, and behavioral outcomes of cocaine-exposed and opiate-exposed infants through three years of age. Messinger DS, Bauer CR, Das A. *Pediatrics* 2004; 113: 1677-1686. Long term multicenter prospective study gave Bayley scales to 474 cocaine babies, 50 opiate babies, 48 cocaine AND opiate babies, and 655 controls. Found that prenatal exposure did not have any measurable consequences.

Pediatric caustic ingestion and parental cocaine abuse. Massa N, Ludemann JP. *Int J Pediatr Otorh* 2004; 68: 1513-1517. A strong base is used to make freebase cocaine. This can include ammonia or KOH. This article reports the cases of two toddlers who drank ammonia and KOH and suffered first and second degree pharyngeal and esophageal burns, and tested positive for cocaine.

Editorial: We were wrong about "crack babies." Are we repeating our mistake with "meth babies?" David Lewis MD. *Medscape General Medicine* 2005; 7(4): 30. Posted 10/31/05. After almost 20 years of research, not one single condition or disorder that could be labelled "crack baby" has been identified. (citing Rose-Jacobs et al., 2002) Punitive legislation was directed at newborns and their mothers. This stigmatized them and hurt their access to proper care. Now it's "meth babies." Let's not rush to judgment. David Lewis, Professor of Community Health and Medicine, Brown University.

A 25-year retrospective review of deaths due to pediatric neglect. Knight LD and Collins KA. *Am J Forens Med P* 2005 Sep; 26(3): 221-228. See below under "Neglect." Includes a case of a chronically neglected toddler who got so hungry that he ate rock cocaine, and at autopsy had numerous rocks in his esophagus, stomach, pharynx and trachea.

Acute neonatal effects of cocaine exposure during pregnancy. Bauer CR, Langer JC, Shankaran S, Bada HS, Lester B, Wright LL, Krause-Steinrauf H, Smeriglio VL, Finnegan LP, Maza PL, Verter J. *Arch Ped Adol Med* 2005; 159(9): 824-834. A prospective multicenter randomized study comparing 717 cocaine-exposed neonates to 7442 nonexposed neonates. Cocaine-exposed neonates were born 1 week earlier and weighed 536 grams less and had HC 1.5 cm smaller ($P < .001$) CNS overstimulation was seen more in the exposed group: jitters, high-pitched cry, irritability, excessive sucking, hyperalertness, autonomic instability. Had more infections, including hepatitis, syph, and congenital HIV. No difference was found in organs by ultrasound; this observation contradicts earlier work finding organ system abnormalities. Because the symptoms were transitory, the authors consider that they may be direct intoxication effects. The more serious risk is that of **infections**, particularly sexually transmitted infections, in exposed infants.

Impact of prenatal cocaine exposure on child behavior problems through school age. Bada HS, Das A, Bauer CR, et al. *Pediatrics* 2007 Feb; 119: e348-e359.
<http://www.pediatrics.org/cgi/doi/10.1542/peds.2006-1404>

A longitudinal study that matched exposed and nonexposed children and observed them for seven years, assessing them for behavioral problems at age 3, 5, and 7 by means of the Child Behavior Checklist. Looked for internalizing, externalizing, and total behavior problems. Results: "High prenatal cocaine exposure was associated with the trajectory of internalizing, externalizing, and total behavior problems... Caregiver depression and family violence had independent negative influence on all behavior outcomes."

FOREIGN BODY

Mechanisms of unexpected death in infants and young children following foreign body ingestion. JFS 1996; 41: 438-441.

See also under "Esophagus"

DROWNING

(See also "Bath seats," below)

The Consumer Product Safety Commission (CPSC) maintains documentation on the child drown hazard with respect to household products including pools, bath seats, and containers. <http://www.cpsc.gov>

Non-accidental immersion in bathwater: another aspect of child abuse. Nixon J and Pearn J. Br Med J 1977; 1: 271-272. (have) Arose from an Australian extensive total population study of immersion accidents affecting children, which revealed to them that some BT drownings are not accidents. Doesn't give numbers. These are their homicide cases; see next below for their accident cases. They found an *unusual time of day* as a marker for these non-accidental cases. They note that an important clue is "a sense of disquiet, on the part of the clinical attendants, that all is not what it seems." "An older, solitary child, odd bathing time, and the father as caretaker in a time of emotional upheaval," as summarized of this article's findings by Lavelle et al. (infra.)

	Accid	Non-acc
Age	9-15 mos	15-30 mos
Class	IV, V	--
Parents	nl	abuser profile
Child	nl	?handicapped
Circumstances	hs routine upset	parental stress, unusual time of day

Bathtub immersion accidents involving children. Pearn J, Nixon J. Med J Aust 1977; 1: 211-213. (have) These were true accidents involving 19 children aged 9 mos to 36 months. These tended to be working-class homes with large numbers of children in which the usual household routine was temporarily broken by something such as sickness or marital strife. All of their nonaccidental immersions shared these features:

bathtub
older child
unusual time of day
child alone in the tub
sociopathologic profile of abusive parents
marital strife

Chil who were immersed four minutes or less survived; five minutes or longer died.

An investigation of socio-demographic factors surrounding childhood drowning accidents. Nixon J and Pearn J. (Aust) Soc Sci Med 1978; 12: 387-390.

Bathtub drownings: report of seven cases. Pearn JH, Brown J, Wong R., et al. Pediatrics 1979; 64: 68-70

Bathtub-related drownings in the United States, 1979-81. Budnick LD, Ross DA. Am J Publ Health 1985; 75: 630-633

Child abuse by drowning. (not necessarily bathtubs) Griest KJ and Zumwalt RE. Pediatrics 1989 Jan; 83(1): 41-46. (New Mexico) These were six strange cases from the New Mexico ME's office in which drowning was not at first acknowledged and the cause of death, and sometimes even the death itself, were concealed by the perpetrators. Two may have involved bathtubs. Findings included contusions of the lips, lacerations of the buccal mucosa, scalp hemorrhages, ocular petechiae, cerebral edema (due to post-immersion survival, wet body, contusions of the extremities, low vitreous electrolytes, waterlogged hands and feet, foreign material in the lungs, old fractures, history of contact with child protection. Says that the dx cd not hv bn made without cooperation with law enforcement. Notes that there is often post-immersion survival leading to cerebral edema and anoxic changes. Also lays out an investigative approach to fetus toilet incidents.

Their six cases:

Case 1: a 2 ½ year old boy found unr in bed, described as being “wet.” Drug abusing parents. No one knew how he got dead. Autopsy showed contusions of the lips, lacerations of the buccal mucosa, and frontal scalp hematomas. Edematous brain and lungs.

Case 2: a 3 y o girl found on the couch when a concerned neighbor called params. Her hands and feet were waterlogged. Prominent oculofacial petechiae. Contusions of the lips and cheeks. Cerebral edema. Vitreous Na 119, Cl 105. This turned out to have been caused not by immersion but by the mother, a religious fanatic, who cleaned out the devil by forcibly pouring water down the child’s throat.

Case 3: A 2 y o boy brought to ER comatose by his father. Father told police child had fallen off the monkey bars earlier in the day. Survived two days on a ventilator. Topsy showed numerous scars and contusions, pulmonary edema with foamy macrophages. Father confessed to holding the child’s head under water in the tub.

Case 4: Newborn found in the toilet. 35 weeks. Placental hematoma. Partially inflated lungs with inhaled vegetable matter. “The position of the infant in the toilet was not consistent with delivery into the toilet. The mother was charged with child abuse.

Case 5: a 3 month old girl recovered from a shallow grave. Topsy sh edema of the lungs and brain + healing fractures. Lung sh numerous macrophages and giant cells, + vegetable matter and spores in the lungs. The boyfriend had a police record in another state for attempted drowning of a child in the same hot spring.

Case 6: a 9 mo boy found unr in BT by mother. He had been left in the tub with his 2 y o brother for 15 minutes while his mother went to get some clothes for the chil. The params were not called for another 20 minutes. Topsy: lungs and brain were edematous, water in the small intestine, small abrasion-contusions of the forehead, lung showing aqueous emphysema. The mother was charged with child neglect.

Describe “the postmortem pulmonary features of the postimmersion syndrome:” widespread intraalveolar infiltrates of polys and edema fluid. Cerebral edema indicates a delay between immersion and death. If non-bathtub, will have organic material in the lungs.

Compares these cases to those of Nixon & Pearn, 1977(above), as having “described several features that they believed helped to differentiate accidental from nonaccidental immersion.”

Ten year study of pediatric drownings and near-drownings in King County, Washington. Quan L, Gore EJ, Wentz K et al. Pediatrics 1989; 83: 1035-1040. Found that inadequate supervision was the common factor. See AAP Technical Report, 2003, below.

Safety practices and living conditions of low-income urban families. Santer LJ and Stocking CB. Pediatrics 1991; 88: 1112-1118

Death during immersion in water in childhood. Smith NM, Byard RW, Bourne AJ. Am J Forens Med P 1991 Sep; 12(3): 219-221. Reviewed autopsy records of 58 child drownings over 27 years. Found that in six cases, “careful examination ... suggested a more complex antemortem sequence of events. Four of these had epilepsy. One died of an AVM. One 11 year old boy had a witnessed collapse in the public pool from hypoplasia of the right coronary artery. The authors suggest a high index of suspicion, not only for non-natural death, but also for natural death from preexisting conditions.

Submersion injuries in children younger than 5 years in urban Utah. Jenson LR, Williams SD, Thurman DJ, Keller PA. West Med J 1992; 157: 641-644. Confirms Pearn’s pattern of typical features of accidental, with a child being left alone or with a sibling in the tub, often for a brief interval.

AAP Policy Statement on Child Drownings. Pediatrics 1993 Aug; 92(2): 292-294, <http://www.aap.org/policy/04482.html> Does not refer to the negligence/ neglect aspect. Children should never be left alone, even for a moment. Pool fencing the only way for pools. Pool drownings are class-correlated. This statement did not have any input from the child abuse & neglect community.

When is childhood drowning neglect? Ken Feldman. Child Abuse & Neglect 1993; 17: 329-336. 95 admissions for near-drowning at UW. In 28% the clinical staff made a social work referral (suspected neglect.)

Accidents and child abuse in bathtub submersions. Kemp AM, Mott AM and Sibert JR. Arch Dis Child 1994; 70: 435-438. (Scotland) 44 BT drownings. They divide them into

Accidental	28
Epilepsy-related	4
Nonaccidental	2
Intentional	10

The intentional cases were characterized by the usual indicia of child abuse: delayed rescue, inconsistent or varying history, previous or subsequent history of abuse, age outside the usual span for tub accidents of 8 – 24 months, and unexplained injuries. Also there was a maternal history of mental illness or lactation/postpartum depression. The "nonaccidental," (i.e. negligent) drownings were associated with the children being left with an unsuitable caregiver. Also delayed discovery. The accidental ones were 8 – 15 months who cannot right themselves once they slip down in the water. Sanders & Cobley, 2005 -- see under "Epidemiology" -- cite this article for the proposition that there were no cases of accidental bathtub drowning over the age of 18 months, and that in that age range all the cases were due either to epilepsy or abuse.

Ten year review of pediatric bathtub near-drowning: evaluation for child abuse and neglect. Lavelle JM, Shaw KN, Seidl T, Ludwig S. Ann Emerg Med 1995 Mar; 25(3): 344-348. (Philadelphia) 21 patients treated for near-drowning in BTs over a ten year period, ages 4 mos to 6 years. 67% of these were considered suspicious for abuse or neglect, where the criteria for suspicion were (a) history inconsistent with the child's developmental stage; (b) multiple histories for the event; (c) severe neglect or other injuries – bruises, fractures, RH; (d) previous child abuse reports – 25%; (e) psychiatric history of the caregiver; (f) concerns noted in the social work note.

Intentional drowning	29%
Inconsistent or varying history	63%
Physical abuse or severe neglect	38%

"An older, solitary child, an odd bathing time, and the father as caretaker in a time of emotional upheaval."
 "The overwhelming majority of these injuries occurred in the home, at the peak age fo child abuse (less than 3 years,) with 505 of previous cases having a history of abuse. In our series, 25% had previous involvement with child protective services."

Death in the bathtub involving children. Schmidt P and Madea B. FSI 1995; 72(2): 147-155. A German study of 215 total deaths in the bathtub included 12 children aged 9 mos to 13 years. Seven children under four were all accidentals. Five were toddlers left unattended or with juvenile caregiver. Five children four or over were all nondrowning deaths (scalds, SDH, Hurler's syndrome, sz dso.)

Inflicted submersion in childhood. Gillenwater JM, Quan L, Feldman KW. Arch Pediatr Adol Med 1996 Mar; 150(3): 298-303. King County ME's Office reviewed 205 submersions, including 34 BT subms, of chil under 19. Of these subms, 8% were judged to be inflicted (16 chil), due to the presence of (a) "objective signs of abuse" (i.e. physical injuries); or (b) history inconsistent with child's stage of devel or physical findings. The inflicteds were young (avg age 2.1 yrs) and tended to be the youngest of several siblings. 56% of the inflicteds were in BT; and 26% of all BT subms were inflicted (9/34.) Criteria used for inflicted:

- A. Definitely inflicted
 1. Confession; 2. Eyewitness; 3. CPS or court confirmed; 4. Coroner confirmed

5. Multiple fractures; 6. Physical injuries c/w abuse; 7. History inconsistent with the child's developmental stage
- B. Probably inflicted
 1. Historical inconsistencies: (a) Main caregiver changed history; (b) Other family members gave a different history; (c) ...
 2. Delayed rescue
 3. Professionals suspected abuse at the outset
 4. Previous history of abuse
 5. Suspicious findings on physical examination but not diagnostic of abuse

As far as ages, the inflicted in bathtubs were avg age 1.5 years, the accidentals 1.1 years, no difference. There were no inflicted BT drownings over 4 years of age. There were accidental BT drownings in this older age group.

The role of bathtub seats and rings in infant drowning deaths. Rauchschwalbe R, Brenner RA, and Smith GS. Pediatrics electronic edition 1997; 100(4). Pediatrics.org/cgi/content/full/100/4e1.

Identification of a family with inherited long QT syndrome after a pediatric near drowning. Ackerman MJ, Porter CJ. Pediatrics 1998 Feb; 101(2): 306-308. This 10 year old boy was rescued in torsade de pointes, converted to nsr with a prolonged QTc of .56 sec (.46 is the top end of normal). He had a recurrence of VT four days later while sleeping in his hospital bed. Then it turned out that he had blacked out on two previous occasions, and other relatives had a history of fainting or falling. The family was tested, and found to have it. Meanwhile, a whole literature has developed on swimming-induced arrhythmias, and there is a web site called <http://www.sads.org> for Sudden Arrhythmia Death Syndrome.

Drowning without aspiration: is this an appropriate diagnosis? Modell JH, Bellefleur M, Davis JH. JFS 1999 Nov; 44(6): 1119-1123. There is no such thing as a dry drowning. The original articles on which this concept was based are in error.

Where children drown, United States, 1995. Brenner RA, Trumble AC, Smith GS, Kessler EP, Overpeck MD. Pediatrics 2001; 108: 850-89. Ages 0-20 years, a death certificate study. For infants:

Tubs	55%
Buckets	12%

Shared bathing and drowning in infants and young children. Byard RW, deKoning C, Blackbourne B, nadeau J, Krous HF. J Paediatr Child Health 2001 Dec; 37(6): 542-544. Search of autopsy databases in Australia and San Diego. Found 17 cases of children under 2 who drowned in a bathtub while in bath with another child. Age range 8-22 months (avg =11.8 mos). The surviving children were older (age range 19-48 mos, avg 30.4 mos). The survivors averaged 18 months older than the victims. "In every case, the children had been left unsupervised for variable periods of time." Shared-bathing drowning represented 22% to 58% of bathtub drownings in the under-2 age group, a very significant component. But the authors state that their findings do NOT prove that sharing *per se* increases the risk of drowning. What they do conclude is that "[S]hared bathing for young children and infants may only be acceptable if adult supervision is constant."

Policy Statement: Prevention of Drowning in Infants, Children, and Adolescents. AAP Committee on Injury, Violence, and Poison Prevention. Pediatrics 2003 Aug; 112(2): 437-439.

1. "Parents and caregivers need to be advised that they should never—even for a moment—leave children alone or in the care of another young child while in bathtubs, pools, spas, or wading pools or near irrigation ditches or other open standing water. They should also be reminded that infant bath seats or supporting rings are not a substitute for adult supervision.¹¹ They should remove all water from containers, such as pails and 5-gallon **buckets**, immediately after use. To prevent drowning in toilets, young children should not be left alone in the bathroom, and unsupervised access to the bathroom should be prevented.

2. Whenever infants and toddlers are in or around water, be it at their own home, the home of a neighbor, a party, or elsewhere, a supervising adult should be within an arm's length providing "touch supervision." The attention of the supervising adult should be focused on the child, and the adult should not be engaged in other distracting activities, such as talking on the telephone, socializing, or tending to household chores.
3. If a home has a residential swimming pool, it should be surrounded by a fence that prevents direct access to the pool from the house. Rigid, motorized pool covers, pool alarms, and other protective devices, which may offer some protection if used appropriately and consistently, are not a substitute for 4-sided fencing.
4. Children are generally not developmentally ready for formal swimming lessons until after their fourth birthday..."

Reported level of supervision of young children while in the bathtub. Simon HK, Tamura T, Colton K. *Ambul Pediatr.* 2003(3) :106 –108

Inadequate supervision as a cause of injury deaths among young children in Alaska and Louisiana. Landen MG, Bauer U, Kohn M. *Pediatrics* 2003; 111: 328-331.

Office-based counseling for unintentional injury prevention. Gardner W, MD, and the Committee on Injury, Violence, and Poison Prevention. *Pediatrics* 2007 Jan; 119(1): 202-206.

Technical report: Prevention of drowning in infants, children, and adolescents. Ruth A. Brenner, MD, MPH and the AAP Committee on Injury, Violence, and Poison Prevention. *Pediatrics* 2003 Aug; 112(2): 440-445. Here is the text of the abstract: "Drowning is a leading cause of injury-related death in children. In 2000, more than 1400 US children younger than 20 years drowned. Most (91%) of these deaths were unintentional and were not related to boating. For each drowning death, it is estimated that at least 1 to 4 children suffer a serious nonfatal submersion event, many of which leave children with permanent disabilities. Environmental strategies, such as installation of 4-sided fences around swimming pools, and behavioral strategies, such as increased supervision of children while around water, are needed to prevent these tragedies." Covers sociodemographic factors, location and circumstances, lapses in adult supervision, alcohol, swimming ability, underlying medical conditions, fences, pool covers, etc. As far as lapses in adult supervision, cites the ten-year King County study (Quan et al., 1989) as finding that "inadequate supervision" was the most common factor. (See also the CPSC findings under "Bath seats," below.) "In most cases, the adult reports leaving the child for a short time to answer the phone or attend to household chores."

Spectrum and frequency of cardiac channel defects in swimming-triggered arrhythmia syndromes. Choi G, Kopplin LJ, Tester DJ, Will ML, Haglund C, Ackerman MJ. *Circulation* 2004; 110: 2119-2124.

Children may be able to climb into bathtubs earlier than previously thought. Allasio D and Fischer H. *Pediatrics* 2005 May; 115: 1419-1421. These guys were actually studying the issue of scalds. They set up an (empty) bathtub with toy boats in it, and placed 176 toddlers aged 10 months to 18 months outside the tub. 35% successfully climbed into the bathtub, including one ten month-old who couldn't even walk.

Pediatric drowning: A 20-year review of autopsied cases: I. Demographic features. Somers G, Chiasson DA, Smith CR. *Am J Forens Med P* 2005 Dec; 26(4): 316-319. From the Hospital for Sick Children, Toronto. [The authors have a forthcoming article on III: Bathtubs coming, see below.] Drowning is the second most common cause of accidental death in infancy and childhood, behind auto accidents. The authors found 81 drownings among the 2,422 medicolegal autopsies performed at the hospital. 18 of these were bathtubs, and were excluded from this research for treatment in a separate article (see above). That leaves 63 nonbathtub cases, 45 male and 18 female. The average age of the males was 5 years 9 months; the average age of the females was 4 years 4 months. These nonbathtub cases were heavily concentrated in

the spring and summer months. Thirty drowned in lakes and rivers, 28 in pools. also 2 hot tubs, 1 bucket, and 1 ditch. The open-water children were older, the pools younger.

This article is reviewed by Ken Feldman in the Spring 2006 issue of the *Quarterly*. He says that it is limited by not being population-based and not including any autopsy data. But it continues already-known epidemiologic trends: boys drown more, but more girls are starting to drown in pools. Resuscitation rarely does any good in terms of recovering brain function.

Pediatric drowning: A 20-year review of autopsied cases: II. Pathologic features. Somers GR, Chiasson DA, Smith Cr. *Am J Forens Med P* 2006 Mar; 27(1): 20-24. The incidence of frothy exudate, pleural effusion, and increased lung weight was 43%, 36%, and 80% respectively. Resuscitation tended to abolish the frothy exudate, as did long postmortem interval.

When is lack of supervision neglect? (Clinical Report). Kent P. Hymel MD and the AAP Committee on Child Abuse and Neglect. *Pediatrics* 2006. <http://www.pediatrics.org/cgi/doi/10.1542/peds.2006-1780>. See below under "Neglect."

BATH SEATS (TUB SEATS AND RINGS)

CPSC votes against rulemaking for baby bath seats. Press release, June 15, 1994. The commission voted 2-1... today against initiating formal rulemaking proceedings on baby bath seats, which are associated with 14 deaths and 7 near-drownings since 1983 of babies age 6 to 15 months. The Commission believed that under the Federal Hazardous Substance Act, the design and manufacture of these products do not present a mechanical hazard or an unreasonable risk of injury to consumers. The Commission also voted (2-1) ... to work with industry to initiate a public information campaign focusing on the risks taken by parents and other caregivers who leave children unattended in bathtubs. Vice Chairman Mary Sheila Gall, noting that parents and caregivers left the victims unattended for lengthy periods of time, stated, "It is clear that the irresponsible actions of those entrusted with caring for these children have, almost without exception, caused their deaths... Commissioner Jaqueline Jones-Smith said, "Bathtubs and unattended babies are a deadly combination. No product, no device, no convenience of any kind can substitute for the physical presence of a parent or caregiver. The incidents associated with bathtub seats and rings that have occurred were all tragic and preventable events. But these were all human tragedies and not product failures. These bath seats and rings contained no manufacturing or design defects that constituted a mechanical hazard." Chairman Ann Brown said, "I'm disappointed with the Commission's decision on baby bath seats, which encourage dangerous consumer behavior by instilling a **false sense of security** in a parent, who would normally never leave a baby alone in water -- not even for a second..." ...With 1.4 million baby bath seats in use today, the CPSC urges parents and caregivers who are using baby bath seats to:

Never leave a baby alone in the water for even a second. Keep baby in arms reach.

Never use the baby bath seat in a non-skid, slip-resistant bathtub.

Check to see that the suction cups are securely attached to the bath seat and tub surface.

The role of bathtub seats and rings in infant drowning deaths. Rauchschiwalbe R, Brenner RA, Smith GS. *Pediatrics* 1997 Oct; 100(4): e1. From the abstract: "Thirty-two drowning deaths involving **bath seats/rings** were identified and investigated by the Consumer Product Safety Commission over a 13-year period. The majority of deaths (84%) occurred from 1991-1995, with more than 50% occurring in the 2 most recent years. The victims' ages at the time of the incident ranged from 5 to 15 months with a mean and median age of 8 months. In more than 90% of incidents there was a reported lapse in adult supervision, with a mean reported lapse of 6 minutes and a median lapse of 4 minutes. Focus groups with parents found that while making **bathing** somewhat easier, **bath seats/rings** are useful for a relatively short time period, as the child rapidly outgrows the product. They also suggested that care givers are more likely to leave a child unattended in the tub if one of these products is in use. **Conclusion.** **Bath seats/rings** are associated with an increasing number of reported infant drowning deaths. The use of such products may increase the risk of drowning among infants **by increasing the likelihood that an infant will be left alone in the tub.**

However, in the absence of exposure data in a suitable comparison group it is difficult to assess the overall risk inherent in their use. Educational efforts reinforcing the need for continuous adult supervision of infants and children around all bodies of water should now also include a reminder that **bath seats/rings** are not safety items and are not a substitute for adult supervision. Infants and toddlers should never be left in the **bath**tub unsupervised, even for brief moments.”

Consumer Product Safety Commission: Petition Requesting Banning of Baby Bath Seats. 65 CFR 163 (August 16, 2000). The Commission has received correspondence from the Consumer Federation of America and other consumer groups requesting that the Commission issue a rule banning baby bath seats and bath rings. The petitioners assert that these products pose an unreasonable risk of injury primarily by giving parents and other caregivers a false sense of security that children using the product will be safe in the bathtub. They argue that recent research indicates that parents using bath seats are more likely to engage in “risk-taking behavior,” such as leaving the infant alone briefly and using more water in the bathtub, than caregivers who do not use bath seats. The petitioners state that, to date, 66 incidents of drowning and 37 reports of near-drowning have been identified...

Consumer Product Safety Commission: Public Meeting concerning bath seat rulemaking. (Announcement) 68 CFR 113 (June 3, 2003). ...will conduct a public meeting on July 28, 2003 ... The staff recommends a stability requirement to address the hazard of bath seats tipping over while in use. The staff has identified 30 fatalities and 80 non-fatal incidents or complaints involving bath seats tipping over that were reported from January 1983 through December 2002. The staff recommends a stability requirement that is essentially the same as the stability requirement in the ASTM voluntary standard... The staff has identified 3 deaths and 17 non-fatal incidents of complaints involving children who were submerged or entrapped in bath seats that were reported from January 1983 through December 2002. To address this hazard, the staff recommends a performance requirement specifying that the bath seat’s leg openings not allow passage of probes that represent the shoulder and torso of an infant. This req is identical to one that ASTM approved in March 2003... The staff has identified 19 fatalities and 13 non-fatal incidents or complaints involving children coming out of bath seats... The staff recommends a revised warning label to better alert caregivers to the danger of leaving a child alone in a bath seat.

Prevention of drowning in infants, children, and adolescents. AAP Committee on Injury, Violence, and Poison Prevention. Pediatrics 2003; 112: 437-439.

Reported level of supervision of young children while in the bathtub. Ambulatory Pediatrics 2003; 3: 106-108.

Inadequate supervision as a cause of injury deaths among young children in Alaska and Louisiana. Landen MG, Bauer U, Kohn M. Pediatrics 2003; 111: 328-331.

Office-based counseling for unintentional injury prevention. Gardner WH, MD, and the Committee on Injury, Violence, and Poison Prevention. Pediatrics 2007 Jan; 119(1): 202-206. “Drowning prevention: Because very young infants drown most commonly in **bath**tubs and buckets while unsupervised, advise parents never to leave infants or young children in the **bath**tub or around other bodies of water without constant adult supervision, and advise them to empty and properly store buckets immediately after use.¹⁷⁻¹⁹ Parents should be reminded that infant **bath seats** or supporting rings are not a substitute for adult supervision.” Citing the Committee’s report, “Prevention of drowning in infants, children and adolescents, 2003; Simon et al., Reported level of supervision, 2003; Landen et al., Inadequate supervision, 2003.

BUCKETS

Policy Statement: Prevention of Drowning in Infants, Children, and Adolescents. AAP Committee on Injury, Violence, and Poison Prevention. Pediatrics 2003 Aug; 112(2): 437-439. (See above under “drowning.”) Parents must remove the water from all buckets after use.

Office-based counseling for unintentional injury prevention. H. Garry Gardner and the AAP Committee on Injury, Violence, and Poison Prevention. Pediatrics 2007 Jan; 119(1): 202-206.

DENTAL & FACE (see also "Bruises" and "CPR" and "SIDS v. suffocation")
See ABFO at <http://www.ABFO.org>

Reece & Ludwig, 2d ed. (2001):

"The principal intraoral injuries of child abuse include missing and fractured teeth, oral contusions, oral lacerations, jaw fractures, and oral burns." p. 114. Just as with the face generally (p. 109), the authors differentiate between infancy and older. The analysis is different. Any facial bruising in infants is extremely unusual, while in older it can be 25% of the bruises observed normally in child over 2.

Tooth fractures can be accidental. p. 116.

Frenular tears can occur from learning to walk. p. 117.

Facial injuries associated with the battered child syndrome. Tate. British Journal of Oral Surgery 1984; 9: 41

Donly KJ and Nowak AJ, Maxillofacial, Neck, and Dental Lesions of Child Abuse. In: Reece RM, Child Abuse: Medical Diagnosis and Management, 1994.

Physical manifestations of child abuse to the head, face and mouth: a hospital survey.
Jessee SA. J Dent Child 1995; 62: 245-9

Patterns of facial resuscitation injury in infancy. Kaplan JA and Fossum RM. Am J Forens Med Pathol 1994; 15(3): 187-191 (have) Nine cases described. Fingertips under the chin, mask ring & facial abrasions, perinasal fingertip marks (?bruises), nasal abrasions re benzoin

Sexual abuse of the oral cavity in children. Heitzler GD, Cranin AN, Gallo L. J Mich Dent Assoc 1994 Jul; 76(6): 28-30

Otorhinolaryngologic Manifestations. In: Levin AV and Sheridan MS (eds.), Munchhausen Syndrome by Proxy: Issues in Diagnosis and Treatment. New York: Lexington Books, 1995, pp. 219-230.

Nelson's Pediatrics, 15th edition. Chapter 38.2 (W.B.Saunders, 1996): Nonorganic failure to thrive (NOFTT). "A rejecting mother often feeds her baby with anger and unnecessary force. This may result in a torn frenulum and aversion to feeding." (p. 119).

Manual of Forensic Odontology, 3d ed. Montpelier, Vermont: Printing Specialists, 1997.

Dental neglect: risk factors as determinants of dental neglect in children. Jessee SA. J Dent Children 1998; 65: 17-20. Like others, he says that neglect is not diagnosed until it recurs after the parents have been educated and systemic barriers to their providing care have been removed. Cf. Dr. 's presentation at the 2000 San Diego Conference in the same vein.

The dental practitioner's role in protecting children from abuse: 2. The orofacial signs of abuse. Murphy JM and Welbury RR. Br Dent J 1998; 184: 61-65

Oral and dental aspects of child abuse and neglect. Joint policy statement of the AAP and The AAPD. Pediatrics 1999 Aug; 104(2 part 1): 348-350. (have) Includes oral gonorrhea, bite marks, definition of dental neglect.

A profile of the oro-facial injuries in child physical abuse at a children's hospital. Naidoo S. Child Abuse & Neglect 2000; 24: 521-534. Cape Town. 300 physically abused children over a five year period. 67% had injuries to the head, face, neck or mouth. 41% face. Only 11% intra-oral, but may be higher because none of these pts were seen by a dentist or an oral-maxillofacial surgeon. The authors suggest careful investigation of any facial bruise that isn't over a bony prominence.

Orofacial considerations for pediatric, adult, and elderly victims of abuse. Fenton SJ, Bouquot JE, Unkel JH. Emerg Med Clin N Amer 2000 Aug; 18(3): 601-617.

The dentist's role in reporting suspected child abuse and neglect. Rupp RP. Gen Dent 2000 May-Jun; 48(3): 340-342. Lists criteria useful in det whether an injury is accidental or intentional.

Dentistry's role in the recognition and reporting of domestic violence, abuse, and neglect. Senn DR, McDowell JD, Alder ME. Dent Clin N Amer 2001 Apr; 45(2): 343-363. DV victims often have signs of injury that are readily visible to dentists. Illustrations, and reporting reqs for every state in the US.

Oral lesions due to child abuse: identification of the problem and role of the professional. Serrano Garcia SI et al. (Univ of Valencia) Med Oral 2001 Aug-Oct; 6(4): 276-289.

California Medical Protocol for Examination of Sexual Assault and Child Sexual Abuse Victims, July 2002. <http://www.ojcp.ca.gov/>

American Board of Forensic Odontology. <http://www.abfo.org>

Death by overlaying and wedging: a 15-year retrospective study. Collins KA. Am J Forens Med P 2001 Jun; 22(2): 155-159. Found zero oral-intraoral lesions in 32 cases of child/infant accidental suffocations and hangings. States that "[W]hen a child does have positive findings of multiple contusions, abrasions, or oral-intraoral lesions, these probably don't result from overlaying, wedging, or other accidental suffocation... Furthermore, no oral or intraoral lesions were identified in any of the cases. The pressure required to create such traumatic and often multiple injuries is greater than that pressure from overlaying or wedging. The paucity of physical findings in cases of accidental asphyxia such as those scenarios described here and reported previously, should impress upon investigators that multiple abrasions, contusions, or oral-intraoral lesions are unlikely to be the result of accidental asphyxia." (p. 158).

When is an abnormal frenulum a sign of child abuse? Chan L, Hodes D. Arch Dis Chil 2004 Mar; 89(3): 277. (UK)

Clinical guideline on oral and dental aspects of child abuse and neglect. American Academy of Pediatric Dentistry. Pediatric Dentistry 2004; 26(7): 63-66. Reviews the oral and dental aspects of physical and sexual abuse as well as **dental neglect**. Including bite marks and oral STD's.

Blinding keratoconjunctivitis and child abuse. Ong T, Hodgkins P, Marsh C, Taylor D. Am J Ophth 2005 Jan; 139(1): 190-191. Reports two infants with inferior-half keratoconjunctivitis due to inflicted corneal injury. Unexplained keratoconjunctivitis, particularly in the lower half, should alert to the possibility of child abuse. See above under "DDX."

Torn frenulum and non-accidental injury in children: best evidence topic report. Teece S, Crawford I. (Manchester) Emerg Med J 2005 Feb; 22(2): 125. Is a torn frenulum diagnostic of non-accidental injury? A meta-analysis of 104 papers found that "none presented any evidence to answer the question."

Oral and dental aspects of child abuse and neglect. Nancy Kellogg, M.D. and the American Academy of Pediatrics Committee on Child Abuse and Neglect. Pediatrics 2005 Dec; 116(6): 1565-1568. Covers the

following topics: physical abuse, sexual abuse, bite marks, dental neglect. As to physical abuse, notes that over half of all cases of child physical abuse include injuries to the head, face, and neck. All victims should be examined both for oral trauma and for caries, gingivitis, and other intraoral conditions. The mouth is a center of physical abuse because of its role in both communication and feeding. Injury by the bottle in force feeding, or by eating utensils in force feeding. As to types of trauma, examine for contusions, scalds, lacerations, broken teeth, frenulum, bony fractures. The lips were the most common site of injury in the study by Naidoo, 2000, followed by the oral mucosa, teeth, gingiva, and tongue. Previous trauma may cause discolored teeth due to pulpal necrosis. Pharyngeal injuries or abscesses may be caused by factitious disorder/ Munchhausen's to simulate disease. But accidental injuries are common. As to sexual abuse, the mouth is a frequent target but rarely injured. See the AAP *Guidelines in the Eval of Sexual Abuse of Children*. Discusses oral/ pharyngeal gonorrhea which is rare. HPV warts, but watch out for vertical transmission and accidental manual transmission; oral HPV is NOT diagnostic of sexual abuse. Palatal injuries or petechiae, particularly at the junction of the hard and soft palate, if not explained, may be due to forced oral sex. As to bite marks, animal bites tear the flesh while human bites compress it. An intercanine distance of greater than 3 cm is suspicious for adult human bite. Discusses forensic photography and casting and saliva/trace evidence collection and refers to the ABFO (see above). As to dental neglect, gives the definition of the AAPD (see Dr Reece's review, below). Notes that failure to seek care may be caused by poverty, ignorance, social isolation, and lack of availability of dental services.

This article is reviewed by Dr Reece in the Spring 2006 issue of the *Quarterly*. He notes that dental neglect is "the **wilful failure of parent or guardian to seek and follow through with treatment** necessary to ensure a level of oral health essential for adequate function and freedom from pain and infection." This means that the parent **has to have been alerted** by a health care professional to the need for dental intervention. Dr Reece points us to the PANDA Prevent Abuse and Neglect through Dental Awareness Hotline 501-661-2595, an important resource on dental abuse & neglect.

Early childhood caries. Dr. Leonard B. Smith, 2005. <http://www.drgoodtooth.com> Early childhood caries (ECC) is rampant throughout North America. It's the single most common chronic childhood disease. It leads to SBS, because these children are in pain and they can only communicate by crying. Cites MMWR 2005; 54: 1-44 for epidemiology. Incidence increased 15% 2001-2005. Gives color photos. Tells us to look BEHIND the teeth, not just in front. Begins at 14 mos. "The caries process is infective and invasive. The pulpal tissue becomes acutely inflamed which causes acute pain. The pulpal tissue eventually becomes necrotic resulting in periapical pathology." Cites a Surgeon General's report on the "silent epidemic." Gives a link to the American Academy of Pediatric Dentistry <http://www.aapd.org> and also Nancy Kellogg's AAP report.

Domestic violence: a complex health care issue for dentistry today. Kenney JP. FSI 2006; 159S:S121-S125. According to Dr K Ganda's review in the Autumn 2006 issue of the *Quarterly*, 94% of DV and elder ab vics present with injuries to the head and face, and therefore the dentists is often the first to see them. The same is true for child abuse. Often the history is vague or there is a history of repeated "accidents." Discusses different findings. Dr Ganda says this is an important, well-organized paper.

ESOPHAGUS

Esophageal foreign bodies as child abuse: potential fatal mechanisms. Kurt B. Nolte, M.D. Am J forens Med P 1993 Dec; 14(4): 323-326. Case report of a five month old female with several coins recovered by the surgeons from her esophagus. One month later she was found dead in her crib with three more coins in her esophagus. Had multiple contusions, healed fractures, fat emboli, and aspirated foreign material in the lungs. As fatal mechanisms considered (aside from the fat emboli), vagal stim, spir after esoph obstruc, compresion of trachea or heart, airway occlusion by the introducing finger.

Inflicted esophageal perforation. Marzaria S, Walton JM and MacMilland A. J Pediatr Surg 1998; 33: 871-873. Case of a 2 1/2 year old girl with torn frenulum, burns, bruises fractured humerus and pelvis said

to have occurred by falling down stairs. Found to have prevertebral air on lateral C-spine x-rays, found to have a rupture of the proximal esophagus with a prevertebral abscess.

Esophageal perforation preceding fatal closed head injury in a child abuse case. Pramuk LA, Sirotiak A, Friedman NR. Int J Otorhinol 2004; 68: 831-835. From Dr Reece's review in the *Quarterly* for Autumn 2004, we find that this is a case report of a 3 month old who died of SBS and having at autopsy iron deposits in the optic nerves, retinas and skull. The authors found out that at age 15 days this infant had been brought to the ER by her father because of blood coming from her nose. The father said he fell with the infant. They discovered a tear of the posterior hypopharynx extending down to the level of the cricopharyngeus. The father did not have an explanation for this. The tear was surgically repaired. No other injuries were found and CT scans and skeletal survey were negative. A suspected child abuse report was filed, but no caseworker was ever assigned by the County. The father did not bring the child in for any of her followup appointments. Then three months later she died of SBS. As noted, had hemosiderin.

Pediatric caustic ingestion and parental cocaine abuse. Massa N, Ludemann JP. Int J Pediatr Otorh 2004; 68: 1513-1517. A strong base is used to make freebase cocaine. This can include ammonia or KOH. This article reports the cases of two toddlers who drank ammonia and KOH and suffered first and second degree pharyngeal and esophageal burns, and tested positive for cocaine.

STRANGULATION AND NECK see also Positional see also CPR for larynx see also SIDS versus Suffoc

Strangulation in child abuse: CT diagnosis. Bird CR, McMahan JR, Gilles FH, Senac MO, Apthorp JS. Radiology 1987; 163(2): 373-375 Argues that unilateral cerebral swelling with loss of gray-white differentiation was evidence for strangulation. Presents three cases: Case 1 fatal, child presented with sz and resp arrest from a "fall" occurring that day, expired. Case 2 a 7 month old presenting with sz and posturing of the right arm and leg and facial bruising with RH, no history of trauma, had brain swelling possibly unilateral, and ended up with right-sided cerebral atrophy. Case 3 a 7 month old presented with sz after a reported fall from a standing position onto a carpeted floor, semicomatose with RH and bruises. Cindy Christian comments that this argument is not very convincing; that they often see unilateral severe swelling and loss of g/w diff on the side of a SDH. Unilateral swelling does not prove strangulation. (personal communication, SIGCA, 7/14/98)

Hanging deaths in children. Cooke CT, Cadden GA, Hilton JMN. Am J Med Sci 1989; 10: 98-104. Cited by Kim Collins, 2003, for the proposition that "previous researchers have found few hanged or strangled infants to have intrathoracic petechiae."

Case report -- child abuse -- necklace calcification -- a sign of strangulation. Carty H. Br J Radiol 1993 Dec; 66(792): 1186-1188. Cf. Comment in Br J Radiol 1994 Jul; 67(799): 743-744. Radiographic finding of "necklace calcification" in the neck of an infant with rib fractures and a metaphyseal fracture and a confession of shaking. The necklace calcification is likely to represent fat necrosis as a result of strangulation.

Pathological findings in hanging and wedging deaths in infants and young children. Lynette Moore and Roger W. Byard. Am J Forens Med Path 1993; 14(4): 296-302 (have). Analyzed 14 cases aged from 6m to 3y. One car seat. One pram, one curtain cord, 11 cribs, several clothing-hangings on crib. Facial petechiae in all hangings, but only one wedging. Byard & Krous discuss facial petechiae as being seen only in hangings and mechanical asphyxia at pp. 230-231 of their book, *Sudden Infant Death Syndrome: Problems, Progress & Possibilities* (2001) (see under "Petechiae," above).

Potentially dangerous sleeping environments and accidental asphyxia in infancy and early childhood. Byard WR, Beal S, Bourne AJ. Arch Dis Child 1994; 71: 497-500. (have) Review of 30 cases of accidental asphyxia from loose restrainers, infant seats, chairs, curtain cords, wedging on beds and walls,

facial occlusion by bedding. Excludes overlaying. “As the pathological findings were on occasion identical to those that are typically found in SIDS, adequate death scene examination was vital ...” Two infants got wedged into the back of a couch while cosleeping with adult. Two slipped down in child restraint seats that were not properly buckled. See case 01-7326.

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 lxx. 7 had strap m hems, 18 had Lx mucosal contus etc. Inj mimic strang Be cautious.

Infant mechanical suffocation deaths in the United States, 1980-1997. Drago DA, Dannenberg AL. Pediatrics 1999; 103: e59.

A triad of laryngeal hemorrhages in strangulation: a report of eight cases. JFS 2000; 45: 614

Asphyxial deaths and petechiae: a review. Ely SF and Hirsch CS. JFS 2000; 45: 1274-1277. Pets are caused by impaired venous drainage, a purely mechanical event. Forms of asphyxia that don't involve compression rarely involve pets.

Subconjunctival haemorrhage and respiratory distress. Chiu C-H et al. Lancet 2001 Sept 1; 358: 724. Thought to be from adenovirus infection.

Strangulation injury: a fatal form of child abuse. Jain V, Ray M, Singhi S. Ind J Pediatr 2001 Jun; 68(6): 571-572. Case report with hospital survival and death from anoxic encephalopathy.

Diffuse unilateral hemorrhagic retinopathy associated with accidental perinatal strangulation: a clinicopathologic report. Shaikh S, Fishman ML, Gaynon M, Alcorn D. Retina 2001; 21: 252-255. A severely asphyxiated 35 week newborn with a tight nuchal cord, possible sepsis, and renal anomalies and R IVH had diffuse RH and ONSH in the R eye only. The authors attribute to strangulation but do not explain why this would be unilateral. [I suggest more likely due to the IVH. -JKR]

Unexpected death due to infectious mononucleosis. Byard RW. J Forens Sci 2002 Jan; 47(1): 202-204. Respiratory obstruction by swollen tonsils in a 14 year old boy.

Child abuse and the otolaryngologist, parts I and II. Crouse CD, Faust RA. Otolaryngol Head Neck Surg 2003 Mar; 128(3): 305-310, 311-317. Says child abuse incidence in US is about 1 million chil per year, with a greater than 1% mortality. Nearly 75% of all abuse have inj of the head and neck. Goes on to discuss manifestations in the head & neck with particular ref to the role of the otolaryngologist. Also discussed conditions that may be mistaken for abuse.

Fatal blunt trauma of the larynx in a child. Durak D, Fedakar R, Eren B et al. (Turkey) JFS 2005 Sep; 50(5): 1199-1200. This 11 year-old girl was climbing on some monkey bars and she fell off and hit her throat on one of the lower bars. She then stood up and walked a short distance before collapsing and dying. Autopsy showed a bruise on the neck and laryngeal hematomas with fracture of the larynx. The cause of death was mechanical asphyxia. External examination at autopsy showed violet bruises on both sides of the anterior neck, subcutaneous emphysema, fracture of the right lamina of the thyroid cart with edema. In their discussion, the authors note that blunt laryngeal trauma can be due to mechanisms:

1. Direct blow, as in sports injuries, dashboard injuries, falls, and assault
2. Deceleration injury, as in high-speed auto accident while restrained
3. Crushing injury of the chest leading to sudden incr in tracheal air pressure
4. Strangulation
5. Hanging
6. Bedbars, bedrails

Laryngeal fracture is less common in children bc of their pliable larynx. But they get severe edema.

HEAT INJURY

(Not burns)

Heat-related illness and the automobile. Surrpure JS. Ann Emerg Med 1982; 11: 262-265

Overheating in infancy. Arch Dis Child 1983; 58: 673-4

A mathematical model of life-threatening hyperthermia during infancy. Jardine DS

An autopsy case of infant death due to heat stroke. Ohshima T, Maeda H, Takayasu T et al. Am J Forens Med Path 1992; 13(3): 217-221. A 52 day old infant wrapped up and placed under a foot-warmer, found moribund with first-degree burns, T=41.2

Heat stroke in well-wrapped infants. Bacon C, Scott D, Jones P. Lancet 1979 Feb 24; 1(8113): 422-425
Five infants, four died.

Temperature, age, and recurrence of febrile seizure. Van Stuijvenberg M et al. Arch Ped Adol Med 1998 Dec; 152(12): 1170-1175. Studied 230 children. Young age at first seizure, height of temperature at first sz, and height of temp at second sz were the statistical risk factors for second febrile sz.

Environmental hyperthermic infant and early childhood death: circumstances, pathologic changes, and manner of death. Krous HF, Nadeau JM, Fukumoto RI, Blackbourne BD, Byard RW. Am J Forens Med path 2001 Dec; 22(4): 374-382. Ten documented cases -- eight in vehicles and 2 in beds. Age range 53 days to 9 years. Living or longer-surviving victims had centrilobular necrosis of the liver, myonecrosis or rhabdomyolysis, and DIC. Lung disease renders them more vulnerable. All 8 children discovered in vehicles had intrathoracic petechiae, while one of the two children found in bed had them. Gives an extensive discussion of intrathoracic petechiae. Since SIDS also frequently have intrathoracic petechiae, and SIDS have been shown by Guntheroth & Spiers to be associated with thermal stress, these may be the extreme end of a heat continuum that includes SIDS. In that case, meticulous scene investigation is required to differentiate heat exposure. Says that as to homicide versus accidental manner of death, "The manner of death was considered accidental when the investigation indicated miscommunication among the caretakers, or if the child entered the vehicle without the caretaker's knowledge." (p. 381)... The manner of death was homicide in 5 cases on the basis of inflicted trauma and circumstances indicating neglectful parenting... Verification of the caretakers' understanding of their responsibilities regarding the child's welfare is critical." The autopsy findings mainly vary by the duration of survival *after* the heat exposure.

Temperature variations in automobiles in various weather conditions: an experimental contribution to the determination of time of death. Marty W, Sigrist T, Wyler D. Am J Forens Med Path 2001 Sep; 22(3): 215-219. In the summertime in Zurich, cars parked in the open reached 89 deg C. In the wintertime they only got up to 21 C. The authors offer a 30-60-90 rule of thumb for peak temperatures in cars parked in direct sunlight in temperate climes:

Winter	30 C	86	F
Spring and fall	60 C	140	F
Summer	90 C	194	F

(To convert C to Fahrenheit, multiply by 1.8 and add 32.)

The authors suggest that the temperature behavior of the particular car in question be measured.

POSITIONAL (see also strangulation)

Accidental asphyxial deaths involving infants and young children. Sturmer WQ, Spruill FG, Smith Ra, Lene WJ. JFS 1976 Jul; 21(3): 483-487.

NEGLECT

The California Child Abuse & Neglect Reporting Act, Penal Code Sec. 11164 et seq.

Calif. Penal Code Sec. 11165.2: “As used in this article, ‘neglect’ means the negligent treatment or the maltreatment of a child by a person responsible for the child’s welfare under circumstances indicating harm or threatened harm to the child’s health or welfare. The term includes both acts and omissions on the part of the responsible person.

- (a) ‘Severe neglect’ means the negligent failure of a person having the care or custody of a child to protect the child from severe malnutrition or medically diagnosed nonorganic failure to thrive. ‘Severe neglect’ also means those situations of neglect where any person having the care or custody of a child **willfully** causes or permits the person or health of the child to be placed in a situation such that his or her person or health is endangered, as proscribed by Section 11165.3, including the *intentional* failure to provide adequate food, clothing, shelter, or medical care. [11165.3 has to do with unjustifiably cruel punishment. --JKR]
- (b) ‘General neglect’ means the **negligent** failure of a person ... to provide adequate food, clothing, shelter, medical care, or supervision where no physical injury to the child has occurred.”

Penal Code Sec. 270. “If a parent of a minor child **willfully** omits, without lawful excuse, to furnish necessary clothing, food, shelter, or medical attendance, or other remedial care for his or her child, he or she is guilty of a misdemeanor...” [Sec. 270 has mostly to do with the former common-law duty of child support and spousal support.]

Welfare & Institutions Code Sec. 16509 provides in part that “cultural and religious childrearing practices and beliefs which differ from general community standards shall not in themselves create a need for child welfare services unless the practices present a *specific danger* to the physical or emotional *safety* of the child.”

A national legal definition is offered by ivillage.com at <http://pages.ivillage.com/sacboard/>. “ ‘Neglected child’ means a child less than 18 years of age whose physical, mental or emotional condition has been impaired or is in danger of becoming impaired as a result of the failure of the child’s legal guardian to exercise *a minimum degree of care* in supplying the child with adequate food, clothing, shelter, education or medical care. Neglect also occurs when the legal guardian fails to provide the child with proper supervision or guardianship by allowing the child to be harmed, or to be at risk of harm which includes when the guardian misuses drugs or alcohol him/herself. Most caregivers do not intend to neglect their children. It usually results from ignorance about appropriate care for children or an ability to plan ahead. Neglect occurs when a caregiver fails to provide basic needs such as adequate food, sleep, safety supervision, clothing, or medical treatment.” (emphasis added --JKR) There are comparable definitions in all the standard texts.

National Clearinghouse on Child Abuse & Neglect Information. <http://www.calib.com/nccanch>
800-394-3366 x 352.

Homicide by starvation: the nutritional variant of the "battered child." Adelson L. JAMA 1963; 186: 458-460

Failure to thrive. Cupoli JM, Hallock JA, Barnes LA. Curr Prob Pediatr 1980; 10: 1-43. Cited by Simmons in ASCP CheckSample 04-9 for the proposition that weight goes first, followed by height, followed by head.

Adelson L, The fatally abused child: infanticide and the lethal maltreatment syndrome. In: *The Pathology of Homicide*. Springfield, IL: Charles C. Thomas, 1974. Cited by Laura D. Knight and Kim Collins (2005) for the proposition that the caregiver tends to minimize the duration of the symptoms and the neglect, and to provide a history inconsistent with the investigation and autopsy findings in neglect cases.

Subtle fatal child abuse. Zumwalt RE and Hirsch CS. *HumP* 1980; 11: 167-174

Differentiation of organic from non-organic failure to thrive. Rosenn SW, Loeb LS, Jura MB. *Pediatrics* 1980; 66: 689-704

A forensic science approach to a starved child. Davis JH, Rao VJ, Valdes-Dapena M. *JFS* 1984; 29: 663-669. Cited by Simmons in ASCP CheckSample 04-9 for the proposition that you should exclude “neurological deficits” as a cause of failure to thrive.

Infanticide by starvation: calculation of caloric deficit to determine degree of deprivation. Meade JL, Brissie RM. *JFS* 1985; 30: 1263-1267. Cited by Simmons in ASCP CheckSample 04-9 as a method to calculate the number of “days’ worth” of food missed by an infant under 2 months of age. Piercecchi-Marti et al. find that this method only works if there was a period of absolute deprivation, not if there was continuous but reduced intake. See Piercecchi-Marti et al., 2006, below.

A case of panhypogammaglobulinemia masquerading as child abuse. Copeland AR. *JFS* 1988; 33: 1493-1496. See Laura D. Knight and Kim Collins, 2005, below, for the differential diagnosis of starvational neglect.

Homicide by starvation. Sarveswaran ER (Charing Cross Hospital) *Am J Forens Med Path* 1992; 13(3): 264-267 4 year old girl emaciated found mummified in home. The mother had been under emotional stress. British case.

Chapter, “Growth Failure in Infants,” by Kim Oates and Ruth Kempe in Kempe H., *The Battered Child*, 5th ed. University of Chicago Press.

Chapter 24 in Ludwig S and Kornberg AE, *Child Abuse: A Medical Reference*, 2d ed. New York: Churchill Livingstone, 1992. Refers us to statutes.

Assessment of the child with failure to thrive. Leung AKC, Robson WLM, Fagan JE. *Am Fam Phys* 1993; 48: 1432-1438. Cited by Simmons in CheckSample FP-04-9 for the proposition that you must conduct “a very careful, deliberate search for asymmetry and/or dysmorphic features that may suggest a chromosomal anomaly or a clinical syndrome... This in particular includes the head, genitalia, and dermatoglyphics, which are abnormal with many chromosomal abnormalities.”

Nelson’s Textbook of Pediatrics, 15th edition (Philadelphia: W. B. Saunders, 1996), chapter 38.2 on nonorganic failure to thrive, (or a later edition) mentions the following observable findings in NOFTT:

dietary history may be inaccurate

thin extremities

narrow face

prominent ribs

wasted buttocks

neglect of hygiene

diaper rash

unwashed skin

untreated impetigo

- uncut and dirty fingernails
- unwashed clothing
- flattened occiput with hair loss
- delays in social and speech development
- avoidance of eye contact
- expressionless face
- absence of cuddling response
- amount of time the mother spends holding baby

“A rejecting mother often feeds her baby with anger and unnecessary force. This may result in a torn frenulum and aversion to feeding.” (p. 119)

Chapter 39 states that “FTT usually refers to a child growing below the 3d or 5th percentile or a child whose decreased growth has crossed two major percentiles in a short time.” For the workup, it offers three conceptual areas:

- (1) failure of parent to offer adequate calories
 - lack of knowledge
 - depression
 - dietary beliefs
 - lack of food
- (2) failure of child to take sufficient calories
 - difficulty swallowing due to pain or oral-motor dysfunction
 - anatomic abnormality of the oroesophageal apparatus
 - cardiopulmonary dysfunction
 - chronically infected tonsils
- (3) failure of child to retain sufficient calories
 - vomiting
 - diarrhea
 - malabsorption
 - cystic fibrosis
 - other*

* See the charts given on p. 123.

Mentions clinical findings:

- height and weight
- alopecia
- loss of subcutaneous fat
- reduced muscle mass
- dermatitis
- recurrent infections
- marasmus
- kwashiorkor

Altered consciousness and shock in a malnourished child. Gingold MK, Rajakumar K, Rosas AJ. *Semin Pediatr Neurol* 1996 Sep; 3(3): 248-254. From the abstract: A 3 yr 9 mo child presented unr dehy shock due t neglect abuse and starvation. Kwashiorkor and “intellectual repercussions” (?long term sequelae) of starvation.

The neglect of child neglect. Cantwell HB. In: *The Battered Child*. Helfer ME, Kempe RS and Krugman RD, eds., 1997. (have abstract kindly contributed by Sandi McLeod). See also a *Lancet* editorial by the same name published in 2003 (below).

Child fatalities from religion-motivated medical neglect. Seth Asser and Rita Swan. *Pediatrics* 1998 Apr; 101 (4 part 1): 625-629. Collects 172 deaths. See also The APSAC "Advisor," Summer 1998, vol 11, no. 2, letters.

Community and professional definitions of child neglect. Howard Dubowitz, Klockner A, Starr RH, Black M. *Child Maltreatment* 1998 Aug; 3(3): 235-243 Annabella Klockner for her masters thesis did an empirical study of attitudes of Black and White people of middle and lower-class, and also of child maltreatment professionals, regarding what constitutes psychological neglect and physical neglect. "A clear definition of child neglect is sorely needed." Why? Because the child protective services cannot get involved unless they see something they can objectively define as "neglect." Not just somebody's opinion, but a defined standard that will be constant across different social classes and groups. These authors propose a child-centered definition: "Neglect occurs when a basic need of a child is not met, resulting in actual or potential harm." [This tracks Reece, *supra*.] But they admit that neglect has *usually* been defined as "an omission in care by parents or caregivers that deviates substantially from *community standards*." [emphasis added --JKR] In applying this definition, there has been concern about imposing middle-class standards on lower-class communities. Also about majority cultures forcing their values on ethnic and national minorities. In other words, community standards, but whose community? Previous research has shown that middle-class persons are more concerned about psychological care, while lower-class place more emphasis on physical safety. But the studies show overlap in values between classes, and do not suggest fundamental disagreement. The present study confirmed the above patterns, showing considerable agreement across social class and race. The authors close with a plea again for their objective, harm-based definition, noting that CPS will only become involved when "harm" to the child is likely.

Fatal child neglect. Bonner BL, Crow SM, Logue MB. In: *Neglected Children: Research, Practice and Policy*, edited by H Dubowitz, 1999.

Identifying and documenting findings of physical child abuse and neglect. Cheung KK. *J Pediatr Healthcare* 1999; 13: 142-143. Cited along with Thompson, McCarley, 2003 and Corey & Collins in the 2d edition of Dehner's *Pediatric Pathology* (2002) by Laura D. Knight and Kim Collins in their 2005 review of 25 years of child neglect deaths in North Carolina, below, for the definition of child neglect.

Child neglect: guidance for pediatricians. Dubowitz H, Giardino A, Gustavson E. *Pediatr Rev* 2000 Apr; 21(4): 111-116

Fathers and child neglect. Dubowitz H, Black MM, Kerr MA, Starr RH jr, Harrington D. *Arch Pediatr Adol Med* 2000; 154: 135-141. Longitudinal study of 244 poor Black families in Baltimore City. In 176 families (72%) a father or father figure could be identified. They were able to interview 117 of these men. Found: (1) the *mere presence* or absence of a father figure in the home did not alter the neglect rate. But (2) if the father figure was *involved* and pleased with his role, there was a lower neglect rate.

A fatal case of infantile scurvy. Mimasaka S, Funayama M, Adachi N, nata M, Morita M. *Int J Legal Med* 2000; 114(1-2): 122-124. We initially suspected that the bleeding was due to violence. But it was due to neglect. The parents locked the child alone in a room while they went out during the day.

Transportation barriers a hidden factor in explaining lack of medical care. Reuters Medical News on Medscape, October 2000. The Children's Health Fund did a study showing why so few children took advantage of federally funded free health care.

Medical neglect was found to be present in 20% of all admissions for child maltreatment of any type. Reuters Medical News on Medscape, citing a 2000 presentation to the 128th annual meeting of the APHA by Dr. Thomas J. Songer and Lorraine R. Ettaro of the University of Pittsburgh, who analyzed 150 pediatric inpatients on whom a report of child maltreatment had at some time been filed. Of these, 39 patients had been neglected and 30 had suffered medical neglect. Most of the medical neglect pertained to chronic illnesses such as asthma and diabetes. 94% of it occurred with clients of government-sponsored payors.

Chapters 12 and 13, "Failure to thrive" and "Child Neglect" in Reece 2d edition, 2001. Ch. 13 states that child neglect has been difficult to define, and distinguishes what a treating pediatrician might consider "neglect" and the narrower area that a prosecutor would consider provable in court. Notes that societal factors such as poverty impair families' ability to care for children. Suggests a general definition as "the basic needs of children are not met, regardless of cause." (This is not a criminal definition but a conceptual one.) "Basic needs include adequate food, supervision, and protection; clothing, health care; education; a stable home; and the emotional needs for love and nurturance." This definition defines when neglect exists, rather than locating responsibility for it. Refers to state statutes and discusses actual versus potential physical harm. As to medical neglect, Chapter 12 states that parents are (legally) responsible for (a) recognizing health problems, and (b) seeking necessary health care for those problems. Medical neglect occurs when a parent (a) fails to recognize a health problem *that a reasonable parent would recognize*, or (b) fails to seek care for such a problem.

Nutritional neglect and physical abuse in children of alcoholics. Rao KN et al. *Ind J Pediatr* 2001 Sep; 68(9): 843-845. In an academic general hospital in India, 72 children of alcoholics were compared with 81 controls. Malnutrition found in 86% of the alcoholics' children versus 49% of the non-alcoholics'. Physical injuries in 45% versus 23%. [JKR note: Caregiver alcoholism is also a risk factor for elder abuse. See, e.g. Elder abuse: ten years later. Wolf RS. *J Am Geriatr Soc* 1988 Aug; 36(8): 758-762.]

[Medical neglect]. *Arch Ped Adol Med* 2001 Aug; 155(8): 927-933.
See comment by Dubowitz, *infra*.

Medical neglect: a child-focused view. Dubowitz H, Watson D, Farley J. *Arch Ped Adol Med* 2002 Mar; 156(3): 297-298. Comment on *Arch Ped* 2001 Aug; 155(8): 927-933, above.

Fatal child neglect. Berkowitz CD. *Adv Pediatr* 2001; 48: 331-361. Failure to provide the basic needs. Such as food or medical care. N = 30% to 40% of all fatalities from child maltr. Also incl inadequate supervision contributing to accidental deaths by fire, drowning, firearms. Recognizing the involvement of inadequate care requires multidisciplinary child death review teams.

Child neglect: outcomes in high-risk urban preschoolers. Dubowitz H, Papas MA, Black MM, Starr RH. *Pediatrics* 2002 Jun; 109(6): 1100-1107. Tried to separate the effects of poverty from those of parental physical and psychological neglect. Complicated.

The neglect of child neglect. Editorial. *Lancet* 2003; 361: 443. According to Dr Reece's summary in the *Quarterly*, says that nobody is going into child protection. See chapter of the same name from 1997, above. The same problem is referred to later in the court hearings and GMC hearings on the Sally Clark case.

Practical considerations in the evaluation and management of child neglect. Thompson WK, McCarley AL. *Clin Fam Prac* 2003; 5: 1.

Berkowitz C. Child Neglect. Failure to Thrive. In: *Child Abuse and Neglect: Guidelines for Identification, Assessment, and Case Management*. Marilyn Strachan Peterson and Michael Durfee, eds. Volcano, California: Volcano Press, 2003. 370 pp. ISBN 1-884244-21-1. Chapter updates: <http://www.volcanopress.com/>

Refeeding syndrome in a severely malnourished child. Willis TS, Boswell R, Willis M. *Lab Med* 2004; 35(9): 548-552. Case report of a moribund 4 year old black male rescued from a private home with a BMI of 12 kg/M2 (below 5th %), rectal temp 29.3, glucose 20 HR 50 requiring emergent fluid resuscitation. Pressure sores over the sacrum, left ear, and left pelvic rim. Multiple small scars. Had been taken out of foster care and placed with his biological parents 1 year before presentation. At that time he had been 75th %. Developed the refeeding syndrome as soon as they started him on parenteral nutrition. This consists of "abnormalities in fluid balance, glucose metabolism, vitamin deficiency, hypophosphatemia, hypomagnesemia, and hypokalemia in patients exposed to enteral or parenteral nutrition after a period of starvation and weight loss." Article has clinical photographs and lab values. Extensive references on

hypophosphatemia. Also Wernicke's, with the symptoms of Wernicke's listed under "thiamine deficiency" as being delirium, oculomotor paresis, nystagmus, and ataxia.

Estimation of caloric deficit in a fatal case of starvation resulting from child neglect. Nagao M, Maeno Y, Koyama H, Seko-Nakamura Y, Monma-Ohtaki J, Iwasa M, Zhe LX, Kawashima N, Yano T. JFS 2004 Sep; 49(5): 1073-1076. Case report of a 3 year old girl who died of starvation. 70 days before death she weighed 12 kg. The parents kept her in a packing case. The parents said they gave her 100 to 150 ml of milk every few days as long as she was able to take it. The body weighed 5 kg. 89 cm in height. Had lost 58% of her body weight. The authors used a PAS stain of liver to demonstrate a lack of glycogen. Had pressure sores on the head, back and pelvis. Contractures of the knees. Edema of the feet. Low organ weights. No fat. Atrophic sclerae, through which the choroid could be seen. Empty intestines. Sed that the requirement for basal metabolism in such a girl was 700 kcal/day. You multiply that by activity factors: 1.3 for low activity level, 1.5 for moderate activity, 1.7 for moderate-heavy, and 1.9 for heavy. Then you add an amount required for growth, which is 16 kcal/day. In this case, there was no growth, so the growth component was not used. What the authors did was estimate the activity factor based on what the parents said, as 1.5 (moderate activity level) for the first 14 days, 1.3 for the next – days, and 1.0 for the remaining period of complete inactivity/ inanition. They somehow estimated her daily caloric intake (apparently from what the parents reported). They subtracted the intake from the daily requirement and got the absolute caloric deficit per day (chart) and added them up. Then they assumed an initial percentage body fat of 20%, giving an initial total body fat content of 2.4 kg. That would be good for 7.2 kcal/gram. The remainder of the weight loss (4.6 kg) was assumed to be protein. That would be good for 4 kcal/gram. They then did a retrospective prediction of her weight loss based on the historical food intake and these caloric figures, and found that it came out right on the money, giving a final weight of 4.988 kg. They used this method to test the reliability of the parents' statements, and found them to be reliable. This calculational method can be used to backtest the reliability of parents' statements about how much they fed a starved child. "The statements of the parents therefore appear to be true."

This article is cited in Piercecchi-Marti et al., 2006, below. In that case, the authors found it impossible to derive the time of deprivation per the methods described by Nagao et al., because in their case there was no period of absolute food deprivation.

ASCP Check Sample FP 04-9 (FP-300). Gary T. Simmons, MD. Case of a 14 month male who weighed 10.4 pounds and was 65 cm tall with a head circ of 43.5 cm. Clean skin, no diaper rash, well trimmed nails, no dysmorphism, organ weights typical for 2 months of age except the brain of 930 grams typical for 11-12 months of age. The thymus was undetectable grossly but was detected by sectioning the entire anterior superior mediastinum histologically. The GI tract contained 8 ml of yellow liquid. Colon had solid stools. The differential diagnosis of fatal malnutrition is

criminal neglect
parental ignorance
congenital disease

Cites Leung, 1993 for the proposition that you should search carefully for asymmetry, abnormal dermatoglyphics, abnormal head and face or genitalia typical of a dysmorphic syndrome. Cites Cupoli, 1980, for the proposition that "In the competition for nutrients, weight is lost first, followed by an arrest in height growth, and finally an arrest in head growth." The height can be used to give a rough estimate of the period of deprivation. Meade and Brissie, 1985 give a method for estimating the number of "days' worth" of food deprivation in infants 2 months or under. Get vitreous electrolytes because dehydration is common. If all fat stores are depleted, ketones, which are products of incomplete fat metabolism, will be low to absent. Test for lead. Rotavirus on intestine. Investigate the home and the caregivers with respect to the above differential diagnosis. All available history and records on the child. A dietary history. Look for dilution or adulteration of formula. Also note the siblings: if their growth is normal, that is evidence against an intrinsic cause and in favor of an environmental cause for malnutrition.

Clinical guideline on oral and dental aspects of child abuse and neglect. American Academy of Pediatric Dentistry. Pediatric Dentistry 2004; 26(7): 63-66. Reviews the oral and dental aspects of physical and sexual abuse as well as **dental neglect**. Including bite marks and oral STD's.

Failure to thrive as a manifestation of child neglect. AAP clinical report. Block RW, Krebs NF, and the Committee on Child Abuse and Neglect, and the Committee on Nutrition. Pediatrics 2005 Nov; 116(5): 1234-1237. Definition: FTT “is a significantly prolonged cessation of appropriate weight gain compared with recognized norms for age and gender after having achieved a stable pattern (e.g. weight-for-age decreasing across 2 major percentile channels from a previously established growth pattern; weight-for-length < 80% of ideal weight.) This is often accompanied by normal height velocity.” Refers us to the AAP’s *Pediatric Nutrition Handbook* for a thorough discussion of FTT. FTT can be unintentional: nursing difficulties, poor diet selection. It can be organic:

- cystic fibrosis
- cerebral palsy
- HIV
- metabolic disease
- celiac sprue
- renal disease
- lead poisoning
- major heart disease

Often multifactorial, which is why the old “organic/nonorganic” dichotomy is no longer used. Factors may include dysfunctional parent behavior and parent-child interaction difficulties.

If neglect is being considered, one should look for risk factors for neglect, but the significance of risk factors should be considered in the context of each family’s unique situation. Risk factors:

- maladaptive socialization of parents
- mental retardation of parent
- parental history of abuse, stress, divorce, marital strife, DV
- noncompliance
- premature birth
- low birth weight
- separated at birth due to NICU
- lack of an extended family
- social isolation [reminds us of elder abuse risk factors --JKR]
- substance abuse
- single parent
- employment instability
- dietary faddism

Any of these factors may lead to inconsistent feeding patterns. Discusses attachment issues. Technique for evaluation of FTT includes feeding observation and a home visit. 72-hour dietary record, prenatal history, family history of organic disease, growth charts, assessment of suck-swallow coord. Lactation assessment if nursing. Laboratory tests are needed only if the above does not reveal a cause. Its purpose is to (a) rule out organic disease, and (b) ascertain nutritional deficits. The yield of lab data even if positive is less than 1%. Multidisciplinary approach is essential. FTT must be considered a medical emergency if the growth curve documents that the **weight-by-length is less than 70% of predicted**. Otherwise, management is per the Handbook. If maltreatment is suspected, refer also to child protection authorities. Hospitalization is appropriate if severe FTT or if neglect is suspected. This is also a diagnostic maneuver: a neglected child will eagerly eat and gain weight once taken out of the home environment. Notes that the correction of severe malnutrition is a delicate task -- the refeeding syndrome (see Willis et al., above). Parental support and teaching. Foster care placement is a last resort, only after the above measures have failed.

A 25-year retrospective review of deaths due to pediatric neglect. Knight LD and Collins KA. Am J Forens Med P 2005 Sep; 26(3): 221-228. Neglect defined as the failure of a caregiver to adequately provide safety, food, clothing, shelter, education, protection, medical/dental care, and supervision. These

authors reviewed 16 deaths (average age 1.9 years, age range 6 weeks to 11 years; however, all but one were under 3) due to some form of neglect.

starvation/dehydration	6 cases
toxic ingestion	2 cases
hyper- or hypothermia	2 cases
unusual drowning or aspiration	4 cases
electrocution	1 case
medical neglect	2 cases

Malnutrition/Starvation/Dehydration cases

1. The history did not match the findings. Examples: toddler was said to be fine until one day before death. Or, a history of feeding just before death, with an empty GI tract. Home without running water or formula. Urine-soaked bed.
2. The findings:

- skin tenting
- wrinkled, loose skin
- sunken fontanelle
- sunken eyes
- depressed cranial sutures
- focal alopecia
- prominent ribs and bones
- weight < 5%
- loss of adipose and muscle tissue
- dry mucus membranes
- decreased organ weights
- minimal to no GI contents
- serous atrophy
- adrenal glands with lipid depletion and cortical pseudotubular change
- thymic involution
- bone demineralization leading to pathologic fractures
- vitreous humor showing
 - isonatremic dehydration (high VUN)
 - hypernatremic dehydration (high sodium and VUN)

Toxic ingestions -- inadequate supervision with or without nutritional neglect or medical neglect

One sixteen month-old ate rock cocaine. Another sixteen month-old drank hydrogen peroxide and suffered oxygen embolization. In the cocaine case, the child was malnourished and ate the rocks because he was hungry. In the hydrogen peroxide case, at autopsy there was foamy blood throughout the circulatory system. The sixteen year-old caregiver left the child unattended for twelve hours after it drank the peroxide (medical neglect).

Hyper- and hypothermia

The hyperthermia case was a 22 month-old who was left in a van for 30-60 minutes with the windows down and the air conditioning on, while his mother went to visit with a neighbor. His sibling, also left in the van, was asymptomatic, but the victim was found in DIC and died in the hospital with systemic fat emboli, acute tubular necrosis, acidosis, focal infarction of the adrenal cortex. No admission temperature was documented. The cause of death was somewhat unclear. The hypothermia case was a 3 year-old who was brought to a mortuary by his family, found to be ice cold, bright pink/red muscles, and pattern contusions.

Unusual drownings

Two 9 month-olds fell into buckets of bleach after being left unsupervised. Smelled strongly of bleach and had opaque corneas. The pH of stomach contents was measured but proved noncontributory. One three year-old fell into a disused swimming pool with four feet of weedgrown water where he was often allowed to play. A two year-old was drowned when a recreational boat capsized; she was not wearing a lifejacket and should never have been in the boat in the first place.

Electrocution

A two year-old was in the bathtub supervised by an 11 year-old. Nearby was a 110-volt potential difference between a defective freestanding electric heater and a metal wall vent. The child got out of the tub and walked between the two, and was electrocuted. The trailer had no phone, so there was delayed rescue.

Medical neglect

An 11 year-old was said to have flulike symptoms for one day. Autopsy disclosed 350 ml of pus in the peritoneal cavity with a ruptured appendix. The history of one day of symptoms did not match the findings.

Discussion

The authors note the following common features of the sixteen neglect cases:

1. The caregivers minimized the duration of the neglect or illness.
2. The scene investigation was indispensable.
3. The cases broke out into two categories
 - a. Starvation cases -- infants or toddlers who were not fed
 - b. Lack of supervision cases -- older, mobile children who got into trouble when left unsupervised or not given obviously needed medical care when sick.

With regard to these two types of cases, the authors observe as follows:

- a. Starvation cases:
 - decreased body weight
 - decreased fat content
 - hepatic steatosis
 - thymic involution with microcalcifications
 - stress changes of the adrenal glands
 - remote injuries such as fractures due to demineralization
 - Metabolic and genetic screening should not be overlooked.
 - Differential diagnosis: cystic fibrosis, hypogammaglobulinemia, hypopituitarism, malabsorption, pyloric stenosis, glycogenopathy, global retardation, "and many others."
 - growth chart
 - Microscopic evidence of natural disease should be looked for. Natural disease does not rule out neglect, if it resulted from neglect-induced immunodeficiency.
- b. Lack of supervision cases and medical neglect cases:
 - The scene investigation is critical.
4. The authors discuss the manner of death. The death should be diagnosed as homicidal when it "resulted from the actions of another." This can include acts of omission such as not feeding. Sometimes food or water was withheld intentionally as a punishment. Sometimes the mother had a skewed understanding of nutritional needs, such as the mother of a skin-and-bones infant who thought her child was "getting fat." If there is a lack of investigative information, or if the cause of death is unclear, a diagnosis of Undetermined manner of death is appropriate. Milder forms of neglect, such as children briefly left unsupervised, are classically diagnosed as accidental.
5. "A careful scene investigation, review of medical records, questioning of the caregiver regarding feeding and other daily care activities, complete autopsy with skeletal survey, toxicology, chemical and metabolic testing, and exclusion of any mimickers of neglect are necessary. Only when all of these

components of investigation are well completed can a compelling case be made for a determination of cause and manner of death reflecting pediatric neglect.”

This article is reviewed in the Spring 2006 issue of the *Quarterly*.

Oral and dental aspects of child abuse and neglect. Nancy Kellogg, M.D. and the American Academy of Pediatrics Committee on Child Abuse and Neglect. *Pediatrics* 2005 Dec; 116(6): 1565-1568. [See also the American Academy of Pediatric Dentistry report, (2004), above.] See above under “Dental and Face.”

Criminally prosecuted cases of child starvation. Kellogg ND and Lukefahr JL. *Pediatrics* 2005; 116: 1309-1316. Describes twelve cases. “*Results.* Of the 12 cases reviewed, the median age was 2.7 years, with a range of 2.25 months to 13 years 7 months. Half of the children died shortly before or soon after presentation for medical care or to law enforcement. Survival was more common in older children than in infants. Most of the children were secluded from others, and all had access to food denied or severely restricted. Caretakers claimed few, benign, or no past medical illnesses in the children. Based on weight and height measurements, 10 of the children had severe wasting and stunting, and 2 had mild or moderate wasting. There was a tendency toward more severe wasting in the fatal cases. All children manifested multiorgan effects of **starvation**. All survivors manifested complications with refeeding. Approximately half of the children had past or present injuries or history suggestive of physical or sexual abuse. Parental rights were terminated in all cases. A total of 25 individuals were charged criminally; 23 were found guilty or pled guilty, and trials for 2 individuals were pending at the time of this writing. The types of criminal charges and punishment varied from deferred adjudication to a life sentence.”

Malnutrition, a rare form of child abuse: diagnostic criteria. Piercecchi-Marti M-D, Louis-Borrione C, Bartoli C, Sanvoisin A, Panuel M, Pelissier-Alicot A-L, Leonetti G. *JFS* 2006 May; 51(3): 670-673. Case report of a French six year-old with marasmus. “The essential clinical sign is height and weight retardation, but malnutrition also causes a variety of internal and bone lesions, which lead to neuropsychological sequelae and death.”

Case report: Weight at death was 3.166 kg, ht 59.5 cm, HC 41.5 cm (all 2.5%). Dirt in the skin creases, weeping dermatitis of the buttock area, soft nails, . Signs of dehy: sunken eyes and hypotonia, marked skin folds, and depressed anterior fontanelle, dry tongue. Thymus 3g, fatty liver with a negative PAS stain (no glycogen), no subq fat, reduced muscle mass, empty stomach, empty colon, X-rays showed metaphyseal abnormalities known as **Harris lines** and delayed bone maturation. Central chromatolysis of the neurons in the mammillary bodies. The intestinal villi were not altered, and there was no history of diarrhea, excluding malabsorption. Blood and bile contained high levels of acetone (blood 20 mg/l, n=3.1), suggesting starvation ketosis. History: Weight 2.9 kg at discharge from the birth ward, the nursery nurses reported concern about the mother-child interaction at that time, newborn screening tests were negative for metabolic diseases, only one doctor visit at age 4 ½ months, at that time the child weighed 5 kg, no vaccinations, the parents did not respond to followup letters, two older children did not attend school regularly and were also undergrown. Mother admitted that her child had lost weight, but she had not thought it necessary to see a doctor (the family were North African). Feeding history: the mother was unable to describe the frequency of bottles. Diet appeared to have consisted of bottles of cow’s milk diluted with water plus some vegetable matter provided. On the day of death, the mother stated she had fed the baby a bottle of milk with semolina and fruit compote shortly before death. This statement was contradicted by the autopsy finding of empty lumens and absence of glycogen. The parents were sentenced to 20 years.

Discussion: Death from abusive malnutrition is rare. Harris lines are growth-arrest lines, a nonspecific marker for stress. “On x-ray,” the authors state, “they appear as dense trabecular bone [transverse] to cancellous bone, whereas normal trabeculation is longitudinal. They result from temporary arrest of biological processes, when cartilage is not mineralized and hypertrophic cartilage is no longer transformed into calcified cartilage.” Citing Resnick’s book [Resnick D. *Diagnosis of bone & joint disorders*. Philadelphia: W. B. Saunders, 1995, p. 3353-3355]. Discusses protein-calorie metabolism. Marasmus and kwash, but there are intermediate forms. Says that this case was clinically typical of marasmus (as is obvious from the photograph --JKR), but the fatty liver favors kwash. Marasmus is where both protein intake and calorie intake are reduced, but they are reduced in a balanced fashion. Kwash is low protein intake with adequate carbohydrate intake: its hallmarks are edema and low serum albumin, and the weight loss is much less marked than it is in marasmus. In either case, biological abnormalities are rare

and late. Death takes a few weeks once the condition has set in. Either disease has exudative lesions of the skinfolds or orifices. The development includes reduced physical activity (the child in question was described as calm and slept a lot). Growth failure usually occurs a few weeks after slowing of weight gain, the first clinical sign of malnutrition (citing Berkowitz). Renal function is impaired with inability to concentrate the urine, resulting in polyuria and dehydration, which may be exacerbated by inadequate fluid intake due to inanition in the last few days of life. Decreased bone mass and demineralization. Anorexia, irritability or apathy. Impaired temperature regulation of body may lead to hypothermia even in a heated apartment. Absence of liver glycogen in marasmus may lead to fatal hypoglycemia. After death, if there is no obvious infection, "it is impossible to determine the terminal pathological factor which led to death: dehy, hypogl, or hypoth may be debated."

Discusses diets. Says that cow's milk is not well adapted to the growth curve of human infants because cow infants grow up so much faster. Hence, cow's milk contains protein, casein, and ions that are three to seven times higher than human breast milk or formula, while the lactose content is 50% lower. Iron, essential fatty acids, vitamin E, vitamin C, and folic acid content are markedly inadequate. A diet of cow's milk for an infant leads to protein deficiency because the infant intestine cannot absorb the proteins. And also fluid & electrolyte imbalance and vitamin deficiency. (Citing a French textbook). This leads to a diarrheal syndrome, which the present child did not have, suggesting to the authors that the problem in their case was not the cow's milk diet but simply inadequate total intake.

Refers to the mathematical derivation of the duration of food deprivation as described by Nagao et al. (JFS 2004, above) and Meade (JFS 1985). But says that that was not possible in this case because there was in this case no period of absolute food deprivation; food was given, but just in reduced amounts.

When is lack of supervision neglect? (Clinical Report). Kent P. Hymel MD and the AAP Committee on Child Abuse and Neglect. Pediatrics 2006. <http://www.pediatrics.org/cgi/doi/10.1542/peds.2006-1780>
When should a pediatrician report a family to the child protective services if he (a) becomes aware of children who are inadequately supervised, or (b) treats an ill or injured child whose condition he suspects could have been prevented by better supervision? It is a fact that inadequate supervision is a frequent cause of household injuries. "The AAP believes that supervisory neglect occurs whenever a caregiver's supervisory decisions or behaviors place a child in his or her care at significant ongoing risk for physical, emotional, or psychological harm." Consider the following factors [I have reworded some for brevity -- JKR]:

1. Local published guidelines, e.g. DCFS guidelines on when you must report
2. Consider every allegation individually
3. Consider:
 - a. has that child previously shown an ability to adequately care for itself
 - b. does the child have disabilities?
 - c. when and for how long was it left unsupervised?
 - d. does the caregiver understand why this is a problem?
 - e. how dangerous was the situation? (e.g. tub, pool)
 - f. how comfortable was the child with being left there?
 - g. what was the child doing? (just playing or viewing pornography?)
 - h. did the child know emergency numbers?
 - i. did the child know how to safely answer the telephone etc?
 - j. how near access to another caregiver?
 - k. past allegations of neglect?
 - l. who was the caregiver? good or bad?
 - m. what other children were there?
 - n. was it age-appropriate to leave this particular other child in charge?
4. Remember that some injuries happen even with close supervision. Might this have been one of those?
5. Remember that parents are already remorseful about a preventable injury, even without your butting in.

6. Report it “when a reasonable suspicion exists that a pattern of caregiver decisions or behaviors has placed a child at significant ongoing risk for physical, emotional, or psychological harm.”

SIBLING ABUSE (see also MSBP)

The battering child. Adelson L. JAMA 1972; 222: 159-161. According to Kempe & Helfer's fourth edition (p. 260), Adelson describes five cases of fatal head injuries in young infants inflicted by children 2 ½ to 8 years of age. Four with extensive skull fractures. Stating that the injuries were inflicted with blunt objects such as toys, or by being dropped to the floor. See next below.

Zumwalt RE and Hirsch CS, Pathology of fatal child abuse and neglect. Chapter 13 *In:* Helfer RE and Kempe RS, *The Battered Child*, 4th edition. Chicago: University of Chicago Press, 1987, pp. 260-261. Besides discussing Adelson's paper, the authors present the autopsy of a six month old who was dropped on the floor by her mentally retarded six year old brother. The autopsy photograph (fig. 13.10) shows an extensive displaced parietal skull fracture with intracranial hemorrhage.

Sibling contribution to violent behavior. Gully KJ, Dengerink HA, Pepping M and Bergstrom D. Journal of Marriage and the Family 1981; 43: 333-337

Serious sibling abuse by preschool children. Rosenthal PA and Doherty MD. J Am Acad Child Psychiatry 1984; 23: 186-190. Cited in Giardino et al., A Practical Guide to the Evaluation of Child Physical Abuse & Neglect, 1997, p. 36. Included skull fractures, leg fractures, extensive bruising, lacerations, and stab wounds.

Kahn M and Lewis G, *Siblings in Therapy: Life Span and Clinical Issues.* New York: W W Norton, 1988

Perceived conflict and violence in childhood sibling relationships and later emotional adjustment. Graham-Bermann SA, Cutler SE, Litzenberger BW and Schwartz WE. J Fam Psychol 1994; 8: 85-97

Identification and trauma: an integrative-developmental approach. Caffaro JV. J Fam Vi 1995; 10: 23-40

Caffaro JV and Conn-Caffaro A, *Sibling Abuse Trauma: Assessment and Intervention with Children, Families, and Adults.* Binghamton: Haworth Treatment & Trauma Press, 1997

DRUG ABUSE

Maltreatment of children born to women who used cocaine during pregnancy: a population-based study. Leventhal JM et al, Pediatrics 1997. See under Cocaine Babies.

Incidence of passive exposure to crack/cocaine and clinical findings in infants seen in an outpatient service. Lustbader AS, Mayes LC, McGee BA, Jatlow P, Roberts WL. Pediatrics 1998; 102(1). URL: <http://www.pediatrics.org/cgi/content/full/102/1/e5>. Significant numbers of young children are passively exposed to such levels that it shows up in the urine. It is associated with respiratory symptoms. Commentary by Ira Chasnoff: Silent violence: is prevention a moral obligation? Pediatrics 1998 (below).

Silent violence: is prevention a moral obligation? Chasnoff I. Pediatrics 1998 Jul; 102(1): 145-148 Says that pediatricians should screen ALL families for drug & alcohol abuse; all positives should be referred to treatment. If fail treatment, parental rights should be terminated. Because a drug abusing environment is a toxic environment for children. He refers to the Adoption and Safe Families Act of 1997, which shifted emphasis from family reunification to safety of the child. "There is an indisputable connection between child abuse and substance abuse." 147

Onset of physical abuse and neglect: psychiatric, substance abuse, and social risk factors from prospective community data. Chaffin M, Kelleher K and Hollenberg J. Child Abuse & Neglect 1996; 20(3): 191-203. See under "Epidemiol" above.

Alcoholism See Rao et al., 2001, under "Neglect."

DOMESTIC VIOLENCE

Hotline 800-978-3600

<http://www.ncadv.org/>

See also "Battered Women's Defense," below.

Definition: Title 5 Sec. 13700 of the California Penal Code defines "domestic violence" and "abuse" as follows:

- 1) "Domestic violence" means abuse committed against an adult or a fully emancipated minor who is a spouse, former spouse, cohabitant, or person with whom the suspect has had a child or is having or has had a dating or engagement relationship."
- 2) "Abuse" means intentionally or recklessly causing or attempting to cause bodily injury, or placing another person in reasonable apprehension of imminent serious bodily injury to himself or herself or another."

County policy, as per County Department of Human Resources Policy no. 622

"Domestic violence/abuse may also involve a pattern of behaviors that may include physical, sexual, verbal, emotional, and psychological abuse and/or inappropriate economic control used in an attempt to exercise power and authority, which may have a destructive effect on individual victims, their families, and their communities. Domestic violence is a crime that can cause injury and death, endanger individuals and families, threaten society, and significantly increase health care costs. It is also a complex issue that crosses cultural, racial, ethnic, economic, gender, and political boundaries and can occur anywhere and at any time..."

Possible signs of domestic violence, as per County policy no. 622:

- Uncharacteristic changes in mood and behavior
- Decline in job performance
- Preoccupation
- Tardiness or absences
- Anxiety, hypervigilance
- Bruises
- Low Self-esteem
- Excuses for the above that don't make sense

Penal Code Sec. 1259.5 Domestic Violence Screening Act.

Penal Code Sec. 11160 and 11161.9 Domestic Violence Mandated Reporting

Penal Code Sec. 11161.2 Crime-Related Injury Reporting Act for Health Care Providers.

Penal Code Sec. 273 et seq. Spousal abuse.

Women and male violence; the visions and struggles of the battered women's movement. Schechter S. Boston: South End, 1982.

The co-occurrence of intimate partner violence against mothers and abuse of children. CDC National Center for Injury Prevention & Control, 1999. (have) <http://www.cdc.gov/ncipc/factsheets/dvcan.htm> This is a two-page factsheet with references, reviewing the four studies completed as of 1999. Appears to advocate "dual advocacy" approach for both the mother and her child. It fails to note the following issues: (1) maternal concealment of child abuse (2) the increased risk of violence involved in separating the household (3) the risk to healthcare workers entering the home (4) the needs of law enforcement. It gives additional resources, including the NCIPC child custody and protection website <http://www.vaw.umn.edu> and the Family Violence Prevention Fund, <http://www.fvpf.org> A much more elaborate presentation is made by Susan Schechter and Jeffrey Edleson, below.

In the best interest of women and children: a call for collaboration between child welfare and domestic violence constituencies. Susan Schechter and Jeffrey L. Edleson. Briefing paper. Minnesota Center Against Violence and Abuse electronic clearinghouse, 1994. <http://www.mincava.umn.edu> "About the sometimes tense relationship between child welfare workers and battered women's advocates. More importantly, however, it is about the great potential for collaboration between child welfare and battered women's services." Gives a paradigmatic case example of a women who took good care of her children but repeatedly returned to a man who was beating her and sometimes assaulted the children. The child protection staff (hospital social worker) was not comfortable returning the children to an unsafe home. But they had the unsettling feeling that by taking the children away they were punishing the mother for the behavior of the father. In the end, the children were taken away. But the battered women's advocate asked, "Was it really in the best interests of these children to offer so little protection to their mother?" And both sides wondered, "Can we really help or protect children if we ignore the abuse of their mothers?" Points out and substantiates by references to the literature that:

- (1) research suggests that domestic violence and child abuse frequently occur in the same family.
- (2) children who witness violence by their fathers may be at risk of developing problems.
- (3) men who abuse children also abuse women.
- (4) child protection and DV programs serve overlapping populations.

"Battered women clearly face great economic, social, and safety hurdles when attempting to leave a violent partner. The decision to leave or stay often hinges on the mother's assessment of what will be in the best interests of her children." Supporting the mother-child unit would be a common ground. [The authors do not expressly say this, but what they are saying is that the mother-child unit should be separated from the violent father, rather than the child separated from both parents. Since many women are unable to separate themselves from the violent father, the government should assist them in separating. Reunion of the children with their father should no longer be a consideration. The authors imply, but do not state, that this modality should be written into legislation or at least into the standards of practice in child protection agencies. -JKR]

Cites a seminal study on wife-beating, Schechter, 1982.

Estimation of stress in child neglect from thymic involution. Tanegashima A, Yamamoto H, Yada I, Fukunaga T. [unknown journal] 1999; 101(1): 55-63.

Identified spouse abuse as a risk factor for child abuse. Rumm PD, Cummings P, Krauss MR, Bell MA, Rivara FP. Child abuse & neglect 2000; 24: 1375-1381. Military. Spousal abuse was a strong predictor of child physical abuse (2.36), not so strong for sexual abuse (1.46) or neglect (0.96.)

The relative importance of wife abuse as a risk factor for violence against children. Tajima EA. Child Abuse & Neglect 2000; 24: 1383-1398. National Family Violence Survey, a telephone survey of 2,700 households. The likelihood of physical child abuse increased by 1.69 independent of other factors; but wife abuse was a small contributor to the incidence of child abuse, compared to multiple other factors.

Domestic violence: the shaken adult syndrome. Carrigan TD, Walekr E, Barnes S. J Accid Emerg Med 2000 Mar; 17(2): 138-139. One adult female with grip marks, SDH, RH.

Pregnancy no deterrent to violence. News story from JAMA, reported March 28, 2001. Yahoo! News, Health Headlines. (Copyright HealthSCOUT) Reporting on a UNC study by Sandra Martin, associate professor in the Department of Maternal and Child Health at Chapel Hill. Surveyed 3500 women before, during and after pregnancy. Six percent (6%) said they were abused during pregnancy and three percent (3%) said they had been abused in the puerperium. They did not report this to their infant's pediatrician, even though they took their infant in for well-baby care, and they were not asked about it. 75% of those who were abused said they suffered injuries from it, but only 1/4 of them sought treatment for the injuries.

Type and timing of mothers' victimization: effects on mothers and children. Dubowitz H, Black MM, Kerr MA et al. Pediatrics 2001; 107: 728-735. A clinic-based questionnaire and psychometric study of 419 mothers, measured when their index child was 4 and again at 6. Recruited from a high-risk neonate clinic and a FTT clinic. 50% reported DV. Externalization behavior in the mothers and internalization behavior in the children.

Family Abuse: Consequences, Theories, and Responses. Sylvia I. Mignon, Calvin J. Larson, William M. Holmes. Boston: Allyn and Bacon, 2002. ISBN 0-205-29569-X. My thanks to Dr. Susan M. Larsen of Cal State Fullerton for this current college-level textbook emphasizing typology of all forms of intrafamilial abuse including elder abuse.

The "Medea complex" among men: the instrumental abuse of children to injure wives. McCloskey LA. Violence Vict 2001; 16(1): 19-37. "Children of battered women stood a 42% chance of receiving escalated abuse from their fathers. It is proposed that men's abuse of children is in many instances instrumental in order to coerce or retaliate against women, echoing the Greek myth of Medea who killed her own children to spite their father." Heavy drinking (odds ratio 4.86) and life stress events (odds ratio 1.6) predicted men's abuse of their partners, but these risk factors were unrelated to child abuse. Wife battering, however, placed children at heightened risk (2.77).

The co-occurrence of child maltreatment and domestic violence: examining both neglect and child physical abuse. Hartley CC. Child Maltreatment 2002 Nov; 7(4): 349-358. Research reports a substantial overlap, but few studies characterize these families. This study compares families with both child battering and woman battering to families with only child battering. Finds a significant association between the following factors:

- marital status
- man's biol rel to the child
- mother is perpetrator

Strategies for handling cases where children witness domestic violence. Allison Turkel and Christina Shaw. APRI Update, 2003; vol 16, no. 2. <http://www.ndaa-apri.org> In some states, allowing a child to witness DV is child endangerment. Woman can be arrested for that. One federal court (apparently the Southern District) has held that such a provision violates due process. *In re Nicholson*, 2001 U.S. Dist LEXIS 22322. The present article argues that conflict of objectives between child protection and woman protection should be minimal if agencies work together. Article says that domestic vi advocates have several fears:

- that the child protection agency will automatically remove the children from a home where there is DV (thus penalizing the woman). Citing *Nicholson v. Williams*, 203 F. Supp. 2d 153 (aka *Nicholson v. Scopetta*).

- that women will be charged with child endangerment, as noted above

- there will be sentencing enhancements if children were present during the DV

-- that women will not cooperate with prosecution of the batterer if they fear that successful prosecution will result in the removal of the children or the permanent removal of their domestic partner.

-- batterers will use the child protection system as a sword against the woman. If you pursue this, your children will be taken away, you will have no home.

Child protection advocates have several fears:

-- children in DV homes are at risk for emotional damage and physical abuse

-- sentencing enhancements will not be imposed and thus domestic abusers will go less punished.

Solutions to these fears include that sentencing for DV should always include long term counselling for all parties including the batterer. The fact is that these families generally reunite (after the imprisonment). Provision should be made for that in the original court disposition and by the involved agencies. Coordination is required. The article holds up the San Diego Family Justice Center as a model. <http://www.familyjusticecenter.org/main.htm>. See also the Child Witness to Violence Project in Boston, <http://www.bostonchildhealth.org/>

Building Bridges Between Domestic Violence Organizations and Child protective Services. Spears L. 2000.

Mitchell C. Is there a link between child maltreatment and woman battering? In: *Child Abuse and Neglect: Guidelines for Identification, Assessment, and Case Management.* Marilyn Strachan Peterson and Michael Durfee, eds. Volcano, California: Volcano Press, 2003. 370 pp. ISBN 1-884244-21-1. (have). Chapters updates: <http://www.volcanopress.com/>. Includes being forced to watch vi, being taken hostage to coerce the mother, being hit in mother's arms, being used as a physical weapon, used as a spy, or being involved in the aftermath as having to summon assistance or provide care. Says that the Walker study of 1984 reported that 53% of the women reported that their abuser had also abused their children and 28% of the women themselves had abused their children. Suh and Abel, 1990 found that 40% of the women reported that their spouse also physically abused their children. Straus and Gelles, 1990 by a telephone survey found that 50% of the fathers who frequently beat their wives also frequently abused their children. "The data appears to establish a clear link..." (p. 138) Also frequently injured while intervening. Psychological injury from witnessing: Edleson, 1999, exhibit more aggressive and antisocial behavior (externalized) as well as fearful and inhibited behavior (internalized). Anxiety, self-esteem. Kilpatrick, 1997 four mod to severe sx of PTSD in 85%, plus blunted empathy. Silvern, 1995 fd developmental problems. Singer, Miller, Guo et al., 1998 found exposure to DV was a predictor of subsequent violent behavior by the child itself. The author gives a chart of effects including premature labor, low birth weight, on into infancy, preschool, school age, and teenage consequences. Advises screening for DV in "all children" as well as abused and neglected children. (p. 140)

California Medical Training Center. UC Davis Medical center. <http://www.ucdmc.ucdavis.edu/medtrng> Includes OCJP 502 Forensic Medical Report: Domestic Violence Examination, mandated by SB 502 (2003).

Family Violence Prevention Fund. <http://www.endabuse.org> States that the US Advisory Board on Child Abuse considers that domestic violence is the major precursor to child abuse & neglect fatalities.

The Greenbook. <http://www.thegreenbook.info> This is a project of the National Council of Juvenile and Family Court Judges. Trying to pull an end run around the dichotomy between child protection and protection of women by defining the witnessing of DV as a form of child abuse (by the male partner). As a result, one can avoid what they describe as "the worst case," where "the children are taken from the battered mother who is blamed for allowing the children to be exposed to violence." See also the Child Witness to Violence Project, <http://www.bmc.org/pediatrics/special/CWTV/OVERVIEW.HTML>

The National Domestic Violence Hotline. 800-799-SAFE. <http://www.ndvh.org>

Violence Against Women online resources. <http://www.vaw.umn.edu>

California Family Violence Referral Directory <http://www.dhs.ca.gov/epic/documents/dvrefer.pdf>

Behaviors of children who are exposed and not exposed to intimate partner violence: an analysis of 330 black, white, and Hispanic children. MacFarlane JM, Groff JY, O'Brien JA, Watson K. Pediatrics 2003; 112: e202-e207. Studied 258 children exposed to DV against their mothers and 72 age- and race-matched control children. Got behavior scores from a tool called the Child Behavior Checklist. Found that there was no statistically significant difference between exposed and nonexposed children below 6 years of age. But above 6 years there were significant differences in their behavior scores. This supports routine screening of mothers for DV during well-child visits, as recommended by the AAP.

Injury patterns among female trauma patients: recognizing intentional injury. Crandall ML, Nathens AB, Rivara FP. J Trauma 2004 Jul; 57(1): 42-45. Women who suffered blunt intentional trauma exhibited very different injury patterns than those hospitalized for motor vehicle collisions or falls. The odds ratio of **facial injury** was 4.9, head injury was 1.4.

Recognising and responding to partner abuse: challenging the key facts. Goodyear-Smith F. NZ Med J 2004 Sep 24; 117(1202): U1074. This dissident critic of the abuse establishment here challenges the figure of NZ\$ 141 million conventionally quoted as the annual social cost of family violence.

Screening children for family violence: a review of the evidence for the US Preventive Services Task Force. Nygren P, Nelson H, Klein J. Ann Fam Med 2004; 2(2): 161-169. A literature review found that no trials of the effectiveness of screening in a health care setting have ever been published.

A protocol to diagnose intimate partner violence in the emergency department. Halpern LR, Perciaccante VJ, Hayes C, Susarla S, Dodson TB. J Trauma 2006; 60: 1101-1105. large study statistically establishes a high index of suspicion when women present with facial injuries in the absence of a history of an MVA.

BATTERED WOMEN'S DEFENSE

Defense to child abuse charges against the mother (or charges of concealing or aiding and abetting child abuse).

Battered Woman's Syndrome: A defense to child abuse? Dawn Doran Wilsey, APRI Update 2006 Aug; 19(3). Lenore Walker defined BWS as "a state of learned helplessness" -- learned from the failure and futility of past attempts at escape. Legally this is the defense of Duress. Elements of Duress: reasonableness, imminent danger, and lack of opportunity to escape. Imminence may be a problem because there is commonly a lapse of time between the most recent threat and the incident of child abuse and concealment. But BWS may persuade jurors that the mother reasonably perceived the harm as imminent. The cycle of violence aspect of DV may be used to establish inability to escape: learned helplessness, the futility of past attempts at escape. Says courts have been reluctant to accept BWS as a defense to child abuse, because it is universally accepted that both parents have a clear duty to protect their children. Note that BWS has been asserted as a defense to other crimes, not just child abuse. Embezzlement -- defense was allowed in NJ. Drug offense -- defense was allowed US. Author comments that "Attacking BWS as a defense may place child abuse prosecutors at odds with DV advocates and prosecutors, who may strenuously object to the pros of any battered woman for her actions while she is in an abusive rel." "Each case should be considered on its own merits." Suggests specific inquiries: If the mother had protected the child or called for help, would she in fact have faced imminent harm? Did she ever seek outside aid? Did she work outside the home, or was she a domestic prisoner? Did she ever report abuse of herself? Did she have plenty of food and resources for herself but fail to provide any for the child? Was there an instance

where child protection officials contacted her and she denied that the child was being abused? Did she choose her own welfare over that of the child?

PARENTAL RIGHTS

See Chasnoff, above, under "Drug Abuse," arguing for termination of parental rights of drug abusers.

Adoption and Safe Families Act of 1997. (ASFA) Within one year of child's entry into the adjudication system, the court must decide what the final placement goal will be -- family reunification or out-of-home placement. No later than 15 months post entry, a Termination of Parental Rights petition must be filed if the goal is not reunification.

Toward a reorientation of values and practice in child welfare. Onheiber MD. Child Psychiatry and Human Development 1997 Spring; 27(3): 151-164. The failure of the system stems from overemphasizing parental rights. We need (1) parental licensure, and (2) economic policies favoring parental commitment to children.

Ethical issues in professionals' response to child maltreatment. Howard Dubowitz. Child Maltreatment 1997 Nov; 2(4): 348-355. See below under "Unclassifiable." Discusses the sanctity of the family versus the invasiveness of the CPS, ethical conflicts in reporting possible abuse to incompetent agencies.

Troxel et vir. v. Granville, 000 U.S. 99 (June 5, 2000). Supreme court case holding that under substantive due process (XIV) the state can interfere with a fit parent's decisions in childrearing only after a threshold showing of harm to the child. Parents' fundamental right to rear their children. In this case it was a visitation dispute between the grandparents and the child's mother. Wash Stat 26.10.160(3) permits "any person" to petition the custody court for mandatory visitation rights and they can be granted if the court finds merely that it is "in the best interests of the child." The S Ct holds that this obviously overbroad and poorly drafted statute is an unconst infringement of the "fundamental right" of fit parents to control their children's upbringing. The state cannot interfere with the parents' tutelage unless it makes a threshold showing either that the parent is unfit or that there is harm to the child. Affirming the Washington Supreme Court's invalidation of its own statute as overbroad on due process grounds. This was a 6-3 decision, with Scalia, Stevens, and Kennedy, JJ dissenting. The full text is found at <http://caselaw.findlaw.com/>

See Jones, Finkelhor on declining sexual abuse substantiations, 2001, under "Sexual abuse." Increased legal rights for caregivers as a possible reason for declining substantiations.

PARENTAL ALIENATION SYNDROME

Recent trends in divorce and custody litigation. Gardner RA. The Academy Forum 1985; 29(2): 3-7. Cited by Prof. Gardner in his 1992 monograph (below) as the original introduction of the term.

The parental alienation syndrome. Richard A. Gardner, MD., (1992) (self-published monograph). See APRI, 2003, below.

The parental alienation syndrome: what is it and what data support it? Faller KC. Child Maltreatment 1998 May; 3(2). Refutes Dr Gardner's theory that allegations of sexual abuse in a litigation context are usually false. Research shows that only 5% to 8% are false.

Medicolegal aspects of child abuse. John E. B. Myers. Chapter 21 in Reece & Ludwig, eds., *Child Abuse: Medical Diagnosis and Management*, 2d ed (2001), at p. 559. Says the term is not useful. Gives a discussion. Sometimes a contesting parent's accusation is true; sometimes it is false. "In the final

analysis, everyone would be better off to discontinue the use of the term PAS, and to evaluate accusations of abuse on their individual merits.”

Reece & Ludwig, 2d ed. (2001), p. 559: One parent programs the child to hate the other parent; accusations of abuse by the parent or by the child or both occur as part of the syndrome. Usually during divorce and custody proceedings. The term is used by lawyers to assail the credibility of the accusing parent or child. Author John E. B. Myers concludes, “[E]veryone would be better off to discontinue use of the term PAS, and to evaluate accusations of abuse on their individual merits.”

Does the DSM-IV have equivalents for the parental alienation syndrome (PAS) diagnosis? Gardner RA. Am J Fam Ther 2002; 31(1): 1-21. <http://www.rgardner.com/refs/ar12.html> “A disorder in which one parent alienates the child against the other parent.” “In this disorder we see not only programming (brainwashing) of the child by one parent to denigrate the other parent, but self-created contributions by the child in support of the alienating parent’s campaign of denigration against the alienated parent.” Finds that attorneys for the alienating parent resist the testimonial use of the term “PAS.” Finds that substitute terms exist in DSM-IV.

Parental alienation syndrome: what professionals need to know. Part 1 of 2. Ragland ER and Fields H. APRI Update 2003; vol. 16, no. 6. The authors quote from the original description by the late Dr Richard Gardner, clinical professor of psychiatry at Columbia University, who wrote,

“[T]he parental alienation syndrome is a childhood disorder that arises almost exclusively in the context of child-custody disputes. Its primary manifestation is the child’s campaign of denigration against a parent, a campaign that has no justification. It results from the combination of a programming (brainwashing) parent’s indoctrinations and the child’s own contributions to the vilification of the target parent. When true parental abuse and/or neglect is present, the child’s animosity may be justified and so the parental alienation syndrome explanation for the child’s hostility is not appropriate.” (see reference above)

The present APRI article espouses the proposition that the parental alienation syndrome is not and will not become a clinically recognized entity; the authors advocate that it should not be included in the upcoming DSM-V. Their arguments are (1) The syndrome presupposes a high rate of false accusations (of sexual abuse) in the context of custody disputes; (2) It was not submitted to peer review, but was self-published by Dr Gardner; (3) It consists of anecdotal observations from Dr Gardner’s practice. (Dr Gardner is a professor in the Department of Child Psychiatry at P&S).

Parental alienation syndrome: what professionals need to know. Part 2 of 2. Fields H and Ragland ER. APRI Update vol 16, no. 7, 2003. Judicial approaches to PAS. It is viewed differently by criminal and civil courts. Criminal case law: State v. Koelling, 1995 Ohio App. LEXIS 1056 allowed evidence of PAS in a sexual abuse case. New York courts have consistently refused to admit PAS evidence in criminal cases. One was on *Frye* grounds, that it was not “generally accepted.” California Ct App in an unpublished opinion 2003 held that D was not deprived of DP when he was not allowed to call a PAS expert. People v. Sullivan, 2003 Cal. App. Unpub. LEXIS 3316, partly on *Kelly-Frye* grounds of not scientific enough. Civil case law is more accepting. PAS ev shd be challenged on *Daubert* grounds. Also procedural grounds.

WATER INTOXICATION

see also under RH -- seizures

Salt poisoning: presenting symptom of child abuse. Feldman K, Robertson WO. Vet Hum Toxicol 1979 Oct; 21(5): 341-343. Describes two young children with severe hypernatremia caused by salt administration combined with water deprivation. Obviously this is not water intoxication, but I just threw it in.

Hyponatremic seizures as a presenting symptom of child abuse. Tillelli JA, Ophoven JP. FSI 1986; 30(2,3): 213-217

Child abuse by drowning. Griest KJ and Zumwalt RE. Pediatrics 1989 Jan; 83(1): 41-46. (See above under "Drowning.") Case 2 was a 3 y o girl found on the couch by params who had been summoned by a concerned neighbor. Her clothing was wet and her hands and feet were waterlogged. Topsy showed contusions of the lips and face, prominent oculofacial petechiae, cerebral and pulmonary edema. Vitreous Na 119, CL 105. The grandmother was a religious fanatic who cleansed the devil by forcibly pouring water down the girl's throat. At the time of the params' arrival, the grandmother and several other women were found sitting nude on the floor of the next room, chanting and swaying.

Oral water intoxication in infants: an American epidemic. Keating JP, Schears GJ, Dodge PR. AJDC 1991; 145: 985-990. Poverty infants fed thin formula due to economic deprivation presented with sudden-onset seizures and respiratory failure. Responded to hypertonic saline.

Fatal child abuse by forced water intoxication. Arieff AI and Kronlund BA. Pediatrics 1999 Jun; 103: 1295-1297. Reports three cases aged 16, 7, and 6 who were forced to drink 3 to 6 liters of water as punishment, leading to fatal hyponatremic coma: the symptoms were emesis and fecal incontinence and seizures. Sociums of 109-114. Cerebral edema with herniation and wm petechiae, pulmonary edema, aspiration pneumonia. All had multiple soft-tissue abuse as well. Episodes were repetitive. This is a newly reported syndrome. Two previous cases of forced water intox in children were reported, but were nonfatal and associated physical abuse not documented.

Altered consciousness and shock in a malnourished child. Gingold MK, Rajakumar K, Rosas AJ. Semin Pediatr Neurol 1996 Sep; 3(3): 248-254. Case of starvation and abuse involving central pontine myelinolysis.

Hypernatremia and subdural hematoma in the pediatric age group: is there a causal relationship? Handy TC, Hanzlick R, Shields LBE, Reichard R, Goudy S. AJFS 1999 Nov; 44(6): 1114-1118. No there is no causal relationship. Hypernatremia is a complication seen in critically ill children from any cause. Citing references on electrolyte disorders, including homicidal salt poisoning, cerebral edema, dehydration.

Effects on the central nervous system of hypernatremic and hyponatremic states. Arieff AI, Guisado R. Kidney International 1976; 10: 104-116. Infants with acute hypernatremia: irritability, lethargy, fever, vomiting, respiratory distress, shrill cry. Severe hypern: LOC, hyperreflexia, twitching, rigidity, sz, coma.

Hyponatremic seizures in infancy: association with physical child abuse. Krugman SD, Zorc JS, Walker AR. Pediatric Emergency Care 2000; 16: 432-434. One shaken baby with sz and Na 122, one water intoxicated baby with sz and Na 116. Both had RH, the latter quite severe.

Retinal hemorrhages in infants with hyponatremic seizures. Rubin D, Christian C. Pediatric Emergency Care 2001; 17: 313-314. Here Cindy Christian and her colleague present the case of a six month old with posterior-pole RH in the presence of Na 116 sz with no trauma.

COUGH MEDICINE

AAP Policy Statement: Use of codeine- and dextromethorphan-containing cough remedies in children. AAP Committee on Drugs. Pediatrics 1997; 99: 918-920. Here are its conclusions:

1. No well-controlled scientific studies were found that support the efficacy and safety of narcotics (including codeine) or dextromethorphan as antitussives in children. Indications for their use in children have not been established.
2. Suppression of cough in many pulmonary airway diseases may be hazardous and contraindicated. Cough due to acute viral airway infections is short-lived and may be treated with fluids and humidity.

3. Dosage guidelines for cough and cold mixtures are extrapolated from adult data and clinical experience, and thus are imprecise for children. Adverse effects and overdosage associated with administration of cough and cold preparations in children are reported. Further research on dosage, safety, and efficacy of these preparations needs to be done in children.
4. Education of patients and parents about the lack of proven antitussive effects and the potential risks of these products is needed.

As to point (2) above, the statement says that “In some pathologic states (e.g. asthma, bronchopulmonary dysplasia, cystic fibrosis, and a variety of inflammatory conditions), excessive and/or abnormal airway secretions may be produced. The cough reflex serves to maintain airway patency by clearing these secretions. ... Cough suppression may adversely affect patients with these conditions by promoting pooling of secretions, airway obstruction, secondary infection, and hypoxemia.” Discusses the chemical agents: notes that many cough products are elixirs that may contain up to 25% alcohol(!) Taylor’s study showed that these were not any more effective than placebo in children. As to toxicity, says that decongestant sympathomimetic amines in these have been associated with irritability, restlessness, lethargy, hallucination, hypertension, and dystonia. The clearance mechanism may vary with age (citing Kearns et al., 1989). “The relative immaturity of hepatic enzyme systems that metabolize drugs in young children may enhance the risk of adverse effects of such medications, especially in infants younger than 6 months. metabolism and/or toxicity may be altered by concurrent use of medications such as acetaminophen. Unfortunately, the dosing guidelines for these agents are based on extrapolation from adult data...” Also, there is enterohepatic recirculation. Now as to codeine specifically, says that in adults it does suppress cough in a dose-related fashion (as does dextromethorphan), but at no dose was cough completely suppressed. There are no studies establishing the dose in children. Codeine toxicity: respiratory depression and obtundation. In children, somnolence, ataxia, miosis, vomiting, rash, facial swelling, and pruritus. Respiratory depression occurred in 3% of children receiving doses greater than 5mg/kg/d and two of these patients died. (Citing von Muehlendahl et al., Codeine intoxication in childhood. *Lancet* 1976; 2: 303-305). “The hepatic glucuronidation pathway is incompletely developed in infants, which places them at particular risk for adverse dose-related effects...” Now as to dextromethorphan, it is chemically derived from opiates but has no analgesic or addictive properties. In adults, dosages of equal antitussive potency to codeine produce comparable levels of CNS depression.

This statement was reaffirmed by the Academy as of October, 2006; see the announcement at page 405 of the February 2007 issue of *Pediatrics*.

Infant deaths associated with cough and cold medications: two states, 2005. MMWR Jan 12, 2007/ 56(01);1-4. In one year, 1,519 children under two years were treated in US emergency departments for adverse events, including overdoses, associated with cough and cold medications. (Estimated from the NEISS). Therefore, the CDC joined with NAME to investigate deaths of US infants associated with these medications. Found three infants in 2005, all under 6 months. “The dosages at which cough and cold medications can cause illness or death in chil aged <2 yrs are not known.” There are no FDA recommended doses for that age group.

The three fatalities identified ranged from 1 mo to 6 mo. Their postmortem blood pseudophedrine levels were 4,743 ng/ml, 6,832 ng/ml, and 7,100 ng/ml. Cases 1 and 2 were negative for antihistamines. Cases 2 and 3 had dextromethorphan levels of 1,909 and 390 ng/ml respectively. The CODs were

Case 1	Pseudophedrine intox	OC: interst pn, recent hosp for fever
Case 2	Pseudophedrine and dextromethorphan intox	OC brpn + empyema
Case 3	Drug poisoning	OC: prem, fx tibia, acute anoxic enc

“Because of the unproven efficacy of the cough suppressant codeine and dextrom in young child and the potential for adverse events, in 1997 the AAP issued a policy statement advising that parents should be educated regarding the lack of antitussive effects, risk for adverse events, and potential for overdose in child from these medications. In 2006, the American College of Chest Physicians released clinical practice guidelines for management of cough, advising health-care providers to refrain from recommending cough suppressants and other over-the-counter cough medications for young child because of associated morbidity and

mortality.” The FDA has stopped the manufacture of carbinoxamine (an antihistamine)... Pseudoephedrine has been removed bc it can be used in the manuf of methamph.

“Few data exist regarding the therapeutic or toxic levels of cough and cold medications in children aged <2years. (citing Gunn et al., 2001; Marinetti et al., 2005, and PedTox registry, Am J Forens Med P 2005). Blood levels of cough and cold medications revealed in postmortem studies might not reflect levels in the bloodstream at the time of administration. However, in this report, the blood levels of pseudoeph found ... were approx 9 to 14 times the levels resulting from administration of recommended doses to children aged 2 – 12 years.”

Comment by JKR: The authors admit that there are no established standards for diagnosing this type of intoxication in infants, and that they simply accepted the individual medical examiners’ say-so for the cause of death. Accordingly, this report must be considered anecdotal.

STICKLER'S SYNDROME (not child abuse)

Stickler's syndrome is a genetic disorder which affects connective tissues, including joints, eyes, palate, heart, and hearing. Visual problems, hearing loss, arthritis, cleft palate, heart murmurs, and TRACHEOMALACIA and retinal detachment, Pierre-Robin syndrome, reduced muscle tone, loose joints, enlarged bone ends, mitral prolapse.

Because the presentation is highly variable, diagnosis is difficult. See paper in file under SUDS. The COL2A1 gene mapped to chromosome 12, and the COL11A1 gene mapped to chromosome 6. Discovered at the Mayo Clinic in 1965.

Case 98-8511 or 5811 Dr. Wang

Stickler Involved People
15 Angelina
Augusta, Kansas 67010
316-775-2993

For genetic testing (\$2,400)
Send EDTA blood to
Matrix DNA Diagnostics
MCP Hahnemann University
10310 NCB, Mail Stop 421
245 North 15th Street
Philadelphia, PA 19102-1192

SANDIFER'S SYNDROME (not child abuse)

Torticollis or seizurelike dystonic posturing associated with GERD. May be mistaken for seizures, with resulting erroneous prescription of antiepileptic drugs. Actually it is simply a manifestation of GERD, often seen in neurologically impaired children, especially cerebral palsy. It resolves with management of the GERD.

Sandifer's syndrome in a child with asthma and cerebral palsy. De Ybarrondo L, Mazur L. Southern Med J 2000; 93(10): 1019-1021. (have).

HANDICAPPED CHILDREN

Child abuse and disabilities: a medical perspective. Ann S. Botash. The APSAC Advisor, Spring 1999; 12(1): 10-18, with refs.

Maltreatment in psychiatrically hospitalized children and adolescents with developmental disabilities: prevalence and correlates. Ammerman RT, Hersen M, et al.. J Am Acad Child & Adol Psychiatr 1994; 33(4): 567-576

FOSTER CHILD

Intensive intervention for maltreated infants and toddlers in foster care. Zeanah CH and Larrieu JA. Child Adolescent Psych Clin N Amer 1998 Apr; 7(2): 357-371

Starting young: improving the health and developmental outcomes of infants and toddlers in the child welfare system. Silver J et al. Child Welfare 1999 Jan feb; 78(1): 148-165

Factors Associated with Child Maltreatment by Family Foster Care Providers. Mary I. Benedict and Susan Zuravin. Baltimore: Johns Hopkins University School of Hygiene and Public Health, June 30, 1992.

Out of home maltreatment: an analysis of risk in various settings for children. Spencer JW and Knudsen DD. Children and Youth Services Review 1992; 14: 485-492.

Lee Ann Stephenson, State of Wyoming DFS, Children's Services Unit. Personal communication Child-Fatal Oct 1999. States work very hard to provide (to the Federal Government) this information to NCANDS, and it appears in their reports. Copies of the NCANDS report can be obtained from National Clearinghouse on Child Abuse and Neglect, 330 C Street SW, 20447. 800-FYI-3366.
<http://www.calib.com/nccanch>.

National Coalition for Child Protection Reform <http://www.nccpr.org> An advocacy group that opposes arbitrary placement in foster homes and prefers family preservation. "We believe that the problem is not that there are too few foster parents; rather, there are too many foster children. By that we mean that the system is overwhelmed with children who do not need to be there. They could safely remain in their own homes if proper services were provided." The countervailing view favors so-called "therapeutic" foster care. As to this, see Reddy and Pfeiffer, below, which argues that therapeutic foster care is better than in-home care:

Effectiveness of treatment foster care: a review of outcome studies. Reddy and Pfeiffer, Journal of the Academy of Child and Adolescent Psychiatry 1997 May; 36: 5

Foster care, an update. Rosenfeld et al. Journal of the Academy of Child and Adolescent Psychiatry 1997 Apr; 36: 4. The current foster care system is seriously underfunded and requires major reform

Abuse of children in foster and residential care. Hobbs GF, Hobbs CJ, Wynne JM. Child Abuse & Neglect 1999; 23: 1239-1252. (From Leeds). Concludes that children in foster care are more often abused than children in the general population. In great detail.

LONG TERM SEQUELAE

Prevalence of post-traumatic stress disorder and other psychiatric diagnoses in three groups of abused children (sexual, physical, and both). Ackerman PT, Newton JE, McPherson WB, Jones JG, Dykman RA. Child Abuse & Neglect 1998; 22(8): 759-774. One third of the victims had PTSD, often comorbid with other affective disorders.

Peer relationships and self-esteem among children who have been maltreated. Bolger KE et al. Child Dev 1998; 69(4): 1171-1197. Severity of disturbance correlated with the severity and duration of abuse.

Inflicted traumatic brain injury: relationship of developmental outcome to severity of injury. Ewing-Cobbs et al. Pediatr Neurol 1999; 31: 251-258. Prospective study of 28 pts with a control group of 28 with no injuries, matched for age race, birth history, and class. Followed out to three months post-admission. The inflicted TBI group scored lower on the Bayley Scale and the Glasgow Outcome Scale. The acute conditions most predictive of poor outcome were cerebral edema and infarction.

EEG abnormalities in survivors. Dr. Martin H. Teicher of McLean Hospital in Belmont, Mass, *Cerebrum* 2000; 2: 50-67, quoted in Reuters Medical News Dec 21 2000 on Medscape, that left-sided EEG abn, rare in normals, were present in 72% of survivors of physical or sexual abuse. EEG sh diminished devel of the L temporal lobe. Other inv hav obt MRI ev that the L hippo of abused pts is smaller than that of controls. Also reduction in the size of the mid corpus callosum. Anxiety dso.

Childhood abuse, family dysfunction strongly associated with suicide attempts. Reuters Medical News on Medscape, Dec. 25, 2001. Citing work by Dube et al in JAMA for Dec. 26th to the effect that adverse childhood events ranging from abuse to incarceration of a family member strongly affect the risk of suicide attempts later in life. (This seems so obvious as to be nearly platitudinous.) Such adverse childhood events found to be associated included frequent emotional abuse, frequent physical abuse, any instance of sexual abuse, domestic vi, drug or alcohol abuse, mental illness in the family, separation and divorce, or incarceration of a family member. Says the lifetime risk of suicide attempt among the studied at-risk cohort was 3.8%, compared to ? among nonexposed controls. The risk went up along with the amount of the exposure. Used a score to measure the amount of adverse event exposure.

Scars that won't heal: the neurobiology of child abuse. Teicher MH. (McLean Hospital, Boston). Scientific American 2002 Mar; 286(3): 68-75.

Long term effects of childhood abuse on brain and neurobiology. Bremner JD. (Emory) Child and Adolescent Psychiatry Clinics of North America 2003 Apr; 12(2): 271-292. Early stress affects the HPA axis and norepinephrine systems. Also benzodiaz, op, dop, neuropeptide systems. These modulate the hippo, amygdala, and prefrontal cortex. Hypothesized to cause PTSD, depression, and other psychiatric symptoms.

The neurobiology of childhood trauma and abuse. van der Kolk BA. Child and Adolescent Psychiatry Clinics of North America 2003 Apr; 12(2): 293-317. Trauma-induced alterations in threat-perception. In responding to threats or stress, they tend to recreate the past instead of reacting rationally.

The Quarterly Update (Child Abuse Quarterly): reviews of current child abuse medical research. Spring Issue 2004. Multiple articles reviewed on long-term consequences.

MEDLINE abstracts: violence against women -- consequences of child abuse. A literature collection created by MedScape section on Ob/Gyn & women's health, 2003.
<http://www.medscape.com/viewarticle/463965>

The legacy of child maltreatment: long-term health consequences for women. Arias I. J Women's Health 2004; 13(5): 468-473. <http://www.medscape.com/viewarticle/482931> Recites that the Administration on Children, Youth, and Families estimated that over 903,000 children (12.4/1000) were victims of some form of maltreatment including neglect and psychological maltr. The estimated rates were:

neglect (including medical neglect)	59% of reported cases of maltr
physical abuse	18% “
sexual abuse	10% “
psychological abuse	7% “

Says that in 2001, approximately 1300 children died as a result of abuse & neglect (citing US Department of Health and Human Services, Administration on Children, Youth, and Families. *Child Maltreatment*, 2001. Washington, DC: US GPO, 2003) “Children who are maltreated exhibit problematic school performance, attention deficits, poor social skills, and physical aggression.” (Citing National Research Council. *Understanding child abuse and neglect*. Washington, DC: National Academy Press, 1993.) Girls are more likely to internalize, while boys are more likely to externalize. Adult survivors have similar problems. Psychological consequences among women include PTSD, depression, anxiety, somatization, substance abuse, eating disorders, personality disorders, and suicidal behavior. Poor social and academic

outcomes. Unwanted first pregnancy.

Cognitive and neuroimaging findings in physically abused preschoolers. Prasad MR, Kramer LA, Ewing-Cobbs L. Arch Dis Chil 2005 Jan; 90(1): 82-85. Did cognitive, language, and motor testing on 19 children who had been hospitalized for physical abuse (without neurological injury) and 19 age- and class-matched controls. Results: The abused children scored significantly lower in all three areas. Conclusion: Physically abused children are at high risk for delays at the preschool level.

MEDICOLEGAL (see also “Shaken” and “Unclassifiable”)

David Chadwick and Henry F. Krous, Irresponsible medical testimony by medical experts in cases involving the physical abuse and neglect of children. 2 (4) Child Maltr 313-321 (1997).

Paul Stern, Preparing and Presenting Expert Testimony in Child Abuse Litigation: A Guide for Expert Witnesses and Attorneys. Thousand Oaks: Sage, 1997.

State v. Schumpert, 435 SE2d 390 (S.C. 1993); *Commonwealth v. Azar*, 588 NE2d 1352 (Mass 1992); *State v. Dumlao*, 491 A2d 404 (Conn 1985). The law permits medical experts in child abuse cases to offer an opinion on the ultimate issue.

Margaret A. Berger, Procedural paradigms for applying the Daubert test. 78 Minn L Rev 1345 (1994)

F. R. Ev. 104(a). *Daubert v. Merrell Dow Pharmaceuticals*, 113 S.Ct. 2786, 125 L.Ed.2d 469 (1993); *Kumho Tire Co. v. Carmichael*, 119 S.Ct. 1167 (1999).

Investigation and review of unexpected infant and child deaths. AAP policy statement RE9921. Pediatrics 1999 Nov; 104(5): 1158-1160. (have) “Adequate death investigation requires the participation of numerous persons....” Includes autopsy, invest circs, rev of medical and family history, rev of info rfrom relevant agencyies and health care profs. Circs includes a scene invest and interivews. Interagency coop is necessary. All medical records from birth on.

Tips for investigating child fatalities. Devon Lee, James May, Erin O’Keefe. APRI Update, vol.13, no. 1, 2000.
1. Identify and interview every possible witness., including anyone who had contact with the victim’s family, hospital receptionists, EMTs. 911 dispatchers, patrolmen. 2. Speak with everyone who recently saw the victim or who knew him. 3. Preserve the crime scene and search it as soon as possible; children’s bodies, unlike adults, are often moved and cleaned. 4. Collect the child’s clothes and bedding and the caregivers’ clothes, plus the instrumentality of death (chair), food containers, search hampers, trash. 5. Look for stress triggers such as soiled clothes, spilled food, ev of financial or marital stress. 6. Create a time line. This can substantiate delayed rescue and time of injury, and contains possible revisions to the caregivers’ story.

In re Ethan H, 135 NH 681, 609 A2d 1222 (1992); *Petition of Doe*, 126 NH 719, 495 A2d 1293 (1985); *Tracey Hildreth v. Iowa Department of Human Services*, 550 NW2d 157 (Iowa 1996). Cited by the AAP Committee on Child Abuse & Neglect, 2002 (see above under “Bruises”) for the proposition that “State supreme courts in Iowa and New Hampshire have ruled that bruising is not necessarily considered to be an injury.”

Suspected child abuse: the potential for justice to miscarry. Hey E. BMJ 2003 Aug; 327(7410); 299-300.

Judges rule that children can sue for wrongful diagnosis of abuse. Dyer C. BMJ 2003 Aug; 327(7410): 305. Commenting on Hey, *supra*.

HIPAA exceptions providing law enforcement officials and social service providers access to protected health information. Alexandra Podrid. APRI Update 2003 Sep; 14(4). HIPAA enacted in 1996 and amended by the DHHS in 2002. HHS General Administrative Requirements, 45 C.F.R. Sec. 160.103 (2003).

A. Exceptions for law enforcement agencies

1. administrative subpoena: Sec. 164.512(f)(1)(ii)(c)

- 1 The information sought must be relevant and material to a legitimate investigation
- 2 The request must be specific and limited in scope to meet its intended purpose
- 3 Information that does not reveal the individual's identity could not reasonably be substituted for the information sought.

2. victims of crime Sec. 164.512(f)(3)

- 1 the individual agrees to the disclosure, OR
- 2 the covered entity cannot obtain the individual's agreement because of incapacity or emergency
In the case of incapacity or emergency, the law enforcement official represents that
 - a such information is needed to determine whether a crime was committed by someone other than the individual and will not be used against the victim
 - b law enforcement activity depends upon the disclosure and would be materially affected by waiting for the individual's consent
- 3 the covered entity, in the exercise of professional judgment, determines that disclosure is in the best interests of the individual

3. decedents Sec. 164.512(f)(4)

If a health care provider suspects that an individual has died as a result of criminal conduct, it may disclose protected health information about the decedent to a law enforcement official.

4. victims of abuse, neglect, or domestic vi Sec. 164.512(c)(2)

Covered entity may disclose to a government agency that is authorized by law to receive reports of abuse, neglect, or DV, but only if the disclosure is required by law.

5. averting a serious threat to health or safety Sec. 164.512(j)(2-4)

If the disclosure is needed to prevent a serious and imminent threat to the health or safety of a person, and the recipient is able to lessen the threat.

B. Exceptions for social service agencies

1. Child abuse, neglect, or domestic vi Sec. 164.512(c)(2)

if the social service agency is authorized by law to receive reports of abuse, neglect, DV.
three types of situations:

- the individual has consented to the disclosure, OR
- the disclosure is required by law, OR
- the disclosure is authorized by law AND
the disclosure is needed to prevent serious harm, OR

the individual is incapacitated AND
the information is needed for immediate enforcement activity
AND
will not be used against the individual

2. Mandatory State reporting laws Sec. 160.203(c)

HIPAA does not preempt state law provisions that provide for the reporting of disease, injury, child abuse, **death**, or for public health surveillance purposes.

When parental discipline is a crime: overcoming the defense of reasonable force in the investigative stage. Victor I. Vieth. APRI Update, vol. 16, no. 10, 2004. The defense of reasonable force has to do with the fact that physical punishment is accepted as a method of discipline for children. There is the majority-rule states hold that a parent is not criminally liable for an assault on a child if the blows to the child's body constitute reasonable force and are administered as a means of discipline. The minority-rule states hold that a parent is not criminally liable for an assault on a son or daughter even if the force is unreasonable, so long as the parent does not act with malice. Factors in the reasonableness and the malice are: (1) the degree of harm to the child, (2) the child's age, (3) the necessity for the punishment -- whether corporal punishment was "warranted by the circumstances." Even mild blows may be unreasonable if administered purely out of anger or if the child did not deserve any punishment, or was too young to understand. Even if warranted, the punishment may be unreasonable if it continues after its purpose of chastisement has been achieved.

CHILD DEATH REVIEW

<http://www.ican-ncfr.org>

Origins and clinical relevance of child death review teams. Durfee MJ, Gellert GA, Tilton-Durfee D. JAMA 1992; 267: 3172-3175. Cited by Broderick, below, for the proposition that child death review can result in significant numbers of changed manner-of-death diagnoses.

The Missouri Child Fatality Study: underreporting of maltreatment fatalities among children younger than five years of age, 1983-1986. Ewigman B, Kivlahan C, Land G. Journal of Pediatrics 1993 Feb; 91(2): 330-337. Cited by Broderick, below, for the proposition that there is a lot of underreporting of fatal child abuse.

AAP Policy Statement, Journal of Pediatrics 1999 Nov; 105(5): 1158-1160. Encouraging nationwide implementation.

Child Fatality Review: recommendations for state coordination and cooperation. Elster NR, Alcade MG. Journal of Law, Medicine, and Ethics, Summer 2003.

U.S. Department of Defense, Memorandum for Secretaries of the Military Departments. February 12th, 2004. Requiring review of all deaths of children of active-duty military personnel.

Reducing child fatalities through a team approach. Susan Broderick, JD. APRI Update, vol. 17, no. 8, 2004. 2,000 fatalities per year, 40% under one year old. These are conservative estimates, because "Many child fatalities have been systematically misidentified due to inadequate training, insufficient resources, poor inter-agency communication, and lack of cooperation among the parties involved in responding to these cases." (citing Durfee et al., JAMA 1992). Prevention requires a unified approach: hence the child death review teams. In 1978 Dr Durfee started one in Los Angeles. As a result, within the first five years of operation, seven deaths were changed from "natural" or "accidental" to "homicide," and one other one was changed from homicide to natural. In 1993 a landmark study from Missouri in the *Journal of*

Pediatrics highlighted the extent of underreporting and the success of review teams in reclassifying missed cases. There are two forms of child death review:

- (a) contemporaneous team investigation as soon as possible after the death
- (b) retrospective team review after the entire investigation has been completed

Current challenges:

(1) The biggest challenge is lack of nationwide uniformity in case inclusion. There are no standardized national criteria for child death review. This frustrates efforts to compare information across state lines and even local lines. There should be standardized data collection forms, integrated databases, and a national clearinghouse of effective practices. (citing Webster et al., 2003). This is the critical next step in bringing about prevention of fatalities. Progress has been made. Los Angeles ICAN is a resource for teams. Its National Center on Child Fatality Review (NCFR) in El Monte acts as a national clearinghouse. 626-455-4586, <http://www.ican-ncfr.org> Also the National MCH Resource Center for Child Death Review, based out of Michigan. 517-324-7330. <http://www.keepingkidsalive.org>

(2) The other challenge is “the ‘buck-passing’ phenomenon that can occur when agencies reviewing a child death are all hoping to avoid responsibility for failing to protect the child in some way.”

UNCLASSIFIABLE (see also under “Heart: Stress cardiomyopathy”)

Salt poisoning: presenting symptom of child abuse. Feldman K, Robertson WO. *Vet Hum Toxicol* 1979 Oct; 21(5): 341-343. Describes two young children with severe hypernatremia caused by salt administration combined with water deprivation.

Subtle fatal child abuse. Zumwalt RE, Hirsch CS. *HumP* 1980; 11: 167-174. Cited by Kirschner and Wilson in Ch. 19 of *Reece, 2d ed.* (2001) to the effect that soft tissue hemorrhage may cause enough anemia to “play a role in some deaths, but we did not consider it to be a factor in our cases.” Also noting stress cardiomyopathy (focal necrosis due to pain/ catecholamines), and fat embolism.

Diagnostic and surgical implications of child abuse. Ledbetter DJ, Hatch EI Jr, Feldman KW et al. *Arch Surg* 1988; 123: 1101-1105. Cited by ASCP Check Sample FP 03-9, *infra*, for the proposition that “A characteristic feature of child abuse is the marked delay that occurs from the time of injury to the time of presentation to medical care.”

Death due to superficial soft tissue injuries. Lee KA, Opeskin K. *Am J Forens Med Path* 1992; 13: 179-185. According to Alison D. Cluroe, 1992, 1995, this article described five alcoholic aboriginal women with extensive soft-tissue hemorrhages and delayed death due to hypovolemic shock attributed to blood loss into the tissues.

Superficial soft-tissue injury. Alison D. Cluroe. *Am J Forens Med Path* 1995 Mar; 16(2): 142-146. (have). Referring to Lee & Opeskin, above, and presenting the case of a chronically abused Polynesian woman beaten to death with a cricket bat, with multiple soft tissue hemorrhages and a history of increasing thirst over the previous 24 hours. In this case the soft tissue injuries were both recent and old, and included pockets, abscesses, myositis ossificans, and extremity fractures of different ages, 125 cutaneous scars, and intrafascial hematomas. Fat emboli were present but not considered significant. No myocardial lesion was found, although stress cardiomyopathy was considered. Brown-red granular casts were found in a few of the distal and collecting tubules, suggesting myoglobinuria. Death was attributed to hypovolemic shock due to blood loss into the tissues over a 24 hour period.

Ethical issues in professionals' response to child maltreatment. Howard Dubowitz. Child Maltreatment 1997 Nov; 2(4): 348-355. Dr Dubowitz discusses nine ethical issues, which I have heavily reworded here in order to translate them from academicese into plain english:

1. The high threshold for state intervention in family life. We wait until after harm has occurred before we allow the state to intervene in the home, because of our traditional respect for the sanctity of the family and the authority of parents. But in a world, and a nation, where many homes are poor and violent, this results in a large number of children going unprotected and getting injured.
2. What if the removing the child [and prosecuting the parent] will destroy the family? Is the cure worse than the disease?
3. What to disclose to the child and the parents?
4. Confidentiality? Whose privacy is at risk? The child's, the family's or the agency's? "...CPS withholding information from professionals perhaps stemming from a concern that the information may embarrass the agency."
5. Poor service by agencies? "The crisis of the child welfare system." "Even minimal standards for child welfare demand vastly improved services -- trained caseworkers, reduced caseloads, ..."
6. Drug-exposed newborns. Perhaps automatic removal is too drastic.
7. "When is it ethical to report a child to a child welfare system that one might not have confidence in?" This is partly a reprise of #5.
8. Religious refusal of care. And what about nonreligious refusal of care, as when the imposed care in fact will merely prolong dying?
9. Folkloric remedies that bruise or burn.

Guidelines for Ethical Behavior of Pathologists. ASCP, 1999.

Analysis of missed cases of abusive head trauma. Carole Jenny, Kent P. Hymel, Arlene Ritzen, Steven E. Reinert, Thomas C. Hay. JAMA 1999; 281: 621-626. Retrospective study of 173 head-injured children under 3 that were AHT. Found that 54 cases the abusive nature of the head trauma was missed (31%). Statistical analysis showed that four independent variables predicted successful dx of AHT: (1) abn resp status, (2) sz, (3) facial or scalp inj, (4) parents not living together. Cases were confused with gastroenteritis, sepsis, accidental head injury, otitis media, and idiopathic epilepsy.

Estimation of stress in child neglect from thymic involution. Tanegashima A, Yamamoto H, Yada I, Fukunaga T. [unknown journal] 1999; 101(1): 55-63.

Abused children have more severe injuries than those with unintentional injuries. Reece RM and Sege R. Arch Pediatr Adol Med 2000 Jan; 154: 9-22. Retrospective chart review of 287 trauma admissions to Tufts Univ Hosp aged 1 week to 6 ½ years. The abused group (19%) was much younger than the accidental group (81%). Abuse age 0.7 years, accidental 2.5 years.

<u>Type of injury</u>	<u>Abused</u>	<u>Acc</u>
SDH	46% had	10% had
SAH	31% "	8% "
RH	33% "	2% " *
Skin injuries	50% "	16% "

* No RH in accidental falls of under 4 feet. "RH are, if not diagnostic, compelling findings; most are seen in abusive head trauma."

MortalityAbused
13%Acc
2%

Accidents were 23% MVA, 58% falls, 2% playing.

Outcomes from television sets toppling onto toddlers. DiScala C, Bartel M, Sege R. Arch Ped Adol Med 2001; 155: 145-148. Newer TV sets are bigger and have more of the weight forward, making them less stable. 183 chil injured from National Pediatric Trauma Registry. 28% had moderate to severe injuries. 1/3 reqd adm to ICU. 5 died, all from intracranial hemorrhage. Marked increase in this injury modality since 1993. The CPSC has started working on this problem, at least with regard to tall TV racks used in schools. <http://www.cpsc.gov/>

Improving Joint Investigations of Child Abuse: A summary Report. Washington, DC: Police Foundation

Understanding Child abuse and Neglect, 5th edition. Cynthia Crosson-Tower. Boston: Allyn and Bacon, 2002. ISBN 0-205-33795-3. A current college textbook giving comprehensive coverage from a social-sciences perspective. My thanks to Dr. Susan M. Larsen of Cal State Fullerton for this excellent text.

Pediatric "body packing." Traub SJ, Kohn GL, Hoffman RS, Nelson LS. Arch Ped Adol Med 2003 Feb; 157(2): 174-177. Now they're using children. Case report of a 16 year old and a 12 year old.

Plagiocephaly and brachycephaly in the first two years of life: a prospective cohort study. Hutchison B L, Hutchison LAD, Thompson JMD, Mitchell EA. Pediatrics 2004 Oct; 114(4): 970-980. Nonsynostotic plagiocephaly (NSP). Significant univariate risk factors at age 4 months were male sex, firstborn, limited passive neck rotation at birth, limited active head rotation at 4 months, supine sleeping at birth and at 6 weeks, lower activity level. See above under SIDS biology.

Avoidable pitfalls when writing medical reports for court proceedings in cases of suspected child abuse. David TJ. Arch Dis Chil (GB) 2004; 89: 799-804.

1. *The overriding duty of the doctor is to the court*, not to the prosecution, the defense, or the child. This is different from the clinical care setting, where the primary duty is to the child. Do not exaggerate items such as the likelihood of abuse, the amount of force, the degree of pain suffered.
2. Make a *chronology*.
3. Do not *infer intent* from injuries.
4. *Take your own history* in person from the child and the caregivers.
5. Get the *full medical records*.
6. *Get all clinical photographs*.
7. *Don't try to age bruises based on color.* But histology has a role.
 - a. Clinically, color schemes are unreliable. "Attempts to age bruises based on their colour is fraught with difficulties." (citing Langlois & Gresham, 1991; Stephenson & Bialas, 1996; Munang et al., 2002; Bariciak et al., 2003). "The time course of the appearances of bruises may vary with the location, depth, extent, and nature of a bruise. The only established fact is that the presence of a yellow colour within a bruise indicates that it is at least 18 hours old." (citing Langlois, Stephenson.)
 - b. "Histopathology of bruises is worth performing in some fatal cases. Histology can rule out skin lesions such as blue naevi and can confirm bruising in those with dark skin. Histology of a bruise may enable the pathologist to say it is very fresh (no cellular reaction), recent (infiltration with neutrophils), or more than approximately two days old (presence of haemosiderin)."
8. Don't assume that normal coags rule out a significant *clotting disorder*. Routine coags only exclude the commonest conditions. If there is an individual or family history of easy bruisability or bleeding tendency, referral to a haematologist and further clinical investigation will be required. An example is the Hermansky-Pudlak syndrome, which can cause subdural and retinal haemorrhage. (citing Russell-Eggitt et al., 2001).
9. X-rays should be interpreted only by experienced *paediatric radiologists*.
10. *Force needed to cause an injury.* "One is well advised to exercise caution and avoid dogmatic statements about ... how enormous the forces must have been. The truth is that the requisite scientific

studies, in which human infants are deliberately injured in different ways ... have not been performed and never will be. The only exception is [Weber's skull fractures studies].

11. *Pain resulting from fractures.* Fractures cause two pains: acute pain and ongoing pain. "The immense variability means that overconfident assertions are worth avoiding. While the occurrence of the fracture itself is certain to cause significant immediate pain, the way that this pain is communicated to carers or parents can vary between different children and at different ages. In some cases the ongoing pain is obvious for all to see, and the limb of a child is manifestly not being used ... However, ongoing pain after a fracture is highly variable, and, for example, in infancy, rib fractures and metaphyseal limb fractures often produce no detectable ongoing pain at all, presumably because the bone is stable and the periosteum often little disturbed. These injuries commonly pass undetected by clinicians examining the child... The point is that caution is required before concluding that a reasonable carer should have known that something was seriously amiss in a child with rib or metaphyseal limb fractures."

12. *Bias.* "It requires constant vigilance to avoid bias creeping into a report." For example, mentioning that the parents missed three appointments without mentioning that they kept 15 other appointments.

13. Don't read too much into negative information of a general nature. In other words, don't build up scattered evidence of neglect (dirty child, missed appointments) into a conclusion that the parent is unfit. Such a conclusion is not for the physician, particularly if he has never met the family. It is for social workers and the courts.

14. Don't rely on the *personality profile of the parent* to make a diagnosis. Yes there are risk factors present for abuse. But diagnose abuse only from the clinical findings. The fact that an adult has risk factors for abuse does not mean that he did abuse.

15. Be prepared to *consider each injury separately*, as to its nature and possible causes. The court will require you to do this. "The evidence for abuse may be weak or nonexistent for individual lesions, but the overall pattern may nevertheless point to non-accidental injury, maybe because of the extent or distribution of the injuries."

16. Don't comment *outside your area of expertise*. "This means, for example, that a histopathologist whose expertise is in conducting postmortem examinations and dealing with the examination of tissue samples is not in a position to comment on the symptoms, diagnosis, treatment, or prognosis of living children."

17. *Selective reference to the medical literature.*

a. "What is not acceptable is to selectively provide a few references that bolster the point of view that one wishes to advance, while ignoring all material that points in other directions."

b. "It is important to bear in mind that a high proportion of the medical literature on child abuse is deeply flawed..."

18. Stay away from *new medical theories*. These belong in the journals, not in court.

19. *Admit uncertainty.* "The reality is that there are many cases in which doctors can do little more than voice suspicion or anxiety... Never be afraid to say that one is simply not sure."

20. *Admit errors.* "If, on reflection, one realises that one has arrived at an incorrect conclusion, it is a strength and not a weakness to readily acknowledge this. It is a common experience at experts' meetings...that opinions change when one has a chance to better understand the reasoning of a colleague, or when one learns of new facts of which one was unaware. Experts who change their opinions for good reason on receipt of fresh information are respected by the court rather than criticised. However, if one changes one's opinion, one should always explain the reasons for the change." "All doctors make mistakes. The most serious error is to refuse to admit one has made a mistake, even when it is pointed out."

Chapter 22, "Fatal child abuse" in: Saukko P and Knight B, *Knight's Forensic Pathology, Third Edition*. New York: Oxford University Press, 2004. Gives a definition as above under "Definitions." Cautions against applying the pejorative of abuse in cases that have lesser degrees of trauma, because these could be due to "exasperation, panic, or even attempts at resuscitation." Surface bruising: certain sites of predilection: wrists, forearms, upper arms, thighs, ankles. Buttocks. Face, especially the cheeks and mouth area. Bruises on the chest, abdomen and neck are usually from finger pressure ("sixpenny bruises"), not blows, but abdomen and lower chest may be associated with deep visceral injury. Occasionally a whole handprint may be discernible. As to aging of bruises, see under "Bruises;" but "any bruise with yellow coloration must be more than 18 hours since infliction." (p. 463). Bruises of markedly different colors cannot have been inflicted during the same episode. Skeletal damage: Referring to a study by Worlock et

al., 1986, he mentions rib fractures and spiral fractures of the humerus. "A spiral fracture of the diaphysis of a long bone must be considered a suspicious injury in infants, as such a lesion is likely to be the result of a twisting strain, unlikely to occur in accidental circumstances." (p. 467) (As to spiral fractures, see Pierce, Bertocci et al., 2004, below). Skull fractures: Cites Harwood-Nash et al., 1971, to the effect that although in fatal child abuse skull fractures are often associated with intracranial hemorrhage, less than half of infants with SDH have skull fractures and SDH is twice as frequent in the non-fracture cases. An infant's skull is flexible and may dimple in without fracturing. The most common abusive fracture is occipitoparietal, but "differentiation from accidental falls is impossible on anatomical or radiological grounds alone, in spite of the dogmatic claims of some radiologists." Goes into the reasons why one cannot be dogmatic about this. (p. 464) There is controversy over the height of an infant fall needed to cause (a) skull fracture or (b) brain injury. "It is impossible to forecast what will happen following a fall of even minor magnitude. Though many paediatricians will deny that a passive fall from adult waist level -- or even higher -- can cause either skull fracture or intracranial damage, there is experimental proof and witnessed cases on record where this has occurred." (p. 465) Reviews the literature on falls and skull fractures, finding, with Reiber, that the literature breaks out into two competing camps -- the *major injury from major fall* set and the *major injury from minor fall* set. Weber's experiments on 34-inch drops, getting a few fractures even on thick foam or folded blankets. Reichelderfer et al., 1979, concerning serious head injuries from playground falls over 50G, with a 3-inch fall onto concrete generating 100-200 G and one foot generating 475-500G. Helfer et al., 1977, finding no serious injuries in 246 low-height falls, although two skull fractures. Nimityongskul & Anderson, 1987, finding that 75 in-hospital falls caused only one skull fracture. Williams, 1991 studying 398 falls including 106 independently witnessed, finding that below 10 feet there were three skull fractures and no life-threatening injuries. Reiber, 1993 finding three **fatal** skull fractures in infants after witnessed falls of under 3 feet. Hall, 1989 confirms that severe or fatal damage can occasionally arise from low falls; 18 deaths from falls less than 3 feet, two of which were witnessed by medical personnel. Chadwick, 1991, 317 childhood falls, as we know. Hobbs, 1984, 89 skull fractures of which 29 were abused; 19/20 fatalities were abuse, Hobbs arguing that abusive fractures were complex, multiple, depressed, or growing fractures, while the accidentals were linear and single, usually parietal. Leventhal et al., 1993, 104 infant skull fractures, concluded that 34% were definitely abusive, 62% definitely accidental, and 4% undetermined. Billmire & Myers, 1985, 95% of serious intracranial damage was abusive and 64% of all infant head injuries excluding simple fractures. "These varying figures emphasize the lack of consensus in the literature." (p. 466) Discusses bilateral fractures from being dropped on the vertex. Limb fractures: As to metaphyseal fractures, says that "Swinging the child by the wrists or ankles, dragging it by an arm or shin and violent shaking using the limbs as 'handles' are the usual mechanisms." (p. 466) Also wrenching and twisting. Cites the atlas of the battered child syndrome by Cameron and Rae, 1975 as the source of the doctrine that metaphyseal fragments are virtually pathognomonic of child abuse. Mentions tibial SPNBF as being due to handling of the legs during breech delivery. Rib fractures: Says that the incidence of accidental rib fractures is "a matter of considerable controversy." Says that the radiological dating of fractures is "notoriously variable." Says that callus forms in about 10 days. Sometimes the radiological and dissection diagnosis does not concur. As to lateral fractures, "With fresh fractures found at autopsy, the possibility of chest compression during CPR must always be considered, even though some paediatricians and radiologists will strenuously deny the possibility of this happening. ...It is admittedly very uncommon." Dating of fractures: The absolute dating of healing fractures by the state of callus formation is far from accurate. ...Histological appearances are also very unreliable... Bone diseases: OI types II and III have obvious dysmorphisms and will not be confused with child abuse. Type I is the classical variety (70% of cases), with blue sclera, Wormian bones, dentinogenesis imperfecta, and a family history, and again will not be confused with child abuse. That leaves Type IV (about 5%). Type IV-A is the one likely to be confused with child abuse. In fact, some of the reported cases of IV-A may actually have been child abuse. Type IV-A has no family history, no blue sclera, and no osteopenia. It has Wormian bones. It has frequent skull fractures and metaphyseal fractures. Type IV-B has dentinogenesis imperfecta, Wormian bones. Type IV is very rare; if there is no family history, no Wormian bones, and no dentinogenesis imperfecta, but there are fractures, the incidence of such a picture from OI would be one in 1 to 3 million. Caffey's disease is extensive periosteal new bone around diaphyses, mostly in the upper body -- ribs, clavicles, ulnae, and especially the mandible. Congenital syph gives periosteal thickening which tends to be symmetrical and metaphyseal separations with porotic bone adjacent. Copper deficiency gives periosteal thickening, spurring of the metaphyses, osteoporosis, and cupping of the metaphyses, plus hair abnormalities. These babies are

mentally retarded, floppy and pale with sideroblastic anemia and neutropenia. Menke's syndrome also associated with copper deficiency affects males only and gives kinky hair, Wormian bones, abnormal metaphyses, and mental retardation. Shaking controversy: The most common cause of death in child abuse. The classical lesion is SDH. "In infants, it most often occurs from direct impact upon the skull -- as in a blow or fall." (p. 469) Goes on to say, "For some years, it has been held that vigorous shaking, without an impact, is also a common cause of subdural bleeding. This is a view strongly held by many paediatricians, who may prefer this mode as a first choice over a blow or fall, but recent research has thrown doubt on the common acceptance of this mechanism." Goes on to quote DiMaio's textbook on SBS. Points out the experimental data of Duhaime -- 50X more acceleration from impact. Says, "It is the rate of change and the duration of deceleration -- the 'strain rate' -- that is most damaging, rather than a steady deceleration." Shaking is low strain-rate. Cites Howard, Bell & Utley, 1993, to the effect that low strain-rate events will cause cerebral contusion, and cause SDH or vascular injury only if high-energy. High strain-rate injury causes SDH even with low-energy events. [But that argues that SDH should frequently result from low-height falls onto hard surfaces, which it doesn't. -JKR] Goes on to discuss Geddes, to the effect that DAI does not happen from trauma in SBS cases, that AI in such cases is global-ischemic, that there is craniocervical junction trauma which would explain the global ischemia, and that hypoxia causes dural hemorrhage and maybe even subdural blood ("hypoxia is sufficient to cause extravasation of significant amounts of venous blood both inside and under the dura.") (p. 470) Concludes that "Although shaking presumably may cause subdural haemorrhage (SDH), it is likely that it is a relatively uncommon cause, compared with impact. This situation may well have arisen because a blunt impact upon the head of infant, if spread over a wide area following contact with a flat surface, can leave no external scalp mark, no subscalp bleeding and no fracture of the skull -- yet transmitted forces can still be sufficient to cause high-strain shearing stresses within the cranium leading to subdural bleeding." (p. 470) Says that a number of factors combine to make the pathologic demonstration of impact injury impossible in many cases, so that the shaking explanation becomes attractive. These factors include rapid death, which obviates diagnostic signs of DAI, the fact that axonal injury tends to be less distinct in the infant brain, and the above-described scalp difficulty. Says, "Presumably, as vigorous shaking can sometimes rupture vessels in the subdural space, this must still be accepted as an alternative to impact in the causation of subdural haemorrhage.." (p. 471) Fall versus inflicted: Skull fractures are easy to cause from low-height falls, as shown by Weber. But skull fractures correlate poorly with the presence or absence of brain injury. "The evidence that low falls may cause brain or meningeal lesions is much less convincing than that proving skull fractures -- but the possibility exists and cannot be dismissed by inflexible, dogmatic opinion." (p. 471) Experimentation is impossible. Animal models are useless. Statistical evidence is weak in a given case; if something has happened even once, it can happen again. Describes the study of Howard, Bell & Utley (1993): 28 SDH in children under 18 mos. 14 of them were low-height falls by history; the authors concluding that falls were a fairly frequent cause of SDH. [Compare the recent work of Kirsten Bechtel et al. (*Pediatrics* 2004, below under "Head Injury -- Fall vs. Inflicted") finding that 18 out of 67 accidental head injuries in children under two had SDH's; these included numerous low-height falls. -JKR] Visceral injury: ok, ok. Eyes, ears and mouth: Whether CPR can cause RH is controversial. Injury to the external ear is frequent. Sometimes deafness has resulted. Lips and mouth is very common, due to slapping. Teeth breakage. Burns: Both dipping scalds and pouring scalds occur. The cigarette burn is seen relatively often in abused children, usually on areas not covered by clothing. Bite marks: More often inflicted by the mother. Self-biting does occur and needs to be excluded. Other injuries: Hair pulling finger breaking. Straps & ropes. Pinch marks are two small opposing triangles with a white space between them. The autopsy in child abuse: He refers us to his other textbook, *Paediatric Forensic Pathology* (1989) for a full description of the autopsy and report. Makes the following points here: (1) The scene should be visited whenever possible, re floor coverings, heights. (2) Examine the child with the clothing on if possible. (3) Don't wash body. (4) Learn about the nature of resuscitation attempts. (5) Do x-rays. (6) Take measurements. (7) External exam must cover every square centimeter of the body, including orifices. (8) Full photography. (9) Extensive dissection of the subcutaneous areas if bruising is present or suspected. (10) Full tox. (11) Skin samples for fibroblast culture if indicated; blood for DNA. (12) Swab bite marks. (13) Specimen radiographs. (14) Fix the brain. (15) Eyes.

Meadow faces GMC over evidence given in child death cases. Dyer O. BMJ 2004 Jan 3; 328(7430): 9. This is a news item. It relates that Sir Roy will face charges of serious professional misconduct from the General Medical Council, arising out of his testimony in the trials of Trupti Patel, Sally Clark, and Angela

Cannings for suffocating their babies. They are also charging Dr. Alan Williams, a Home Office forensic pathologist. Trupti was acquitted. Sally and Angela had their convictions overturned on appeal. Trupti lost three babies, Sally lost two, and Angela lost three. Professor Meadow told the jury that the chance of two cot deaths occurring in an affluent family was one in 73 million. The Crown Prosecution Service will review 50 convictions in which Dr Williams gave evidence going back seven years. It is alleged that in the Sally Clark case, Dr Williams withheld evidence that in one of her babies there was *Staph aureus* in the CSF. The editors of the Lancet sprang to Dr Meadow's defence; *Lancet* 2005; 366: 3-5.

Guilty innocents and the road to false confessions. *Lancet* 1994 Nov 26; 344(8935): 1447-1450.

Viewpoint. Roger W. Byard. *Med J Aust* 2004; 181: 52-54. The Sally Clark case.

America's gulag archipelago. Fost N. *NE J Med* 2004 Dec 2; 351(23): 2369-2370.

Physical abuse of children. Sirotnak AP, Grigsby T, Krugman RD. (University of Colorado at Denver). *Pediatric Reviews* 2004 Aug; 25(8): 264-277. A review article.

U.N. Reports say poverty causes deeper worry south of Sahara. *The New York Times*, June 10, 2005. By Celia W. Dugger. Two reports: the Millennium Development Goals Project and the United Nations Development Program. 28 million African children will die needlessly over the coming decade... Almost half the world's deaths of children under 5 occur in sub-Saharan Africa. Globally half the 11 million annual child deaths are the result of five diseases: **pneumonia, diarrhea, malaria, measles, and AIDS.**

Women who kill their children. Rouge-Maillart C et al. *Am J Forens Med P* 2005 Dec; 26(4): 320-326. See above under "Epidemiology and risk factors."

Fatal dog maulings associated with infant swings. Chu AY, Ripple MG, Allan CH, Thogmartin JR, Fowler DR. *JFS* 2006 Mar; 51(2): 403-406. Presents two cases with photos. One pit bull mix. The swinging motion causes them to pounce. I heard this presented at the AAFS Annual Convention in 2005.

Sudden unexpected death in infancy and childhood due to undiagnosed neoplasia: an autopsy study. Somers GR, Smith CR, Perrin DG, Wilson GJ, Taylor GP. *Am J Forens Med P* 1006 Mar; 27(1): 64-69. Found eight cases over twenty years in the autopsy files of the Hospital for Sick Children:

<u>Tumor</u>	<u>Age</u>
ALL	1 mo
AML infiltrating myocardium	2 mo
Papillary fibroelastoma of the mitral valve occl coronary ostium	2 y 9 mo
Wilm's tumor huge bilateral	2 y 11 mo
T-call lymphoma	4 y 11 mo
T-cell lymphoma	6 y 7 mo
Medulloblastoma	13 y
Adenocarcinoma	11 y

How good is the evidence available in child protection? Sibert JR, Maguire SA, Kemp AM. *BMJ* Jan 30 2007. *Arch Dis Child* 2007; 92: 107-108. Refuting a lead article by David Chadwick on 22 July 2006 stating that the published evidence base for child protection is robust. On the contrary, say these authors, who are part of an evidence-based medicine review team. They have reviewed the literature on an evidence-based basis and find it severely wanting due to small numbers, the unavoidable lack of controls or a gold standard for diagnosing abuse, the fact that most articles are of the case-series design, which inevitably suffers from selection bias and lack of any comparative data. They single out a few specific areas where they find the evidence to be robust. These are the fact that rib fractures are highly specific for abuse, the geometry of inflicted scalds, the fact that you can't age bruises, the fact that you can't age fractures more than broadly. They mention the lack of literature defining the recognition of human bite marks, the questionable specificity of torn frenulum for abuse, and the recognition of cigarette burns. They

discuss the legal difficulties that court testimony of child protection experts has run into in recent years, and the broadscale lack of credibility of the specialty, and the reasons therefor, of which the main one is the above-mentioned thinness of the medical literature. They say that a new, evidence-based research effort is needed to repair the thinness of the literature. They also say that many practitioners in the field possess a wealth of private experience which if it were only published would be valuable in adding to the evidence base. But it isn't being published.

This is part of the ongoing train wreck in the UK regarding the forensic/ judicial aspects of child abuse diagnosis. Numerous careers have been ruined, doctors have come down with mental illness as a result, and there has been wholesale flight from the specialty of child protection. How long before that syndrome comes here? Dr Sibert and colleagues warn against overcertainty in diagnosis, lest we suffer the same fate that befell poor Drs Meadow and Southall and many of their colleagues.

Spring 2006 Children and the law symposium, 28 Hamline J. Pub. L. & Pol'y (Fall 2006). This volume includes a monograph by Victor Vieth entitled "Unto the third generation: a call to end child abuse in the United States within 120 years (revised and expanded)." Vieth argues that child abuse can be ended, and quotes Dr Chadwick to that effect, but also to the effect that it will take over 100 years. He states five obstacles to ending child abuse:

- A. Many children suspected of being abused are not reported into the system.
- B. Even when reports come into the system, most children will never have their cases investigated
- C. Even when cases are investigated, the investigators and other front line responders are often inadequately trained and inexperienced.
- D. Even when an investigation successfully substantiates abuse and gets a victim into the system, the child is typically older and it is more difficult to address the physical, emotional, and other hardships caused by the abuse.
- E. Because the child protection community lacks a unified voice in communicating the needs of maltreated children, these victims receive an inadequate share of our country's financial resources.

Then he gives a "battle plan" for ending abuse.

- A. Abused children must be reported into the system and those reports must be of high quality.
 - 1. Every university must teach students entering mandated reporting professions the necessary skills to competently perform this task.
 - 2. Mandated reporters in the field must receive annual training on the detection of abuse and their obligations to report.
- B. Child protection workers and law enforcement officers must conduct a competent inv of every child abuse case that comes to their attention and, when abuse is substantiated, pursue appropriate civil and criminal actions.
 - 1. Children reported as victims or witnesses to an act of child abuse must be interviewed by a social worker, police officer, or other professional trained in the science and art of speaking to children. (Refers to the APRI's "finding words" model.)
 - 2. child protection workers called on to investigate and repair families damaged by abuse must be competent to perform these tasks.
 - a. Every university must teach child protection professionals necessary investigative skills.
 - b. Every university must teach child protection professionals to work meaningfully with families impacted by abuse.
 - c. Graduate schools must adequately prepare professionals to work with child victims. Law schools, medical schools, other graduate schools.
 - d. Once in the field, civil child protection professionals must have access to ongoing training and technical assistance.
 - 3. Prosecutors must be adequately trained to prosecute egregious child abusers.
- C. We must teach police officers, social workers, prosecutors and other child protection professionals to be community leaders in the prevention of child abuse.

1. This training must begin in college and continue once these professionals are in the field.
2. Child protection professionals must enlist the support of the faith-based community.

...

My criticism of all this would be that it imprisons itself in the current criminal-justice model. Indeed, it reifies that model. But that model is a demonstrated failure. We need a different model that addresses the values of society, so that harming children becomes as morally unacceptable as , for example, going naked in public. –JKR.

WEB SITES

National Clearinghouse for Child Abuse & Neglect <http://www.calib.com/nccanch/>

Web site of the Uniformed Services University of the Health Sciences: child abuse radiology images and prevention materials. <http://rad.usuhs.mil>

Child welfare database, maintained by the Child Welfare League of America, in cooperation with the state child welfare agencies: the National Data Analysis System (NDAS). Intended to promote integration of research, policy, and practice. Includes child welfare data from many sources in tabular and graphic form. Links to child welfare agency web-sites, state statutes. <http://ndas.cwla.org>

National Clearinghouse on Child Abuse & Neglect Information. Sandi McLeod, Senior Information Specialist. 800-394-3366 x 352

Childhood, infant, and perinatal mortality, 1996: social and biological factors in the deaths of children under 3. Schuman J. Population Trends 1998 Summer; 92: 5-14. Birth weight, mother's youth, marital status, father's social class, multiple birth, sex.

Evaluation for possible physical or sexual abuse. Kini N and Lazoritz S. Peds Clin N Amer 1998 Feb; 45(1): 205-219. Child abuse requires a *multidisciplinary approach*. The clinician documents suspicion of abuse. The final determination of abuse is under the purview of the legal system.

National Clearinghouse on Child Abuse and Neglect, (NCCAND) 330 C Street SW, 20447. 800-FYI-3366. <http://www.calib.com/nccanch>. Makes various reports including statistical reports.

Bureau of Criminal Justice Statistics <http://ojjdp.ncjrs.org/facts/ezaccess.html>
Also <http://www.ojp.usdoj.gov/bjs>

ICAN National Center on Child Fatality Review,
Funded by a grant from the U.S. Department of Justice, Missing and Exploited Children's Program, Office of Juvenile Justice and Delinquency Prevention. Has many links to other sites.
<http://www.ICAN-NCFR.org>

Los Angeles County Inter-Agency Council on Child Abuse and Neglect
4024 North Durfee Avenue
El Monte, CA 91732
626-455-4585
ajlangsta@co.la.ca.us

CDC guidelines for investigation of child abuse
MMWR 45 (RR-10: 1-22) June 21, 1996

US DOJ guidebooks on investigation
Office of Justice Programs
800-638-8733
puborder@ncjrs.org

National Guidelines for Death Investigation. US Department of Justice. Available on the web at <http://www.ojp.usdoj.gov/nij>. These are very basic general gdl's for any death or disaster scene.
<http://www.ncjrs.org/pdffiles/167568.pdf>

The Harry Kempe Center in Denver runs training courses. <http://www.kempe.org/>

The Mayerson Center for Safe and Healthy Children at the University of Cincinnati puts out a healthcare professionals' toolkit. http://www.chmcc.org/programs_services/152/tools/update.asp

The Consumer Product Safety Commission has evaluated cribs, toys, bunk beds, walkers etc. See CPSC web site at <http://www.cpsc.gov>

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

The National Center on Shaken Baby Syndrome. <http://www.dontshake.com>

A child abuse site on ivillage.com at <http://pages.ivillage.com/sacboard/>

The National Center for Prosecution of Child Abuse (NCPA) of the National District Attorneys Association. <http://www.ndaa-apri.org> and its monthly update newsletter <http://www.ndaa-apri.org/apri/programs/ncpa>

The National Exchange Club Foundation. Basically a fundraising site, but has statistics.
<http://www.preventchildabuse.com/>

Back to sleep home page <http://www.nichd.nih.gov/sids/>

Helping those falsely accused of child abuse. <http://www.accused.com/>

The National Coalition for Child Protection Reform. <http://www.nccpr.org/>

Cornell Family Life Development Center. <http://www.human.cornell.edu/centers/fldc/>

The AMA Family Violence Prevention Program. <http://www.ama-assn.org/ama/pub/category/>

The ABA Center on Children and the Law. <http://www.abanet.org/child/home.html>

ABA Commission on Domestic Violence <http://www.abanet.org/domviol/>

APSAC. <http://www.apsac.org/>

The American Psychiatric Association. <http://www.apa.org/books/>

Minnesota Center Against Violence and Abuse <http://www.mincava.umn.edu> has both child abuse and domestic abuse areas.

Volcano Press. <http://www.volcanopress.com/> publisher of Michael Durfee's "Guide to the Eval of Child Abuse (2003), with periodic updates.

Childabuse.org <http://www.childabuse.org/>

California Office of Criminal Justice Planning (protocols for medical examination of abuse victims).
<http://www.ocjp.ca.gov/>

California Medical Training Center at UC Davis Medical Center. <http://www.ucdmc.ucdavis.edu/medtrng>
 Wrote the California OCJP medical protocols of 1987 and 2003.

California Attorney General
<http://www.safestate.org>
 Contains extensive material on child abuse, DV, elder abuse, etc.

ICAN-National Center for Child Death Review. <http://www.ican-ncfr.org>

National MCH Resource Center for Child Death Review. <http://www.keepingkidsalive.org>

GW Medical Publishing, Inc. <http://www.gwmedical.com> Series of top-level textbooks and educational materials

APSAC <http://www.child-abuse.com/> Mostly prevention

The Child Abuse Team/ Mayerson Center at Cincinnati Children's Hospital maintains a useful web site with textual reference material for professionals at <http://www.cincinnatichildrens.org/svc/prog/child-abuse>

The American Academy of Pediatrics has professional resources and educational material.
<http://www.aap.org>

Testing frozen tissue for long QT syndrome. <http://www.familion.com>

Emergency medicine textbook chapter quite detailed on child abuse all forms workup.
<http://www.thomsonhc.com/>

PANDA Prevent Abuse and Neglect through Dental Awareness Hotline 501-661-2595, an important resource on dental abuse & neglect

Expert witnesses <http://www.seak.com>

And let's not forget <http://www.dontshake.org/>

Los Angeles Community Child Abuse Councils. <http://www.lachildabusecouncils.org> Includes suicide prevention cards.

The Chadwick Center at Children's Hospital of San Diego. <http://www.chadwickcenter.org>