

# Childhood Immunizations and Abrupt-Onset Apnea: An Unresolved Issue in Shaken Baby Syndrome

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## Introduction

Based on personal experiences of the authors in reviewing many cases diagnosed as shaken baby syndrome (SBS), there has been a common pattern of unexpected and sudden onset of apnea with respiratory collapse (cessation of breathing) in a time-related fashion following routine childhood immunizations. For the most part these collapses have occurred during the first 6 or 7 months of life during the time period of routine 2, 4, and 6-month immunizations. It is the purpose of this article to review existing medical evidence and literature indicating that it is both possible and plausible that there is a direct causal relation between immunizations and the abrupt onset of apnea seen in many SBS cases. Part of this evidence comes from disclosures from ongoing US Congressional hearings on issues of vaccine safety sponsored by the Congressional Committee for Government Reform, which have revealed fundamental deficiencies in scientific infrastructure and safety testing of vaccines; and that as a result of these deficiencies many adverse vaccine reactions are taking place unrecognized. Part comes from scientific publications by primary researchers in the field of biomechanics indicating that shaking alone cannot generate sufficient force to cause brain damage or brain hemorrhages in infants, and that some of the fundamental doctrines of shaken baby syndrome are based on assumptions not supported by

scientific evidence. Part comes from limited but specific medical literature indicating vaccines as a potential cause of apnea. Based on these different lines of evidence, it is both possible and plausible that many vaccine and SBS cases are being misdiagnosed. Each of these areas will be addressed in the following:

### **Deficiencies in Scientific Safety Infrastructure of Childhood Vaccines**

As reviewed in current Physician's Desk References, potentially toxic and/or sensitizing substances found in childhood vaccines may include aluminum phosphate, mercury, formaldehyde, phenols, alcohols, mineral oils, antibiotics, animal serums, animal DNA, and aborted fetal tissue. In addition, the Hepatitis B vaccine, which is cloned in yeast cells, runs the risk of causing sensitivity reactions in infants who may be sensitive to yeast. Based on this list alone, one would think that safety considerations in the combinations and scheduling of vaccines would be considered of preeminent importance, but has this actually taken place?

Since 1999 there have been ongoing hearings in the U.S. Congress concerning growing concerns about vaccine safety. Primarily these hearings have dealt with concerns about a possible link between vaccines and growing epidemic of childhood autism in the USA. From these hearings there is now an emerging background pattern of deficiencies in basic science in vaccine safety testing. (1) As a result of these deficiencies it is reasonable to assume that many vaccine reactions are taking place unrecognized as to their true nature, especially those of a delayed nature. Based on these hearings, scientific evidence does not support the safety of immunizations in that safety studies have been limited to short periods only: several days to several weeks. There are no long-term (months or years) safety studies on any childhood vaccine in use today.

There have been no controlled, systematic before-and-after studies on the effects of vaccines on the immune, hematologic, brain, and neurologic systems of babies, studies which should be considered indispensable for any ongoing medical intervention. As an example of this type of before-and-after testing, as reported in the New England Journal of Medicine in 1984, 11 healthy adults had tests involving T-lymphocyte subpopulations (white blood cells) following routine tetanus booster vaccines showing a significant though temporary drop in T-helper lymphocytes.(2) Special concern rests in the fact that in 4 of the subjects the T-helper lymphocytes dropped to levels found in active AIDS patients. If this was the case with healthy adults, it is sobering to think of the immune consequences of the multiple vaccines given to infants with their immature and vulnerable immune systems. This test should have served as a pilot for further safety studies, but as far as can be determined by surveys of the literature, it has never been repeated.

There has been inadequate consideration to the additive or synergistic adverse effects of multiple simultaneous vaccines, although in cases of toxic chemicals, two chemicals together may be 10 times as toxic as either separately, or 3 chemicals 100 times more toxic. (3-4)

## **Medical-Legal Issues**

As reviewed in “the amicus brief for SBS,” and provided through the courtesy of Toni Blake, (5) one of the current beliefs on which SBS accusations and convictions are based is that shaking alone in an otherwise healthy child can cause a subdural hematoma, there are a number of publications which lead to the conclusion that this syndrome is an assumption which is not supported by scientific evidence. Foremost among these is an article by Mark Donohoe (2003, American Journal of Forensic medicine and Pathology) in which he stated that half of articles about SBS were published before 1999 and half after 1999. Given that 1998/1999 is regarded as a turning point in acceptance of the tenets and practice of evidence-based medicine, it seemed reasonable to the author to assess the quality of evidence before 1999 and to compare it with the quality of evidence on the same subject since that time. Qualities of evidence were placed in IV broad categories, with level I or level II evidence showing compelling evidence from consistent findings in 2 or more well-constructed, controlled trials or population-based epidemiologic studies. In contrast, clinical practice guidelines with level IV evidence represent consensus statements of the expert panel according to clinical experience and limited scientific data. Following a review of articles on SBS published before 1999, Donohoe found that the majority of evidence showed a level of IV, “opinions that shed no new light upon SBS and did not add to knowledge about SBS.” None were found that exceeded a level III-2. (6)

In the fall issue of The Warrior, Journal of the Trial Lawyers College, 2003, Attorney Elaine Whitfield Sharp wrote a comprehensive article reviewing the history of SBS. (7) Beginning in 1966 and 1968 Ayub K Ommaya, MD, a Pakistani-born and Oxford-educated neurosurgeon, set out to determine the amount of force it takes to cause certain types of brain injuries and hemorrhages from rear-end car crashes. In experiments with Rhesus monkeys (experiments now prohibited), Ommaya used the monkeys to mimic car accidents by accelerating them on chairs fixed to a track and decelerating them without impact to their heads. Ommaya's experiments showed that it took between 35,000 to 40,000 radians per second (squared) of angular or rotational acceleration to cause brain hemorrhages in the monkeys. Transposing the size of monkey brains to that of human brains, Ommaya calculated that the amount of force required to cause brain hemorrhages in humans would be 6,000 to 7,000 radians. According to the review by Sharp, other notable names in the field of SBS transposed Ommaya's findings to the field of SBS.

It was not until 1987 that a bio-mechanician and a group of neurosurgeons set out to prove that subdural hemorrhages in babies were not caused by shaking but by impact. The bio-mechanician was Lawrence E Thibault who, with team members made a surrogate baby model and attached an accelerometer to its neck. First they asked some burley football players to shake the model as hard as they could. The most force they were able to generate was a mean of 1,138 radians, far below the 6,000 to 7,000 radians required to cause human brain hemorrhages. (8) Other publications since that time tend to confirm rather than falsify these findings, one example being a report by Prange, Coats, Duhaime, and Margulies which concluded that “there are no data showing that the maximum change in angular velocity... during shaking and inflicted impact against unencased foam is sufficient to cause subdural hemorrhages or primary traumatic axonal (nerve) injury in an infant.” (9)

### **Increased Hazards of Vaccines in Premature Infants (10)**

As reviewed in a previous article in Townsend Letter, (11) a series of unpublished cases of SBS collected by Toni Blake, jury counselor of San Diego, found a striking pattern of subdural (brain) hemorrhages occurring in “fragile infants” in a time-related fashion following routine immunizations given during the first 6 months of life. Risk factors included prematurity, low birth weight, maternal drug or alcohol problems, maternal diabetes, or toxemia of pregnancy. Among these risk factors, the best information to date connecting vaccines to apnea is to be found in studies of premature infants. Because of their importance, five of these will be reviewed below.

The authors of many well-documented studies have concluded that the risk and benefit of vaccination in preterm infants should be evaluated prior to administering the vaccines. They also emphasized that preterm infants who have received vaccines should be monitored. The following are descriptions of several selected studies conducted in the USA and other countries to illustrate these points.

In a report in *Acta Paediatr* (2001), case histories of 45 preterm babies who were vaccinated with DPT/Hib (diphtheria, tetanus toxoids, and pertussis and *Haemophilus influenzae* type B conjugate) in the neonatal intensive care unit of the Royal Gwent Hospital, Newport, UK between January 1993 and December 1998 were studied retrospectively. (12) Apparent adverse events were noted in 17 of 45 (37.8%) babies; 9 (20%) had major events, i.e. apnea, bradycardia or desaturations, and 8 (17.8%) had minor events; i.e. increased oxygen requirements, temperature instability, poor handling and feed intolerance. Age at 70 days or less was significantly associated with increased risk ( $p < 0.01$ ). Of 27 babies vaccinated at 70 days or less, 9 (33.3%) developed major events compared with none when vaccinated over 70 days. The authors concluded that vaccine-related cardiorespiratory events are relatively common

in preterm babies. Problems were much more common if vaccine is administered at or before 70 days. Therefore babies should therefore be monitored post-vaccination.

After observing the occurrence of apnea (a respiratory pause of 20 seconds or longer, usually associated with bradycardia, heart rate less than 80 beats/min) in two preterm infants following immunization with DTP and Hib, Sanchez et al

(1997) conducted a prospective surveillance of 97 (50 girls and 47 boys) preterm infants younger than 37 weeks of gestation who were immunized with DTP (94 also received HibC at the same time) in the neonatal intensive care unit in Texas USA to assess the frequency of adverse reactions, and in particular the occurrence of apnea. For each infant data were recorded for a 3-day period before and after receipt of the immunizations. (13) Their study showed that apneic episodes occurred in 34 infants (34%) after immunizations. Twelve infants (12%) experienced a recurrence of apnea, and 11 (11%) had at least a 50% increase in the number of apneic and bradycardia episodes in the 72 hours after immunization. This occurred primarily among smaller preterm infants who were immunized at a lower weight ( $p=0.01$ ), had experienced more severe apnea of prematurity ( $p=0.01$ ), and had chronic lung disease ( $p=0.03$ ). Some of these infants required new medical intervention for the increased apneic/bradycardiac episodes. (23)

Bothan et al (1997) conducted a prospective study of 98 preterm infants (53 males and 45 females) of gestational age 24-31 weeks who were immunized at approximately 2 months postnatal age with diphtheria-tetanus-whole cell pertussis vaccine (DTPw) in the neonatal intensive care unit (NICU) at King George V Hospital in Sydney, Australia. Half the infants also received Haemophilus influenzae type b conjugate vaccine (Hib) simultaneously. All infants were monitored for apnea and bradycardia in the 24 hour periods pre-and post immunization. Their study showed only one infant had apnea and/or bradycardia pre-immunization compared with 17 post-immunization. For 12 infants these events were brief, self-limiting and not associated with desaturations (oxygen saturation  $<90\%$ ). However, for five infants (30%) these events were associated with oxygen desaturation, and two of these infants required supplemental oxygen. When considering immunization for preterm infants, the benefits of early immunization must be balanced against the risk of apnea and bradycardia. (14)

Slack et al., (1999) from the United Kingdom stated that four premature infants developed apneas severe enough to warrant resuscitation after immunization with diphtheria, pertussis, and tetanus (DPT) and Haemophilus influenzae B (Hib). One required intubation and ventilation. They also reported that, although apneas after immunization are recognized, they are not well documented. They concluded that it is time for further research to elucidate the best time to immunize such infants. (15)

Botham et al ( 1994) conducted a prospective study in ninety-seven preterm infants who were immunized with diphtheria-tetanus-pertussis to document respiratory and cardiac events. (16) The mean gestational age at birth was 28.1 weeks (range 24 to 34) and the mean age at immunization was 80.6 days (range 44-257). They found that nineteen (20%) infants developed apnea or bradycardia within 24 hours of immunization. The infants who developed apnea and/or bradycardia had a younger gestational age at birth than those who did not ( $p=0.03$ ), were artificially ventilated for longer ( $p=0.01$ ), and were more likely to have a diagnosis of chronic lung disease ( $p=0.006$ ). Two infants who developed concurrent upper respiratory tract infections required additional oxygen, and one of them was treated with oral theophylline. They stated that cardiorespiratory function should be monitored after immunization in very preterm infants who had prolonged ventilatory support and/or chronic lung disease.

In Nelson Textbook of Pediatrics, 16 th Edition, there are precautions in the use of potentially toxic medications in premature infants because of diminished renal clearances for almost all substances excreted in the urine (17) The text also cautions about drugs that are detoxified in or conjugated by the liver in premature infants. Should not vaccines be included in the category of potentially toxic medications? The five references cited above show unmistakable evidence that there is a significant increase in apneic episodes following immunizations, to which preterm/premature are exceptionally vulnerable.

### **Diphtheria-Pertussis-Tetanus (DPT) Vaccines and Infant Apnea in Sudden Infant Death Syndrome (SIDS)**

According to a report by WC Torch of Reno, Nevada in 1986, over 150 DPT-postvaccinal deaths had been reported in the literature by 37 authors in 12 countries. (18) Although 90% of reactions occurred with one week of DPT, the remainder as long as 20 months following protracted reactions. About one-half were sudden infant death-like (SIDS) or anaphylactic; about one-half followed neurotoxic or systemic symptoms (dyspnea, seizures, shock, irritability, lethargy, apathy, coma, decerebrate-decorticate rigidity, spasticity, hypotonia, paralysis, or apnea ). In deaths within 3 hours of DPT the brain was normal; between 6 and 72 hours, varying degrees of brain edema, vascular congestion, petechia or (brain) hemorrhage, monocytic infiltrates, and neuronal degeneration were seen. In some later deaths demyelination, gliosis, or atrophy was seen. The author and others maintained a causal relationship between DPT vaccine and yet-to-be determined SIDS fraction.

(NOTE: although the former DPT has been replaced with the a-cellular DTaP, many current cases of shaken baby syndrome involve infants who received the DPT vaccine, so that this report remains pertinent. Also, lacking appropriate and definitive



studies, there is no assurance that the same process may not still be taking place with the DTaP vaccine).

## **Vaccines, Vitamin C Depletion and Apnea**

In the next 25 years or so, when there is greater knowledge about the adverse reactions and aftermath from current childhood vaccine programs, physicians and scientists, as well as the lay public, may look back on these programs with embarrassment if not worse. This is not to say that vaccines cannot have a proper role in preventive health, but not with neglect in safety considerations.

The rationale for these statements is based in part on the work of Dr. Archivedes [Kalokerinos M.D.](#), who worked as a medical officer among the Australian aborigines in the “outback” in the 1960s and 1970s. Being troubled by very high infant mortality, in some areas approaching 50%, he began to investigate possible causes. Having noticed signs of scurvy in some of the infants, and observing that the children often died following immunizations, especially if they had colds or minor respiratory infections, the thought occurred to him that there might be a connection between vitamin C deficiency and deaths following vaccines. With improved nutrition, routine oral vitamin C supplementation of children and infants, avoidance of immunizations during minor illnesses, even if just a runny nose, and large doses of injectable vitamin C during crises, infant mortality was virtually abolished. Although [Kalokerinos M.D.](#) was awarded the Australian Medal of Merit in 1978 for his work, it has never been acknowledged by mainstream medicine. What is worse, it has never been subjected to systematic, meaningful scientific studies.

In contrast to classical scurvy of earlier times in the days of wooden sailing ships, when scurvy was characterized by a total lack of Vitamin C, what we may be seeing today is something quite different. As described by Dr. [Kalokerinos M.D.](#) (19) and Alan [Clemetson](#), MD (20) subclinical scurvy is a condition in which apparently healthy infants with marginally low but adequate levels of Vitamin C in unstressed conditions may be suddenly thrown into states of critical Vitamin C depletion by combinations of stresses from common infections and toxins, including the toxins found in vaccines. As one example of marginal Vitamin C deficiency on the modern scene, in a study of people attending an HMO (Health Maintenance Organization Clinic) in Tempe, Arizona in 1998, 30% were found to be depleted with plasma Vitamin C levels between 0.2 and 0.5 mgs/100 ml and to be deficient in 6% with levels below 0.2%. (21) In regards to infants, it is true that infant formulas have been mandated to include Vitamin C at levels providing the required 30 mgs per day. However, this is a maintenance level and makes no allowances for additional stresses which may bring about many-fold increases in need for Vitamin C. Common colds, for instance, have been shown to reduce Vitamin C levels up to 50%. (22) No one knows the effects of

vaccines on Vitamin C levels in infants, because before-and-after studies of this type have never been done, but Vitamin C is known to neutralize the toxins of diphtheria, (23-26) tetanus, (27) typhoid endotoxin, (28) and four varieties of gas gangrene. (29) As will be described below, in the process of neutralizing these toxins, Vitamin C is necessarily used up and depleted.

If the reader will consult with these references, which were extracted from an article by A [Clemetson](#), (30) it will be found that most of these studies are quite old and some published in foreign languages. To us that is the pity of it, as our own scientific & medical system has never recognized their importance or followed through with further investigation.

Returning to the importance of vitamin C in relation to vaccines, one of the prime roles of Vitamin C in the body is its action as an antioxidant in donating electrons to quench free-radical inflammatory damage from infections and/or toxins, with our consideration here being vaccine toxins. However, in the process of donating electrons, Vitamin C necessarily becomes depleted. Once the level of Vitamin C is reduced to the point that it can no longer protect the brain, which is unduly susceptible to toxic and infectious damage, it (the brain) may become subject to free-radical damage. By definition a “free-radical” consists of a molecular fragment with one or more unpaired electrons in its outer orbital ring, causing it to be highly oxidative, unstable, and to react instantaneously with other substances in its vicinity. Within a few millionths of a second, free radicals have the potential to react with and damage nearby molecules and cell membranes with a chain reaction of damage. (31-33) When uncontrolled, these can be very destructive to the body, such as may take place when exposed to harmful radiation. Vitamin C is critically important in protecting against free-radical proliferation because in donating electrons, it neutralizes the unpaired electrons in the “free-radical” molecular fragments. Of all the organs of the body, the brain appears to be most vulnerable to this type of damage because of its relatively high fat content.

For these reasons, combinations of vaccines given to fragile infants may be an invitation to disaster with the brain being potentially subjected to a firestorm of free-radical inflammatory damage. Once this pattern has been set in motion, there is a variable latent period with gradual progression of inflammatory brain edema (swelling). The breathing center, located at the base of the brain, appears to be uniquely vulnerable to the process. This in turn may result in respiratory paralysis and collapse. In other instances there may be seizures. Among the cases of SBS that we have reviewed, this has been a common pattern, too frequent to be coincidental.



As described in his autobiography, Dr. [Kalokerinos M.D](#) describes the mechanisms involved in the production of brain edema with retinal and brain hemorrhages in much the same fashion: (18)

“1. Endotoxin (endogenous and/or from vaccines) damages the endothelial linings of the brain's blood vessels.

2. endotoxin then ‘leaks’ through to the surrounding brain tissue. This includes the retina that is an extension of the brain.

3. The brain tissue is damaged.

4. The blood supply to the portions of the brain involved is reduced.

5. Insufficient oxygen, glucose, and Vitamin C follows.

6. Parts of the brain are ‘rich’ in ‘bound’ (controlled) iron. This is released.

7. Violent free radical reactions result, and these cannot be controlled because of a lack of immediately available Vitamin C and other antioxidants.

8. So further, and rapid, brain tissue damage results, with more free radical reactions.

9. Hemorrhages occur in the area/areas involved.

10. After a variable period (depending on a host of factors) some of the red blood cells in the hemorrhages break down and release their stores of iron and copper.

11. This results in a further cascade of free radical reactions and tissue destruction.

12. Cerebral edema (brain swelling) occurs.”

By way of comparison, in Vienna in the 1840s, long before recognition of the importance of sanitation and the role of microbes in causing disease, a doctor named Ignaz Semmelweis was assigned to an obstetrical post at a birthing center which was notorious for its high maternal mortality rates. Based on simple observation, Semmelweis deduced that doctors and nurses were carrying infections from one patient to another and subsequently required that they wash their hands between patients. As a result, the mortality rate among maternity patients under his care was reduced from nearly 30% in other wings of the hospital to less than 2% for patients under his care or supervision.

Was Semmelweis honored by his peers for this discovery? No, at least not at that time. Instead he was dismissed from the hospital staff because his procedures did not conform with the medical thinking of the time. In the cases of Drs.

Archivedes [Kalokerinos M.D](#) and CA [Clemetson](#), could history be repeating itself?

### **Inverse Relations between Plasma Ascorbic Levels and Whole Blood Histamine; Elevated Histamine the True Cause of Capillary Fragility in Scurvy**

In 1980 A [Clemetson](#) reported that the whole blood histamine levels of human subjects are inversely proportional to their plasma vitamin C levels, (34) in that 34 percent of people who had subnormal but not deficient ascorbic acid levels were found to have significantly increased blood histamine concentrations. The 2 percent of subjects who were markedly vitamin C depleted (<0.2 mg/100 ml) had a four-to-five-fold increase in their blood histamine concentrations. Frank scurvy does not occur until blood histamine is increased more than ten-fold. Nevertheless, the blood histamine concentration returns to normal very rapidly following the oral administration of ascorbic acid.

Indications that elevated blood histamine is the true cause of capillary fragility in scurvy comes from electron-microscopic studies by Gore et al in guinea pigs with scurvy, in which widening of the intercellular junction gaps were demonstrated in the vascular endothelium. (35) Moreover, Majno and Palade have observed similar widening of the endothelial junction gaps and leakage of tracer particles through endothelial gaps in rats following the injection of histamine. (36) Consequently it seems that histaminemia is the crucial factor causing bleeding in scurvy and may be responsible for the fragility of the bridging veins and venules between the brain and the dura mater, as well as the retinal petechiae.

As a matter of opinion, [Clemetson](#)'s work in elucidating the inverse relationship between vitamin C and blood histamine levels, with elevated histamine being the primary cause of capillary fragility, is of critical importance in shaken baby syndrome, so that there should be mandatory requirements for obtaining blood plasma levels of vitamin C and whole blood histamine in hospital emergency rooms before bringing charges of SBS.

### **An Hypothetical Analysis of the Vaccine/Apnea Connection and its Pathogenesis in Causing Brain and Retinal Hemorrhages**

Current theories surrounding shaken baby syndrome maintain that subdural and/or retinal hemorrhages are diagnostic of shaken baby syndrome in absence of known accidental trauma. In opposition to this assumption, Jennian Geddes, Neuropathologist at Royal London Hospital, and colleagues have shown evidence that many of these

cases are the result of injuries to the respiratory center located at the base of the brain, injuries not necessarily involving either violence or impact. (37-38) Once respiratory collapse takes place, brain swelling rapidly ensues as a result of hypoxia according to Geddes. Locked as it is inside a rigid skull, the brain then becomes its own tourniquet, quite effectively blocking off venous blood outflow from the brain. As a result there is an increase in central venous pressure, the true cause of subdural and retinal hemorrhages, as well as the primary cause of apneic episodes in these infants. ([39-41](#))

Assuming next that vaccines can and do bring about respiratory collapse in infants, what would be the mechanism? Based on evidence provided here, it is almost certain in these cases that there is smoldering brain inflammation with gradual swelling of the brain. This may reach the point where the respiratory center at the base of the brain becomes constricted from the brain swelling, or possibly herniated into the spinal canal. In the absence of brain edema, it may be the effects of the vaccine toxins.

## **Conclusion**

Based on our review of the cited literature, on our involvement as consultants in medico-legal cases involving vaccines, shaken baby syndrome, and pediatric head injury, and on the growing concerns of safety in pediatric drug/biologic administration, we see a common pattern of apneic conditions following immunizations. These findings in turn are often (even routinely) being attributed to parents or caretakers with false charges of child abuse. Meaningful and objective investigation is warranted, as we believe many children are suffering unrecognized vaccine reactions, and many parents/caretakers are being falsely accused, some even imprisoned for life.

## **References:**

1. Miller NZ, Vaccines, Autism and Childhood Disorders, New Atlantean Press, PO Box 9638, Santa Fe, New Mexico 87504, 2003. (This book provides an excellent overview of the US Congressional Hearings on issues of vaccine safety that have taken place on a regular basis for the past several years).
2. Eibl M et al, Abnormal T-lymphocyte subpopulations in healthy subjects after tetanus booster immunization, (letter), NEJM, 1984; 310(3):198-199.
3. Arnold SF et al, Synergistic activation of estrogen receptor with combinations of environmental chemicals, Science, 1996; 272:1489-1472.

4. Abou-Donia AB et al, Neurotoxicity resulting from exposure to Pyridostigmine bronide, DEET, and Permethrin; implications of Gulf War chemical exposures, J Tox & Environ Health, 1996; 48:35-36.
5. The Amicus Brief was prepared by Toni Blake, attorney and jury counselor of San Diego, and coworkers for presentation in court in cases dealing with the shaken baby syndrome.
6. [Donohoe M](#), Evidence-based medicine and shaken baby syndrome, Part I: Literature review, 1996-1998, Am J Forensic med Path, September, 2003; 24(3):239-242.
7. Sharp EW, The Elephant on the Moon, The Warrior, Fall, 2003:28-39.
8. Margulies SS, Thibault LE, an analytical model of traumatic diffuse brain injury, J Biomech Engineering, 1989; 111:241-249.
9. Prange MT, Coats BS, Duhaime AC, Margulies SS, Anthropomorphic simulations of falls, shakes, and inflicted impacts in infants, J Neurosurgery , 2003; 99:143-150.
10. The references under this subject were provided through the courtesy of [Mohammed Ali Al-Bayati, PhD, DABT, DABVT](#), Toxicologist and Pathologist, Toxi-Health International, 150 Bloom Drive, Dixon, California 95620.
11. [Buttram HE, Shaken baby syndrome or vaccine-induced encephalitis?, Townsend Letter for Doctors & Patients, October, 2003:72-78.](#)
12. Sen S, Cloete Y, Hassan K, Buss P, Adverse events following vaccination in premature infants, Acta Paediatr, 2001, 33(5):418-421.
13. Sanchez PJ, laptook AR, fisher L et al, Apnea after immunization of preterm infants, J Pediatr, 1997; 130(5):746-751.
14. Botham SJ, Isaacs D, Henderson-Smart DJ, Incidence of apnoea and bradycardia in preterm infants following DTP immunization: a prospective study, J Paediatr Child Health, 1997; 33(5):418-421.
- 15.. Slack MH, Schapira D, Severe apnoeas following immunization in premature infants, Arch Dis Child Fetal Neonatal Ed, 1999; 81(1):F67-68.
16. Botham SJ, Isaacs D, Incidence of apnoea and bradycardia in preterm infants following triple antigen immunization, J Paediatr Child Health, 1994; 30(6): 533-535.

- 17 Nelson Textbook of Pediatrics, 16 th Edition; Behrman, Kliegman, & Jenson Editors, WB Saunders Co., Philadelphia, 2000, Page 483.
18. Torch WC, Characteristics of Diphtheria-Pertussis-Tetanus postvaccinal deaths and DPT-caused sudden infant death syndrome (SIDS): a review, Neurology (Suppl 1), April, 1986.
19. [Kalokerinos](#), A, Medical Pioneer of the 20 th Century, an Autobiography, Dr. Archivedes Kalokerinos, Biology Therapies Publishing, Braeside, Melbourne, Victoria, Australia, Fax 011-61-39587-1720, Publ.2000.
20. [Clemetson](#) CAB, Vitamin C, Volume I in a 3-volume set, CRC Press, Boca Raton, 1989, pages 215-221.
21. Johnston CS, Thompson MS, Vitamin C status of an out-patient population, J Amer Col Nutr, 1998; 17:366-370. Hume R, Weyers E, Changes in the leucocyte ascorbic acid concentration during the common cold, Scot Med J, 1973; 18:3.
22. Zvirbely JL, Szent-Gyorgyi A, The chemical nature of vitamin C, Biochem J, 1932; 27:279-285.
23. King CG, Waugh WA, The chemical nature of vitamin C, J Science, 1932; 75:357-358.
24. Harde E, Acide ascorbique (vitamin C) et intoxications, CR Acad Sci, 1934; 119:618-620.
25. Parrot JL, Richet, Accroissement de la sensibilité a histamine chez le cobaye sournis a un regime scorbutogene, CR Soc Biol, 1945; 139: 1072-1075.
26. Dey PK, Efficiency of vitamin C in counteracting tetanus toxin toxicity, Naturwissenschaften, 1966; 53:310.
27. Fukada T, Koyama T, Prevention by ascorbic acid of liver glycogen depletion in endotoxin intoxication, Nature, (London) 1963; 200:1327.
28. Buller Souto A, Lima C, Activity of L-ascorbic acid on the toxins of gas gangrene, Vol 12, Sao Paulo, Brasil: Memorias do instituto Butantan, 1939:265-295.
29. [Clemetson](#) A, Barlow's disease, Medical Hypothesis, 2002; 59(1):52-56.
30. A Textbook of EDTA Chelation Therapy, Second Edition, Elmer Cranton Editor, Hampton Roads Publishing Co, Charlottesville, VA, 2001, Pages 13-27. (Note: this

chapter gives definition and description of free radicals, primarily as concerns lipid metabolism in the human body.

31. Chemical Sensitivity, Volume I (volume one of four volumes), William J Rea, MD, Lewis Publishers, Boca Raton, 1992, (pages 122-124 discuss the role of pollutants in creating free radicals).
32. Casarett & Coull's Toxicology, the Basic Science of Poisons, Curtis D. Klaassen, McGraw-Hill, New York, 2001, pages 40-42.
33. [Clemetson](#) CAB, Histamine and ascorbic acid in human blood, J Nutrition, 1980, 110:662-668.
34. Gore I, Fujinami T, Shirahama T, Endothelial changes produced by ascorbic acid deficiency in guinea-pigs, Arch Pathol, 1965; 80:371-376.
35. Majno G, Palade GE, Studies on inflammation. 1. The effect of histamine and serotonin on vascular permeability. An electron microscopic study. J Biophys Biochem Cytol, 1961; 11:571-605.
36. Geddes JF, Hackshaw AK, Vowles GH et al, neuropathology of inflicted head injury in children, 1. patterns of brain damage, Brain, July, 2001; 124(7):1290-1298.
37. Geddes JF, Tasker RC, Hackshaw CD et al, Dural haemorrhage in nontraumatic infant deaths: does it explain the bleeding in 'shaken baby syndrome,?' Neuropathol & Applied Neurobiol, 2003; 29:14-22.
38. Smith DC, Kearns TP, Sayre GP, Pre-retinal and optic nerve sheath hemorrhage: pathologic and experimental aspects in subarachnoid hemorrhage, Trans Am Acad Ophthalmol Otolaryngol, 1957; 61:201-211.
39. Lehman RAW, Krupin T, Podos SM, Experimental effect of intracranial hypertension upon intraocular pressure, J Neurosurgery, 1972; 36:60-66.
40. Edlow JA, Caplan LR, Avoiding pitfalls in the diagnosis of subarachnoid hemorrhage, New Engl J Med, 2000; 342:29-36.