

***Shaken Baby Syndrome and Non-Accidental Injury:  
Are Parents and Caretakers Being Falsely Accused?***

(This Material Has Been Prepared As a Medical-Legal Defense Brief for These Cases.)

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Introduction

These cases fall in three general categories: (1) Shaken Baby Syndrome (SBS), in which it is assumed that a parent or caretaker, exhausted and irritated by a fussy baby, picks up the infant by its chest or heels and shakes the baby with such violence that any onlooker would recognize it as excessive and dangerous. (2) Non-accidental trauma in which there may be incidental head or body impact against a solid object during the shaking, and (3) infants with multiple fractures. The latter is addressed in a separate monograph. Based on personal observations of more than 10 years, in each of these categories there have been patterns of precipitous diagnoses of inflicted child abuse without first establishing a differential diagnosis and ruling out other possible causes of the findings. Due to these deficiencies, many parents and caretakers are being falsely accused and criminally convicted. In any other medical specialty, these failures to establish a differential diagnosis of the findings would be considered substandard and unacceptable medical practice.

**Part I: Shaken Baby Syndrome/Non-Accidental Injury**

(A) Origin of the Shaken Baby Syndrome (SBS)

Working with the U.S. Department of Transportation, an Oxford-trained neurosurgeon, AK Ommaya devised an experiment to measure the amount of rotational acceleration required to reach the threshold of brain injury with adult Rhesus monkeys as subjects. As reviewed by R Uscinski:

“A contoured fiberglass chair was built, mounted on wheels, and placed on tracks with a piston behind it. The monkeys were strapped into the chair with their heads free to rotate in such a way that there would be no impact. The piston then impacted the chair, simulating a rear-end motor vehicle collision. The experiment was photographed with a high-speed camera, allowing calculations of generated rotational accelerations. Ommaya was able to demonstrate that a rotational acceleration of 40,000 radians/second (squared) was sufficient to produce intracranial injury in 19 of the animals, with 11 of them also demonstrating neck injuries. Then, using the scaling parameters, he estimated that less rotational acceleration would be required to produce concussion in the larger human brain, perhaps on the order of 6,000 to 7,000 radians/second (squared),”(1)

Calculations were based on the same laws as described in classical Newtonian physics, as applied to movements of planetary bodies, that force is the product of mass and acceleration.

Ommaya's experiments were published in the *Journal of the American Medical Association* in 1968.(2) In 1971 Guthkelch reported on the first diagnosed case of SBS in which he hypothesized that subdural hematomas could be caused by manually shaking an infant without the head impacting on any surface.(3) One year later Caffey alluded to the parent-infant stress syndrome, with manual shaking causing intracranial injury in the form of subdural hematoma and cerebral contusion of infants.(4) Two additional papers published by Caffey over the next two years emphasized shaking as a means of inflicting intracranial bleeding in children.(5,6) It is important to note that each of these four papers referred to Ommaya's publication of 1968 as justification for this concept.

After publication of these four papers, the term Shaken Baby Syndrome became widely accepted as a clinical diagnosis for inflicted (nonaccidental) head injury in infants in which findings of subdural (brain) hemorrhages and/or retinal hemorrhages became accepted as exclusively diagnostic of SBS in the absence of known major accidental injury and remains so today in hospital emergency rooms..

### (B) SBS Theory - Irreconcilable with the Weakness of the Human Infant's Neck

From its origins, the SBS has been based on the assumption that a parent or caretaker, becoming irritated over a baby's prolonged fussiness and crying, loses self-control and, grasping the infant by the chest or heels, shakes the infant with such violence that any onlooker would recognize it as excessive and dangerous. It is true that shaking does sometimes take place when an infant collapses and stops breathing, which is a common presentation in these cases. In such situations a panicky parent, usually untrained in resuscitation, picks up an infant and (not knowing what else to do) *mildly* shakes an infant that has just gone into respiratory arrest. However, these instances do not in any sense constitute SBS.

As pointed out in the Uscinski report,(1) the brain and head of an infant is nearly seven times larger and heavier than that of a monkey. In addition, adult monkeys are known to be incredibly strong, approximately four times stronger than humans. There would be no comparison, therefore, between the neck muscle strength of an adult monkey and that of an infant, barely able to hold up his or her head by age six months, so that if violent shaking were actually taking place, the incidence of neck injuries in the SBS should be exponentially greater than in monkeys. With these facts in mind, consider the following:

‘ Most SBS cases in the USA take place during the first six months of life, when there are negligible infant neck muscles. As will be shown further on, the major impact of violent shaking would fall at the junction between the base of the skull and brainstem area and upper cervical spinal cord. In most if not all instances this would result in instant death or paraplegia from brain stem or cervical spinal cord injuries.

‘ 11 of the 19 monkeys (over 50 percent) in the Ommaya experiments had significant neck as well as brain injuries.

‘ Although human brain injuries may occur in rear-end vehicle collisions, whiplash injuries to the neck comprise an overwhelming majority of adult injuries, in which ligaments and muscles are torn, resulting in destabilization of the cervical (neck) vertebral column. Since infants have only rudimentary neck muscles and connective

tissues as cushions, the full impact of shaking would fall on the highly vulnerable cervical spinal cord at the base of the brain, which almost certainly would cause death or paraplegia.

‘ In view of these considerations, one would expect a far greater incidence of severe cervical skeletal and spinal cord injuries in infants than took place in the monkeys, and yet this type of injury has not been documented in any SBS case to date. In view of these facts, the Shaken Baby Syndrome theory defies both reason and common observation. As a simple statement, **it is physiologically impossible.**

‘ As a final observation concerning “**Nonaccidental Injury,**” in which it is assumed that there is a head impact in the process of shaking, there must be substantiating evidence in the forms of head/scalp bruising and/or subgaleal (scalp) hematoma in order to justify such an assumption.

Reflecting these considerations, F.A. Bandak, Ph.D., a biomechanical research scientist and research professor in the Department of Neurology with the Uniformed Services University of the Health Sciences, U.S.A., and a former director of head injury research at the National Highway Traffic Safety Administration, U.S.A., wrote the following in a paper published in 2005:

“Forceful shaking can severely injure or kill an infant. This is because the cervical spine would be severely injured and not because subdural hematomas would be caused by high head rotational accelerations. We have determined that an infant head subjected to the rotational velocity and acceleration called for in the SBS literature, **would experience forces on the infant neck far exceeding the limits for structural failure of the cervical spine.** (emphasis mine) Furthermore, cervical spine injury from shaking can occur at much lower levels of head velocity and acceleration than those reported for the SBS.(8)

(C) Chris Van Ee, PhD, Subject: Dynamic Biomechanical Findings on SBS-LMF

“Scientific testing has shown that head acceleration levels from anterior/posterior human shaking of a normal 0- to 2-year-old child in the sagittal plane results in head acceleration and force levels that are much lower than those which are associated with traumatic head injury. Repeated testing of this hypothetical has shown that the head accelerations associated with shaking are far below the level associated with injury, and there is no quality data to support the SBS brain injury mechanism. Thus shaking, even if done in a fit of anger, is not expected to result in head dynamics sufficient to cause direct intracerebral trauma.

“Human shaking (*id*) may cause lethal brain stem and cervical spine injuries in a 0-to-2-year-old child, as the forces necessary for these injuries are *well below* the level needed for fatal brain injuries and are consistent with the forces that can be produced in shaking. Put another way, these neck injuries would be expected in any hypothetical-superhuman strength case of SBS where superhuman dynamics resulted in head accelerations leading to intracerebral trauma (if SBS were valid, which it is not).

“If a 0- to 2-year-old child accidentally falls from a height of six feet and impacts head-first on a hard surface such as carpeted cement, the sudden impact has the potential to generate sufficient head acceleration to cause fatal intracerebral injuries. Whether any given fall is fatal depends on a host of variables and the fall mechanics which are different in each accident, but the potential head dynamics that result from a 6-foot high fall could far exceed the tolerance associated with fatal head injury.

“ Intentionally impacting a 0- to 2-year-old child’s head against a hard surface could easily cause fatal brain injuries that would mimic those of a fall, and today’s science cannot distinguish accidental from non-accidental impacts of falls of similar magnitude, barring extraordinary signs, *e.g.*...grip marks or eye-witness accounts.

“The foregoing findings are based on principles universally accepted within my field and concern scientific subject matters that I willing to testify on in this case. The findings are overwhelmingly supported by the following reference list of biomechanical tests and studies.....” (9)

These conclusions are further underscored by injuries of infants in rear-end traffic accidents with single severe hyperextension forces (whiplash) of the neck, which cause cervical fractures, dislocations, spinal cord injury, and torn nerve roots, but **not subdural hemorrhage**.(91-92)

(D) Representative References Supporting the-above Conclusions:

The Shaken Baby Syndrome: A Clinical, Pathological, and Biomechanical Study. Duhaime, AC, Gennarelli, TA, Thibault, LE, Margulies, SS et al, *Journal of Neurology*, 1987; 66:409-415.

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

Models of 1-month-old infants with various neck and skull parameters were instrumented with accelerometers and shaken and impacted against padded or unpadded surfaces. Angular accelerations for shakes Angular accelerations for shakes were smaller than those for impacts by a factor of 50. All shakes fell below injury thresholds established for subhuman primates scaled for the same brain mass, while impacts

spanned concussion, subdural hematoma, and diffuse axonal injury ranges. **It was concluded that severe head injuries commonly diagnosed as shaking injuries require impact to occur and that shaking alone in an otherwise normal baby is unlikely to cause the shaken baby syndrome.**(emphasis mine)

Anthropomorphic simulations of falls, shakes, and inflicted impacts in infants.  
Prange, MT, Coats, B, Duhaime, AC, Margulies, SS. *Journal of Neurology*, 2003;  
99:143-150:

Abstract: *Object:* Rotational loading conditions have been shown to produce subdural hemorrhage and diffuse axonal injury. No experimental data are available with which to compare the rotational response of the head of an infant during accidental and inflicted head injuries. The authors sought to compare rotational deceleration sustained by the head among free falls from different heights on different surfaces with those sustained during shaking and inflicted impact.

*Methods:* An anthropomorphic surrogate of a 1.5 month-old human infant was constructed and used to simulate falls from 0.3 m (1 ft), 0.9 m (3 ft), and 1.5 m (5 ft), as well as vigorous shaking and inflicted head impact. During falls, the surrogate experienced occipital contact against a concrete surface, carpet pad, or foam mattress. For shakes, investigators repeatedly shook the surrogate in an antero-posterior plane, inflicted impact was defined as the terminal portion of a vigorous shake, in which the surrogate's occiput made contact with a rigid or padded surface. Rotational velocity was recorded directly, and the maximum (peak-peak) change in angular velocity and the peak acceleration were calculated.

Analysis of variance revealed significant increases in the peak angular velocity and peak angular acceleration associated with falls onto harder surfaces and from higher heights. During inflicted impacts against rigid surfaces the peak angular velocity and peak angular acceleration were significantly greater than those measured under all other conditions.

*Conclusions:* Vigorous shakes of this infant model produce rotation responses similar to those resulting from minor falls, but inflicted impacts responses that were significantly higher than even a 1.5 meter fall onto concrete. Because larger accelerations are associated with an increasing likelihood of injury, the findings indicate that inflicted impacts against hard surfaces 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 1.5 m or from shaking.

Plunkett, John, Fatal pediatric head injuries caused by short-distance falls, *American Journal of Forensic Medicine and Pathology*, 2001; 22(1):1-12.

Abstract: Physicians disagree on several issues regarding head injury in infants and children, including the potential lethality of a short-distance fall, a lucid interval in an ultimately fatal head injury, and the specificity of retinal hemorrhage for inflicted trauma.

There is scant objective evidence to resolve these questions, and more information is needed. The objective of this study was to determine whether there are witnessed or investigated fal short-distance fall that were concluded to be accidental. The

author reviewed the January 1, 1988 through June30, 1999 United States Consumer Product Safety Commission database for head injury associated with use of playground equipment. The author obtained and reviewed the primary source data (hospital and emergency medical services' records, law enforcement reports, and coroner or medical examiner records) for all fatalities involving a fall.

The results revealed 18 fall-related injury fatalities in the database. The youngest child was 12 months old, the oldest 13 years. The falls were from 0.6 to 3 meters (2-10 feet). A noncaretaker witnessed 12 of the 18, and 12 had a lucid interval. Four of the six children whom fundoscopic examination was documented in the medical record had bilateral retinal hemorrhage. The author concludes that an infant or child may suffer a fatal head injury from a fall of less than 3 meters (10 feet). The injury may be associated with a lucid interval and bilateral retinal hemorrhage.

Denton, S, and Mileusnic, D, Delayed sudden death in an infant following an accidental fall; A case report with review of the literature, *American Journal of Forensic Medicine and Pathology*, 2003; 24(4):371-376.

Abstract: Several controversies exist regarding ultimately lethal head injuries in small children. Death from short falls, timing of head injury, lucid intervals, presence of diffuse axonal injury (DAI), and subdural hematoma (SDH) as marker of DAI are the most recent controversial topics of debate in this evolving field of study. In this area of debate we present a case of delayed death from a witnessed fall backwards off a bed in a 9-month-old black child who struck his head on a concrete floor and was independently witnessed as "healthy" for 72 hours postfall until he was discovered dead in bed. Grandmother, babysitter, and mother all independently corroborated under police investigation that the child "acted and behaved normally" after the fall until death. Autopsy showed a linear nondisplaced parietal skull fracture, diastasis of adjacent occipital suture, subgaleal hemorrhage with evidence of aging, small posterior clotting, subdural hemorrhage, marked cerebral edema, and a small tear of the midsuperior body of the corpus callosum consistent with focal axonal injury. No DAI was seen, and there were no retinal hemorrhages. All other causes of death were excluded upon thorough police and medical examiner investigation.....

Hall, JR, Reyes, HM, Horvat, M et al, The mortality of childhood falls, *Journal of Trauma*, 1989; 29(9):1273-1275

Abstract: Falls accounted for 5.9% of the childhood deaths due to trauma in a review of the medical examiners files in a large urban county. Falls represented the seventh leading cause of traumatic death in all children 15 years or younger, but the third leading cause of death in children 1 to 4 years old. The mean age of those with accidental falls was 2.3 years, which is markedly younger than that seen in hospital admission series, suggesting that infants are much more likely to die from a fall than older children. Forty one percent of the deaths occurred from "minor" falls such as falls from furniture or while playing; 50% were falls from one story or greater; the remainder were falls down stairs.....

Gardner, Horace, A witnessed short fall mimicking presumed shaken baby syndrome (inflicted neurotrauma), *Pediatric Neurosurgery*, 2007; 43:433-435.

An 11-month old infant was witnessed to fall backwards from a sitting position, the head striking a carpeted floor. The infant immediately cried, vomited and exhibited some seizure-like activity including tongue-biting and curling of the right hand. Three hours later a large acute left frontal subdural hematoma was removed surgically.

## **Part II: Subdural (Brain) Hemorrhages**

### **Suggested Differential Diagnoses of Infant Subdural Hemorrhages in Court Cases Attributed to SBS/NAI**

As published in *Child Maltreatment*, (2002), K.P. Hymel et al. listed 74 different conditions and diagnoses that can be caused by or associated with brain hemorrhages in infants.(12) Using these reports as a basis, the following categories are suggested as starting points in recording and addressing a differential diagnosis in the medical records of SBS/NAI court cases involving brain and/or retinal hemorrhages:

Accidental impact, including short distance falls: Plunkett,(13)(2001); Reiber,(14)(1993); Root,(15)(1992).

Birth Trauma: As reviewed in *Nelson Textbook of Pediatrics*, 16<sup>th</sup> Edition, intracranial hemorrhage in the newborn may result from birth trauma or asphyxia. This is especially likely when the fetal head is large in proportion to the size of the mother's pelvic outlet; when for other reasons labor is prolonged; in breech or precipitate deliveries,(16, 17) forceps delivery;(18) or vacuum extraction.(19-23) From *Spitz and Fisher's Medicolegal Investigation of Death*, 4<sup>th</sup> Edition, Page 1056, the more common risk factors for infant head injuries from birth trauma include prolonged labor, abrupt labor, uncontrolled delivery of the head, macrosomia, prematurity, abnormal presentations, forceps deliveries, and vacuum extractions.

According to a "Mail on Sunday" news report from England, dated 2-15-09, an estimated 200 women have been imprisoned in England annually on convictions of Shaken Baby Syndrome. Researchers looked at babies who died of brain haemorrhages before birth or shortly after who had never been out of the hospital. All had symptoms consistent with 'skaken baby syndrome' – brain & retinal bleeding, brain swelling & oxygen deficiency. But they could not have been exposed to parental violence as they were cared for by medics all the time. One of the lead authors, Irene Scheimberg, from Bart's and London NHS Trust said, "We may be sending to jail parents who lost their children through no fault of their own. Our study shows that haemorrhages of the part of the brain known as the subdural is quite common in newborns. Many of these bleeds resolve themselves with no outward sign of damage. But in some children the bleeding can continue & get worse & they then show the symptoms of shaken baby syndrome.....To suggest some babies have been harmed – without additional evidence – is dangerous" she warned.

Hydrocephalus: Piatt, JH, (24)(1999)

Prematurity and low birth weights: Intracerebral hemorrhages occur in 10% to 20% of very low birth weight babies (less than 1500 grams) and is thought to represent a substantial cause of morbidity and mortality in these patients.(25)

Hemorrhagic Disorders: With findings of brain and retinal hemorrhages, hematology consults should be routine. Late-Form Hemorrhagic Disease of the Newborn (Late-Form HDN), which is related to vitamin K deficiency, requires special attention in this category along with vitamin C deficiency. Late-form HDN is a hemorrhagic disease related to vitamin K deficiency. Brain hemorrhages occur in nearly 100% of cases. Spontaneous bruising is often prominent. Routine screening tests for suspected hemorrhagic disorders in hospital emergency rooms include the prothrombin time (PT) and partial thromboplastin time (PTT). When these are abnormally prolonged, the PIVKA test (proteins in the absence of vitamin K) should be requested. Risk factors for Late-Form HDN include prematurity, low birth weight for gestational age, birth asphyxia, traumatic delivery, and antibiotic therapy as a newborn (antibiotics kill out beneficial intestinal bacteria essential for endogenous vitamin K production).(26)

Vitamin C Deficiency (Scurvy): This possibility should always be considered with hemorrhagic manifestations. Because of its unique importance, this subject will be addressed in some depth in Part III below.

Spontaneous rebleeding of old subdural hematomas: As described by Hymel,(12) Piatt,(24) and Parent,(27) once subdural hemorrhages take place, they tend to take on lives of their own. As the red blood cells begin to lyse (break up), scavenger cells come in and begin taking up the released iron in the form of hemosiderin, which is carried out of the clot area. After 3-4 weeks the subdural hematoma takes on the consistency of “crankcase oil,” at which time it has entered the chronic state, with iron having been largely removed from the area. About two weeks following the acute hemorrhage a thin “healing membrane” begins to form around the subdural hematoma. (As a rule, all healing after injuries takes place by healing membranes.) Based on electron microscopy studies, one of the characteristics of these membranes (macrocapillries) is the frequent formation of gap junctions between adjacent endothelial cells, these gaps being large enough to spill blood cells into the subdural clot area. Based on studies of Ito et al.(28-30) it has been demonstrated that mean daily leakage of blood from the healing membranes into the subdural area amounted to 6.7% of their volumes, indicating continuing bleeding into the subdural cavity. These hemorrhages are partly activated by a process of fibrinolysis in the outer membranes of the subdural clots, which tends to liquefy and enlarge the clots. If the clots do slowly continue to expand, they will stretch the bridging veins traveling across the subdural space from the brain to undersurface of the skull, and once stretched beyond a certain point (some estimate 30-35%), these veins may rupture spontaneously or with minimal trauma and cause acute, sometimes massive rebleeding.

Estimates in timing of subdural hematomas:

As outlined in *Forensic Pathology* by Vincent J. and Dominick DiMiao.(31) “Subdural hematomas can be acute, subacute, or chronic. **Acute subdural hematomas** manifest clinically within a 3-10 day period, after which the red cells begin lysing (breaking up) and releasing their iron. This then becomes the

**Subacute phase**, during which macrophage cells move into the area and phagocytize (pick up) the iron, carrying the iron out of the clot area. Once this process is complete (within 3-4 weeks of the acute hemorrhage) the clot enters the **Chronic phase**. The timing of the acute and subacute phases can be determined by brain CT scans and MRIs with some degree of accuracy. This is because an acute subdural hemorrhage shows up on X-rays (head CT scans and MRIs) as a light gray or cream color because of the presence of iron. However, as the iron is gradually removed from the clot area by macrophage cells, the color steadily darkens into a coal-like color, very similar to that of water in the spinal fluid. Once in the chronic phase, any further timing becomes indeterminate.

*Spitz and Fisher's Medicolegal Investigation of Death*, in contrast, to DimMaio & DiMaio, describes the chronic phase as commencing at 4 weeks following an acute subdural Hemorrhage.(32)

### **Part III: Clinical Deficiencies of Vitamin C (Scurvy) as Related to Hemorrhagic Findings in Cases Attributed to SBS/NAI.**

Vitamin C Deficiency: In the 1960s and 1970s, A Kalokerinos, while working as a health officer among the Australian aborigines, became appalled by a nearly 50 percent infant mortality rate. Observing signs of scurvy in some infants, and noting that the infants commonly died following immunizations, especially if ill with common viral infections, Kalokerinos began administering regular vitamin C supplements, giving injectable vitamin C during crises, and avoiding vaccines when a child was ill, even if just a runny nose. Thereafter pediatric death rates dropped nearly to zero in his health district.(33) Since that time, Kalokerinos' observations have been supported academically by Clemetson. (34) Unfortunately, the importance of these observations has not yet been recognized, and meaningful scientific investigation into a possible connection between vitamin C deficiency and many medical complications, including vaccine reactions, remains largely unexplored.

While the recommended 30 mgs of vitamin C per day is generally adequate for a healthy infant, it may be rapidly consumed and totally inadequate when the infant is stressed or ill, as with viral or bacterial infections. The common cold, for instance, has been shown to reduce vitamin C levels in the blood by 50 percent.(35) Vaccines contain numerous toxic adjuvants (to be reviewed below) which create pro-inflammatory free radicals. All adjuvants are pro-oxidants which tend to drain the body's supply of antioxidants including vitamin C.(36) Other risk factors for vitamin C deficiency include the decline of vitamin C content in fruits and vegetables with aging, and possibly the use of microwaves for heating infant formulas.

### **Elevated Blood Histamine as Cause of Capillary Fragility and Bleeding from Scurvy**

Far from being uncommon, vitamin C deficiency does still occur in the Western World. When people attending a Health Maintenance Organization (HMO) clinic in Tempe, Arizona were tested for plasma vitamin C, it was found to be depleted (between

0.2 and 0.5 in 30 percent of subjects, and to be deficient (below 0.2 mgs/100 ml) in 6 percent.(37)

As reviewed by Clemetson, when the human plasma ascorbic acid level falls below 0.2 mg/ml, the whole blood histamine level is doubled or quadrupled.(38) Blood histamine is also increased by vaccines or toxoids, by stresses such as heat or cold, and by various drugs in guinea pigs.(39) Vitamin C has been shown to inactivate tetanus toxin (40) and diphtheria toxin.(41) It has been shown that ***bleeding from scurvy results from increased blood histamine, or histaminemia, which causes separation of endothelial cells from one another in capillaries and small venules.***(42) Subperiosteal hemorrhages (resulting in callus-like bone swellings now commonly misinterpreted as fractures), extensive spontaneous bruising, and subdural hemorrhages were included in early descriptions of classical scurvy.(43-44)

As author of this paper, it is notable that in reviewing the medical records of over 100 SBS cases in the last ten years, I have seen only one case in which plasma vitamin C blood level was tested, and even here the test was performed several months after findings of brain and retinal hemorrhages and was therefore irrelevant.

#### **Part IV: Vaccines and Lipid Peroxidation of the Brain, Resulting in Brain Inflammation and Swelling**

##### **(A) The Human Brain: Uniquely Susceptible to Lipid Peroxidation**

One of the gross errors throughout the history of childhood vaccines has been a failure to take into account the fatty nature of the brain, a large portion of which consists of polyunsaturated fatty acids and therefore highly vulnerable to inflammatory lipid peroxidation.

For definition of terms, ***fatty acids*** consist of chains of carbon atoms with hydrogen atoms attached along their sides and an acid radical at one end. An ***unsaturated fatty acid (UFA)*** is usually liquid at refrigerator temperature and is primarily derived from vegetable products and seafood. In UFAs there is an absence of hydrogen atoms between one or more carbon atoms, resulting in double bonds between the carbon atoms – thus the term “unsaturated fatty acids.” The greater the number of double bonds, the greater the unsaturation; the greater the unsaturation, the more proneness there is to rancidity (lipid peroxidation). Since polyunsaturated fatty acids have lower freezing points, they will be more abundant in the food chain of colder climates, such as seafood from northern waters.

***Lipid Peroxidation*** refers to the damaging effects of out-of-control oxygenation of fatty tissues in the body, the same process that takes place with rusting of metals or rancidity of fats. In the process of lipid peroxidation, highly reactive “free radicals” are created such as nitric oxide, superoxide, and hydrogen peroxide. The body does have enzymes designed for control of these free radicals, such as zinc-dependent superoxide dismutase for superoxide and catalase for hydrogen peroxide. However, the enzymes alone are not sufficient, as the body also requires antioxidant nutrients such as Vitamins C, E, and A, reduced glutathione, selenium, beta carotene, and retinol. Of these, probably vitamin C is the most important. *With today’s highly processed foods, these antioxidants may often be marginally low, inadequate to meet emergency needs.*

Although an infant's brain constitutes about six percent of its body weight, it receives 15 percent of normal cardiac output and accounts for nearly 25 percent of the body's oxygenation.(45) In addition to being a highly oxygenated area, the vulnerability of the human brain to harmful peroxidation rests on the fact that it has by far the highest fat content of any organ of the body with membrane lipids constituting 60% of the solid matter.(46) In addition, both brain and retina contain a relatively high percentage of the omega-3 polyunsaturated fatty acid, docosahexaenoic acid (DHA),(47-53) which serves as a primary building block of the membranes of these structures. As it is highly unsaturated, DHA is far more unstable and prone to damaging peroxidation (rancidity) than other classes of fats.

In essence, the brain might be compared with highly inflammable dry brush in a forest, in a highly oxygenated atmosphere, needing only a spark to set off a conflagration of fire-like lipid peroxidation. The peroxidative adjuvants in vaccines, in all likelihood, are providing this spark far more frequently than is generally realized.

### (B) Hazards of Free Iron in and around the Brain

Standard pediatric texts list prolonged labor, fetal malpresentation, and large babies as risk factors for significant brain hemorrhages. Tauscher et al reported an association between histologic chorioamnionitis (inflammation of the placenta) and brain hemorrhage in preterm infants.(54) Intracerebral hemorrhage occurs in up to 50 percent of very low-birth-weight infants and is thought to represent a substantial cause of morbidity and mortality in these infants.(55) Small subdural hemorrhages (SDH) are not uncommon in uncomplicated births and asymptomatic term newborns. Based on magnetic resonance imaging (MRI), Whitby et al.(56)(2004) reported subdurals in 9 of 111 infants in 2004, all of which had resolved when MRIs were repeated one month later. VJ Rooks *et al* (57)(2008) performed MRI scans on 101 term infants at 72 hours, 2 weeks, one month, and 3 months. 46 had asymptomatic SDH within 72 hours of delivery. All 46 had supratentorial SDH in the posterior cranium. 43% also had infratentorial SDH. Most SDH were < 3 mm, all of which were resolved within one month. Larger hematomas dissolved within 3 months.

Consequently, small hemorrhages are not uncommon even in uncomplicated childbirths, but little consideration has been given to the residual iron. As the red blood cells begin to lyse (break up) and release their iron following a hemorrhage, a process which takes place in two or three weeks, the iron is scavenged by white blood cells and taken up by nearby tissues in the form of hemosiderin.

Free-iron in and around the brain may also result when there are critical drops in levels of vitamin C following vaccines, followed by a precipitous rise in serum histamine, this in turn resulting in capillary fragility and leakage of blood into and around the brain.

It is known that iron overload in the liver, pancreas, and kidneys can be very destructive, a condition known as hemochromatosis. The concern here is that residual iron in and around the brain from an earlier brain hemorrhage, such as from birth trauma, may act as a lighted fuse that could ignite a firestorm of lipid peroxidation in the brain following vaccines.(58)

### (C) Vaccine Adjuvants – Their Role in Causing Prolonged Immune Responses to Vaccines and their Potentially Adverse Consequences

In what may be the most comprehensive review to date on the pathophysiology of adverse vaccine reactions, Russell Blaylock has compiled a mass of evidence that repeated stimulation of the systemic immune system results in first priming of microglia and astrocytes followed by intense microglial reaction with each successive series of vaccinations. This is the result vaccine adjuvants that are added for that purpose.(59-60)

In explanation, microglia and astrocytes are first-line-immunological responder cells located in the brain which defend against foreign infectious invaders. Normally this response, such as to a viral infection, is of limited duration and harmless to the brain. However, when the microglia and astrocytes are over-stimulated for prolonged periods, which vaccine adjuvants are designed to bring about, this extended activation can be very destructive to the brain.

Because of the critical dependence of the developing brain on a timed sequence of cytokine and excitatory amino acid fluctuations, according to Blaylock, sequential vaccinations can result in alterations of this critical process that will not only result in synaptic and dendritic loss, but abnormal (nerve) pathway development. *When microglia are excessively activated by vaccines, especially chronically, they secrete a number of inflammatory cytokines, free radicals, lipid peroxidation products, and the two excitotoxins, glutamate and quinolenic acid, which may become highly destructive for prolonge periods.* (Emphasis mine) This process was suggested as the principle mechanisms resulting in the pathological as well as clinical features of autism.(61)

Vaccine adjuvants are substances added to vaccine formulations during manufacturing that are designed to boost the overall immune system response when the vaccine is injected. These substances include albumin, several forms of aluminum, formaldehyde, various amino acids, DNA residues, egg protein, gelatin, surfactants, monosodium glutamate (MSG), Thimerosal (50% ethyl mercury, which is still in a number of vaccines)(62), and various antibiotics. Regarding mercury, even if it is not added as a preservative, it is commonly used in the manufacturing process which leaves “traces” as residues. Even these trace amounts are potentially toxic because of the universally recognized principle of toxicology that combinations of toxins will increase toxicity exponentially; that is, two heavy metals will increase toxicity 10-fold, or three heavy6t metal increase toxicity 100-fold. In vaccines the combinations would be aluminum and mercury. The same principle applies in other forms of toxic chemicals.(63-65) .

In view of these findings, R. Blaylock has referred to the inconsolable, high-pitched cry that commonly occurs following infant vaccinations as an “encephalitic cry,” as an indication of brain inflammation and swelling.

Representatives of the FDA and CDC have long denied a relationship between the mercury-containing vaccine additive, Thimerosal, and the current epidemic of childhood autism, but the following two studies should remove all doubts of such a relationship:

The first was a study conducted in Lima, Peru by J Laurente and colleagues (66) to determine if Thimerosal administration in amounts equivalent to vaccine content produced neurotoxic effects on the encephalon (brain) in postnatal hamsters and on the experimentation animals’ developments. Three serial thimerosal injections were given on

birth, days 7, 9, and 11, with controls receiving only saline injections. Test animals subsequently showed statistically significant reduction in both weight and stature compared with controls. Neurotoxic effects were also produced at encephalic (brain) level at the hippocampus, cerebral cortex, and cerebellum. On tissue slides there was decrease in neuronal density, neuronal necrosis, and axonal demyelination in test animals.

The second study conducted by Sajkel-Wulkowska *et al* (67)(2008) indicates otherwise. This study was also a first in comparing the cerebellar levels of the oxidative (inflammatory) stress marker 3-nitrotyrosine (3-NT), mercury (Hg), and the (protective) antioxidant selenium (Se) between autistic and normal children. Average cerebellar 3-NT levels were statistically elevated in autism by 68.9%, cerebellar Hg elevated by 68.2%, and mercury levels relative to selenium elevated by 75% in autistic cases in comparison to controls.

In vaccines, virtually insoluble polymeric aluminum hydroxyl compounds serve to dramatically boost and prolong the immune reaction to the vaccination by prolonged activation of the macrophagic immune sub-system in some people.(68-73)

Because vaccine adjuvants are designed to produce prolonged immune stimulation, they pose a particular hazard for the nervous system. Studies have shown that immune activation following vaccines can last up to two years, which means that destructive overstimulation of microglia may also be primed for this length of time or even longer. In addition, it is known that aluminum accumulates in the brain and that this accumulation is associated with Alzheimer's disease, Parkinson's disease, and Gulf War Syndrome.(73-75)

In regard to the Shaken Baby Syndrome, H Gardner has observed that in Japan nontraumatic brain hemorrhages occur later with the incidence peaking at age seven to nine months, while in the U.S.A. the incidence peaks at two to four months, corresponding with the recommended pediatric vaccination schedules in both countries.(76)

A study on primary immunization of 239 premature infants with gestational ages of less than 35 weeks by M Pourcyrous *et al*. (77)(*Journal of Pediatrics*, 2007) to determine the incidence of cardio-respiratory events and abnormal C-reactive protein (CRP) levels associated with administration of a single vaccine or multiple vaccines simultaneously at or about two months age. (CRP is a standard blood test indicator for body inflammation.) CRP levels and cardio-respiratory manifestations were monitored for three days following immunizations in a neonatal intensive care unit sponsored by the University of Tennessee. Elevations of CRP levels occurred in 70% of infants administered single vaccines and in 85% of those given multiple vaccines, 43% of which reached abnormal levels. Overall, 16% of infants had vaccine-associated cardiorespiratory events with episodes of apnea (cessation of breathing) and bradycardia (slowing of pulse). **It can be reasonably assumed that the cardiorespiratory events and CRP elevations primarily reflect brain inflammation and swelling following the vaccines. Most important for our present topic, intraventricular (brain) hemorrhages occurred in 17% of infants receiving single vaccines, with 24% incidence in those receiving multiple vaccines. For the first time this study documents that a significant incidence of brain hemorrhages can result from vaccines in vulnerable infants.**

(D) Probable Mechanism by which Vaccine Induced Encephalitis May Cause Subdural Hematoma, Retinal Hemorrhage, and/or Sudden Onset of Apnea.

It has long been known from animal experiments that vaccines can cause encephalitis with swelling of the brain, often also associated with hemorrhage.(78-80) Small hemorrhages usually resolve in a few days, but when the brain swelling is more than slight, as manifested by bulging anterior fontanelle, the brain surface will impact against the unyielding inner surface of the skull. This in turn will tend to cut off the outflow of venous blood from the brain that normally flows passively through bridging veins to the inner surface of the skull and from there back into the systemic circulation. The arterial flow, in contrast, coming in at a much higher pressure through other circuits, produces a precipitate rise in intracranial venous pressure. According to W. Squier and J Mack, (*Forensic Science Intern.*, 2009)(81), most infant subdural hemorrhages take place as a result of blood seepage into the immature subdural membranes themselves, which in infancy are made up of 10 to 15 layers of loosely arranged flake-like cells with fluid-filled spaces between the layers. These open spaces readily allow seepage of blood which form thin membranes, as described by Whitby (56) and Rooks (57).

In regard to **retinal hemorrhages**, the process would be much the same. There are only three outlets for the brain once the brain impacts against the inner surface of the skull: the two eye sockets and the opening into the spinal canal at the base of the brain. Once brain swelling begins impinging on the inner surface of the eye sockets, the relatively low pressure of venous outflow will immediately be cut off, with the brain acting as its own tourniquet. With continual arterial inflow at much higher pressures, there would be precipitous rises in central venous pressure and consequent retinal hemorrhages.

A review of the medical literature on retinal hemorrhages is entirely compatible with this concept. AC Tongue mentions that “hemorrhages in all layers of the retina occur in a number of nontraumatic disorders associated with changes in cerebrovascular dynamics such as central vein retinal occlusion, high altitude retinopathy, and subarachnoid hemorrhage secondary to ruptured intracranial aneurysms.”(82) *Any sudden increase in intracranial venous pressure may cause retinal hemorrhages.*(83-89) Furthermore, retinal and optic nerve sheath hemorrhages associated with a ruptured vascular malformation are due to an increase in central intracranial venous pressure.(85-86) Patrick Barnes reported that retinal hemorrhages may be seen with a variety of conditions including accidental trauma, resuscitation, increased intracranial pressure, increased intracranial venous pressure, subarachnoid hemorrhage, coagulopathy, and certain metabolic disorders.(90)

Finally, the breathing center at the base of the brain, the **arcuate nucleus**, may also be highly vulnerable to vaccine-induced encephalitis because of the high incidence of abrupt onset of apnea (respiratory arrest) that is seen in these cases.

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