# Shaken Baby Syndrome or Vaccine-Induced Encephalomyelitis?
# The Story of Baby Alan

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**Introductory Note:** Among the many adversities and difficulties facing the American family today, the following article was written to point out a relatively new and growing hazard in which a parent or caretaker may be falsely accused of murdering or injuring an infant by the "shaken baby syndrome," (SBS) when the true cause of death or injury arises from other sources. Very tragically, child abuse does occur and deserves appropriate punishment. However, it is equally tragic when a family, already grieving from the death of their infant, finds a father, mother, or a caretaker injustly accused, convicted, and imprisoned for murder of the infant, a murder of which he or she is innocent. The authors of this article have knowledge of an attorney, an anesthesiologist, a Mormon mother, an Amish mother and others accused and/or imprisoned (we believe falsely) on charges of injuring an infant by the SBS. It could happen to anyone regardless of race, sex, educational, financial or social status. It has and is happening to more than a few. The following article is a report of one case with which the authors have intimate knowledge.

The shaken baby syndrome (SBS), as reviewed in the *Journal of the Royal Society of Medicine* and other journals,*(*l-4) commonly describes a combination of subdural hematoma, retinal hemorrhage, and diffuse axonal injury (DAI) as the triad of diagnostic criteria. The basic issue to be addressed in this review is whether or not in some instances, where a father has been accused of causing the death of his child from the shaken baby syndrome, the true cause of death was from a catastrophic vaccine reaction. The present reviewers believe that the demise of Baby Alan fits with such a vaccine reaction, and that the father was falsely accused and convicted of the murder of his son based on a mistaken diagnosis.

By definition, the word "syndrome" refers to a group of signs and symptoms that occur together and characterize a particular abnormality. The question in the present instance is whether or not the criteria of SBS may have more than one possible cause.

Vera Scheibner, Ph.D., Australian researcher, in an article reviewing the shaken baby syndrome,(5) stated her opinion that many of the cases attributed to this cause have

actually been vaccine-related injuries or deaths. After having reviewed the medical records of the present case, she came to the same conclusion. She offered the following comment in support of this opinion:

"Indeed, vaccines like pertussis are actually used to induce encephalitis (experimental allergic encephalomyelitis) in laboratory animals.(6) This is characterized by brain swelling and hemorrhaging of an extent similar to that caused by mechanical injuries.(7,8)

In a bulletin from the *National Vaccine Information Center*, similar instances of mistaken diagnoses were sited; that is, instances where vaccine injuries were mistakenly diagnosed as SBS, resulting in imprisonments.(9) One of the instances was reviewed in the article:

"Dr. Thomas Schweller, a San Diego pediatric neurologist, who testified (in a case in which a father was accused of brain injuring his child) that the brain damage from interior bleeding was likely triggered by the DPT shot, stressed this in a Gannett New Service Interview: is a tendency in some medical arenas to discount completely the history provided by the family if you find evidence of subdural hematoma - no matter what history is provided. Even a three-foot fall can cause fractures. It doesn't need to come from a shaking event. I'm always leery in medicine of saying something is always due to some factor, or that something is 100 percent.'"

In the present case it is important to point out that a *vaccine reaction was never mentioned by any witness as a possible factor in the baby Alan's death.*

Instances of shaken baby syndrome, tragically, do occur, but it is also tragic when fathers or other family members are falsely accused and imprisoned as the result of mistaken diagnosis, where the true cause of the brain injuries arose from vaccines.

The present case, which the authors have carefully reviewed, will be used as a model. Let the facts, as we understand them, speak for themselves.

**The Story of Baby Alan:** Baby Alan was born on September 16, 1997. Due to a deficiency of amniotic fluid on an ultrasound test, which suggested a possible premature rupture of the membranes, labor was induced at 35 weeks gestation. The admitting officer did note that premature rupture of membranes had taken place, as the indication for the induction of labor. However, it should be stressed that the mother had not noted leakage of her amniotic fluid, as she later recalled, only a moisture at the vaginal outlet, which raises the possibility that the mother did have chronic or

prolonged oligohydramnios (lack or deficiency of aminiotic fluid). (More will be said of this later.) Other conditions which placed the baby into a high risk setting included maternal gestational diabetes, anemia, group B Streptococcal vaginal infection, chronic maternal E coli urinary tract infection with proteinuria, as well as nicotine and caffeine during pregnancy by the mother.

In addition, the mother had suffered from colon problems for many years and had been advised not to become pregnant, as this problem might predispose her to toxemia of pregnancy. After becoming pregnant, she became sick and remained so during her pregnancy, often to the point of dehydration, losing from her original weight of 130 pounds down to 120 pounds at one point and finally coming back to her original weight of 132 pounds at time of delivery, a net weight gain of 2 pounds. She said she was too sick to take her prenatal vitamins. When one considers that the currently recommended weight gain for pregnancy is 25 to 30 pounds, this type of situation would place the fetus at high risk for a wide spectrum of nutritional deficiencies and retarded development.

It should be stressed that each one of these conditions alone would have placed the baby in a high-risk category, so that in their totality they placed a guarded prognosis on the baby at time of birth.

The birth weight was 5 Ibs, 8 ounces; APGAR scores were 8 and 9. However, following birth, respiratory distress of the baby was immediately evident (as shown on birth video) with grunting respirations with marked rib and sternal retractions. The mother observed a persistent grayish color following birth. At approximately 2 hours following birth an

Accu-Chek was 37; a follow-up blood glucose was 32. Arterial blood gasses on room air revealed severe hypoxia and acidosis with pH 7.38, C02 42, pO2 43, and bicarbonate 21. The BUN of 8.0 and creatinine of 0.4, were unusually low, probably from protein lack as a phase of malnutrition The infant was placed in an oxyhood with 50% 02; he was started on ampicillin and gentamycin.

The baby's 7-day hospital course was complicated by continued respiratory distress, spending 3 days in the intensive care unit. 3 daily chest X-rays during this time showed persistent pulmonary infiltrates. Persistantly low serum CO2's indicated continued acidosis throughout hospitalization. Also the baby had neonatal jaundice with a maximum bilirubin of 17.4 and a decrease to 13.2 before discharge. Liver enzymes were elevated with an ALT of 58 and LDH of 520.

According to the mother, symptoms of chest congestion and difficulty breathing never did clear following discharge from the hospital, with grunting and raspy breathing

patterns, and with occasional brief periods of apnea. Also, the baby remained grossly jaundiced for a month after returning home, much longer than would have been expected from benign neonatal jaundice.

Of special importance as related to the rib fractures, which will be discussed below, on no occasion during weekly visits to the pediatric clinic following discharge from the hospital, and one visit to a hospital emergency room, were there any reports of external injuries or bruises, nor of acute pain or discomfort, both of which almost certainly would have been noticed by an examining physicians and nurses had these injuries occurred after being taken home by the mother.

On November 11, 1997, at approximately 8 weeks age (but only 43 weeks true gestational age), the baby was simultaneously given 6 vaccines including DPT, Hib, OPV, and hepatitis B. Within 24 hours of the immunizations the baby developed intermittent diarrhea, irritability, and feverishness, a pattern which progressed into the terminal illness.

As related by the mother, about 10 or 11 days following the vaccines the baby developed a high-pitched cry, and its skin became warmer to the touch. Having been forewarned during the previous office visit that these things might ensue following the vaccines, she did not become overly alarmed. However, she also noticed an increasing lethargy and a falling off of the baby's feeding patterns, which had been a combination of breast and formula.

This pattern continued for 3 days until the morning of November 24, when the father was alone at home with the baby and his 4-year old sister. In rapid succession the father observed that the baby began wheezing, then spit up, then stopped breathing. In efforts to restart breathing the father first lightly slapped the baby's face, then began spanking the baby's bottom while holding him by the heels, all without success. After delays from unsuccessful attempts to revive the baby, and from running to a neighbor's house to borrow the neighbors car, the father then rushed the baby to the Princeton Hospital of Orlando, where the baby was successfully resuscitated. However, according to family estimates, the baby must have been apneic a minimum of 20 minutes, considering the delays and the distances, before resuscitation was accomplished. Incidentally, emergency room records recorded 5 minutes of apnea, presumably the time between arrival in the E.R. and resuscitation. Initial laboratory tests in the Princeton Hospital showed anemia with a hemoglobin of 7.8, Hct 25.3, RBC’s 2.61, elevated white blood count of 20,900 (with 61% lymphocytes, 26% neutrophils, 5% bands, and 8% mononuclears), platelets 571,000, markedly elevated liver enzymes, bilirubin 0.6, blood sugar of 337, mildly prolonged prothrombin time (a bleeding study), and elevated split fibrin products. A blood culture reported light growth of gram positive cocci, coagulase negative, probably a contaminant.

It should be pointed out that the predominance of lymphocytes (61%) in the white blood count is a hallmark of a pertussis reaction (10).

The patient was transferred to the Florida Hospital, Orlando, where he was placed on life support. Admitting temperature was 105 degrees. A brain Ct scan was interpreted as showing a small right subdural hematoma and one or two sites of intraparenchymal bleeding. Chest Xray showed bilateral pulmonary infiltrates (bilateral pneumonitis) and healing fractures of the 6th and 7th ribs on the left.. A spinal tap was not done due to the difficulties and hazards of performing a tap while the baby was on life-support.

Intravenous heparin was administered 3 hours post-admission and was continued after the brain CT scan revealed intracerebral hemorrhages.

Following a hospital admission of 75 hours, the baby was pronounced dead, being 10 weeks of age at time of death.

**Post-Mortem Findings (Performed by the medical examiner for Orange and Osceola Counties):** Findings included minor contusions of both temporal areas of the head and a small bruise of the right lower eyelid. The brain was grossly edematous, (which may have been the precipitating factor of the apnea preceding hospital admission). There were large, fresh subdural hemorrhages, right and left hemispheres, predominantly right; also hemorrhages at the base of the brain and over some areas of the spinal cord. The brain was grossly edematous. There was a small focus of bleeding in the right eye (bleeding absent in the left eye). In addition there were old, healing fractures in the 5th, 6th 7th nd 10th ribs, all posterior on the left. The lungs were mildly hemorrhagic and were congested with scattered inflammatory cells, indicating an interstitial pneumonitis. (The heart, liver, pancreas, small intestines, gall bladder, and spleen had been surgically harvested before death for organ donations).

Based on these findings, it was the medical examiner's conclusion that the baby had died from the shaken baby syndrome.

As previously mentioned, the brain CT scan done soon after hospital admission showed only a relatively small subdural hemorrhage on the right, none on the left. This being the case, it can be assumed that the left subdural hemorrhage commenced following hospital admission, indicating a non-traumatic etiology. It is possible or even probable that the intravenous heparin, started 3 hours following hospitalization, may have caused or contributed to the massive bleeding found at autopsy.

As another important point, the medical examiner testified during the trial that he found no evidence of meningitis on autopsy (an important point to keep in mind in view of subsequent testimony from another witness, who described heavy inflammatory cell infiltration in the meninges); but while denying the presence of meningitis, at the same time he admitted that he had not examined the spinal fluid, nor was there any description of the meninges in the autopsy report.

In regards to postmortem findings in the kidneys, the defense witness pointed out during his testimony that the presence of renal lobulations and the failure of renal tubules to detach from the renal capsules (from which they are genetically derived) was an indication of delayed development or "failure to thrive" of the baby.

**Jury Trial:** At the subsequent jury trial, taking place from February 22 to 24, 1999, the state attorney provided four major witnesses testifying for the state, including the medical examiner, who had performed the autopsy, and a neuropathologist. Against these the defense provided a single witness, a neuropathologist. More than this, two of the state

witnesses were called for repeat testimonies following that of the defense witness, making a total of six witness hearings for the state, one for the defense.

It is of concern to the writers of this review that, as far as can be determined from the court transcripts of the trial, none of the state witnesses had sought nor studied the medical records from the neonatal hospitalization of the baby; at least no mention was made of them. Thereforeone must wonder if they had any awareness of the multiple complications surrounding the neonatal period including severe hypoglycemia, severe hypoxia, critical hyperbilirubinemia, and ongoing respiratory distress during and following release from the hospital.

Any one of these neonatal complications (severe hypoglycemia, hypoxia, and critical hyperbilirubinemia) may have caused brain damage, but the 3 together almost certainly did cause such damage, damage which of necessity had a profound effect on the subsequent course of events and which must have been the true cause for some of the post-mortem findings, rather than the shaken baby syndrome.

The apparent unawareness of the state witnesses concerning these earlier complications places serious question about their understanding of the case and the sequence of events leading to the baby's death, which we believe led to faulty conclusions. At least one of the state witnesses was compelled to admit, under oath, that he had neither sought nor read the newborn hospital records. Another state

witness, the neuropathologist, denied that he saw any evidence of neonatal hypoxia in his review of postmortem findings. In our opinion, no doctor who had carefully read the baby's previous medical records would have made such a statement, with their overwhelming evidence that hypoxic damage could have taken place during or following birth.

Still another area of concern is that, in not a single instance, did a state witness mention the prolonged apnea of the baby preceding the terminal hospital admission, or that the apnea in and of itself could have resulted in the complications and pathologic findings later described by the neuropathologist, including the acute degenerative changes of the brain cells with reddish discoloration and swelling of the blood vessels. One wonders whether or not the connection between these findings and the apneic period ever occurred to the state witnesses, and if it did, why they did not mention it, as clearly it was a matter of major importance in the interpretation of the findings and the outcome of the case.

During the trial, all of the state witnesses agreed with the medical examiner that the father was guilty of child abuse, and the baby had died of the shaken baby syndrome. The defense witness disagreed with this conclusion. It was his opinion that the baby had died from "natural causes."

Although there were a number of issues raised during the trial, for the most part, guilt or innocence of the father revolved around five major issues: the rib fractures, the unitaleral retinal hemorrhage, the cerebral hemorrhages, diffuse axonal (brain cell) injury, and meningitis. These as well as other issues will be addressed in the following:

**The Issue of the Birth Weight:** It is well known that newborn infants of diabetic mothers tend to be larger and plumper (macrosomia) than nondiabetic mothers, a response to increased nutrient supply and hypersecretion of insulin by the fetal pancreas. However, a large retrospective review by Dr. Evelyne Rey of Montreal has also found that infants born from mothers with gestional diabetes have higher rates of newborns who are large-for-gestional-age, in addition to having complications of hypoglycemia and hyperbilirubinemia;(11) (both of these latter complications were present following birth in baby Alan). For this reason, the recorded birth weight for Baby Alan of 5 pounds and 8 ounces may have been falsely elevated, with an increased proportion of adipose tissue in relation to other tissues and organs.

**The Issue of Hyperbilirubinemia:** The term, *hyperbilirubinemia,* denotes an excess of bilirubin, a condition which is potentially neurotoxic. *Kernicterus* is a neurologic sysndrome resulting from deposition of unconjugated bilirubin in the brain. It can occur at much lower levels of serum bilirubin in sick or premature infants than in

healthy, full-term babies. While toxic levels for healthy infants is listed at or above 25 mgs/dL in current pediatric texts, levels as low or lower than l0 mgs/dL can cause brain damage in a sick or premature infant.(12) Other predisposing factors to kernicterus include acidosis, hypoxia, administration of free fatty acids (administered by nasogastric tube in the newborn intensive care unit), salicylates (aspirin) and antibiotics (both administered during newborn hospitalization), and pitocin (the mother's labor was induced with pitocin).

**The Issue of the Rib Fractures:** At autopsy four rib fractures were found, all on the posterior left. All witnesses agreed that these fractures were old, as indicated by callus formation.

The state witnesses tried to suggest that, as one callus was larger than another, this was an indication that the rib fractures had occurred at different times, thus indicating a pattern of child abuse.

There are several considerations that strongly mitigate against this interpretation. First, neither the mother, grandparents nor baby-sitter noted any bruising or indication of damage surrounding the ribs following discharge from neonatal hospitalization, nor were there

any indications of injury found during weekly outpatient visits to the pediatrician. Next, different sizes of callus might just as well have indicated a difference in severity of the injuries rather a difference in time of occurrence. In addition, strong evidence weighing against child abuse is found in the *Journal of Trauma*,(1990) in an article entitled, "rib fractures in children: a marker of severe trauma.(13) The article reviewed a study of 2,080 children seen at a pediatric trauma center in 1985-1988. Among 33 of these children, who were found to have multiple rib fractures, *these injuries were accompanied by severe internal thoracic injuries in 85% of the cases.* Evidence of such injuries were notably absent in the present case, before death and at autopsy.

It was the suggestion of the defense witness that the rib fractures took place during labor, prior to birth. This hypothesis would tend to be supported by the work of Marvin Miller, M.D., with the Children's Medical Center, Dayton, Ohio, who reviewed 26 cases of infants with multiple unexplained fractures that fit the criteria of a recently described condition, the temporary brittle bone disease (TBBD).(14,15) The results of this study showed a striking association between TBBD and decreased fetal movement during pregnancy, something observed by the mother of Baby Alan during her pregnancy.

As an interesting sidelight, the medical examiner testified during the court trial that a "bone had broken while handling" during the autopsy. This would tend to corroborate the assumption that the bones were extremely fragile and prone to spontaneous fractures.

There is also a possible and plausible role of advanced vitamin C deficiency in predisposing to rib the fractures, which will be discussed further on in a section dealing with scurvy.

**The Issue of Chronic Oligohydramnios:** Perhaps the strongest argument that the rib fractures took place either during the pregnancy and/or the mechanical stresses of labor is based on the probability that the mother had chronic oligohydramnios (deficiency of amniotic fluid) as a result of the multiple complications of her pregnancy. The reduced amniotic fluid, in turn, would have constricted fetal movements (the mother did note a lack of fetal movement during pregnancy), this in turn leading to "temporary brittle bone disease" discussed above. *Nelson's Textbook of Pediatrics* has this to say about oligohydramnios:

"Oligohydramnios is associated with congenital anomalies, intrauterine growth retardation, and severe renal anomalies. . . This becomes most evident after 20 wk gestation, when fetal urination is the major source of amniotic fluid. . . The most serious complication of chronic oligohydramnios is pulmonary hypoplasia. . .(16)

In other words, the presence of chronic oligohydramnios would have explained the complications that followed birth including the rib fractures, the retarded development of the kidneys found on autopsy slides, (to be described later) and the pulmonary hypoplasia. In regards to the pulmonary hypoplasia, a video of the birth scene vividly displayed marked retraction of the ribs and sternum on the baby's first cries, a finding virtually diagnostic of the reduced lung capacity, which is characteristic of hypoplastic lungs. (With reduced lung capacity, and the lungs unable to fill the chest cavity, the chest wall would necessarily be sucked in as the diaphragm contracts during inspirations). It should be remembered that the admitting physician for the mother's maternity hospitalization was under the impression that there had been premature rupture of the membranes, as an indication for inducing labor. However, birth records recorded that membranes ruptured 9 minutes before birth which would tend to rule out premature rupture.

It is true that an ultrasound on August 22, 1997, 4 and 1/2 weeks before delivery, reported normal level of amniotic fluid (15 cm). However, given the fact that, following 20 weeks gestation, fetal urination becomes the major source of amniotic fluid, it is highly probable that the mother did have chronic oligohydramnios throughout much of her pregnancy because of the finding on post-mortem slides that

the kidneys showed definite markers of "failure to thrive," or retarded development (see below), and because of the constant sickness of the mother during her pregnancy with a total lack of weight gain, malnutrition, dehydration, and gestational diabetes. With the combination of these conditions, it is doubtful that the fetal kidneys would have been able to maintain a normal volume of amniotic fluid throughout the latter portion of pregnancy.

**Issue of the Cerebral Hemorrhages:** The defense witness held steadfastly to the view that the cerebral hemorrhages were not the result of trauma but were due to a combination of insults to the blood vessels, including the prolonged period of apnea preceding the terminal hospital admission, the presence of brain edema noted at autopsy, and the presence of advanced and extensive meningitis (described below), both of which would result in swelling of the blood vessels with increased friability and fragility, making them prone to spontaneous bleeding.

The hemorrhages described by the defense witness were all fresh, in his estimation taking place hours or at most within 24 hours of death, as indicated by a lack of inflammatory cell infiltration in and around the hemorrhages and by the freshness in the appearance of the red blood cells. This would necessarily place the timing of these hemorrhages to have taken place following hospitalization. A possible or probable contributory factor to fresh hemorrhages following admission was the administration of the blood thinner, heparin, as previously reviewed.

Both state witnesses, in contrast (the medical examiner and the neuro-pathologist consultant), attributed the bleeding to trauma. The former estimated that the bleeding was 2 to 3 days old, the latter 2 to 5 days. Although both attributed the initial bleeding to trauma before hospital admission, these figures imply an admission of the possibility that the major hemorrhages could have started following the terminal hospital admission.

As previously mentioned, neither state witness mentioned the possible role of the apneic episode preceding hospitalization and the role it could have played in the pathologic findings, as did the defense witness.

**The Issue of Meningitis:** During his testimony the defense witness described in some detail his finding of extensive meningeal membrane infiltrations with inflammatory cells, which he felt represented meningitis, possibly viral in origin. Due to the degenerative appearances of the nerve cells, he said that it was necessarily an old process, perhaps weeks in duration, certainly present before the final hospital admission.

The medical examiner, during his court testimony as a state witness, denied any findings indicating meningitis. However, as previously pointed out, he admitted that he had not examined the spinal fluid, nor was there any description of the meninges in his pathological report.

The neuro-pathologist consultant, the second state witness, when asked about the presence of meningitis, replied that there are three possible types: (1) bacterial, or purulent, which was clearly not present in the baby, (2) viral, in the form of aseptic meningitis, and (3) homogenic, the result of meningeal irritation from the hemorrhages. This witness stated that, in his opinion, meningeal inflammation in the present case was the result of the latter. However, from his own statement, that "we don't see homogenic meningitis for 3 or 4 days following hemorrhage," he tended to contradict his claim that the meningeal inflammation was homogenic, or from blood irritation. As we understand his statements, he agreed that most of the hemorrhages found at autopsy were fresh, too fresh to result in inflammatory reactions. Also, the subdural and ependymal bleeds noted on the brain Ct scan when first admitted to the hospital were small and limited, presumably too small to cause extensive reactions. There were also suggestions of small intraparenchymal bleeds on the Ct scan.

Once again, neither defense nor state witnesses mentioned the possibility that these same findings could represent a vaccine-induced encephalomyelitis.

**The Issue of Diffuse Axonal Injury (DAI):** One of the pivotal issues in the case was the timing of the brain cell damage described by both the defense witness and the state witnesses, the former contending that the damage was old, almost certainly taking place during the neonatal period, the latter insisting that it was due to shaken baby syndrome.

For background information, early descriptions of diffuse traumatic white matter damage

(DAI) suggested that the responsible mechanism for injury was a shearing of nerve fibers at time of injury followed by swelling of the nerve axons and later by varying degrees of nerve cell death, with most of the injuries taking place in the mid and hind-brain areas and upper cervical spinal cord.(17-19) However, recent reviews of the subject have stressed that pathologic findings from traumatic axonal injury may be indistinguishable from those brought about by hypoxic events.(20,21) Quoting from a conclusion from the article by Kaur and Rutty: "Axonal bulbs… may occur in the presence of hypoxia and in the absence of head injury. The role of hypoxia, raised intracranial pressure, oedema, shift effects, and ventilatory support in the formation of axonal bulbs is discussed. *The presence of axonal bulbs cannot necessarily be attributed to shearing forces alone."*(20)(Emphasis ours) The article by J.F. Geddes

says much the same thing, as indicated by the following quotation: "Because of potential confusion with hypoxic axonal injury, *we suggest that DAI is never used as a neuropathological diagnosis in medicolegal cases, without the aetiology of the damage being made clear. On occasion it may be impossible to be certain of the cause of axonal damge…"*(21)(Emphasis ours)

As previously reviewed, under direct questioning during the trial, one of the state witnesses testified that he found no evidence of neonatal hypoxia on the pathology slides. In the view of the writers of this article, it is extremely unlikely that he would have made such an unqualified statement had he been aware of events surrounding the neonatal period, during which there was a combination of hypoglycemia, hypoxia, and critical hyperbilirubinemia, the combination of which almost certainly would have caused brain damage.

The defense witness, in contrast, described extensive *old* nerve damage in areas of the brain and in a slide of the spinal cord. In the slide of the spinal cord, in addition to the nerve damage, he identified the presence of extensive revascularization (formation of new blood vessels). Since the revascularization would necessarily have been a slow process, this necessitated the conclusion that the process was old, probably taking place during labor and/or the neonatal period.

**The Issue of Disseminated Intravascular Coagulation (DIC):** It should be pointed out that the supposed presence of acute DIC, allegedly brought about by the shaking of the infant by the father immediately preceding the terminal hospital admission, was one of the cornerstones of the case of the prosecuting attorney brought against the father, as testified by one of the state witnesses, the neuropathologist. According to the laboratory parameters for acute DIC outlined in a review by Cunningham in July of 1999,(22) one of the prime requirements for the diagnosis of *acute* DIC is that of a reduced platelet count. There is no equivocation on this in the Cunnihgham article. Far from being reduced, baby Alan's platelet count was markedly elevated at 571,000 on the terminal hospitalization, which would be more compatible with the timing of a vaccine-induced injury, taking place a number of days before hospital admission:(23)

**Defense Witness's Summation:** In the defense witness's conclusion that the baby had died from natural causes, he based this conclusion on several findings:

The baby was admitted to the hospital during its terminal illness with two advanced and long-standing conditions: bilateral pneumonia and meningitis. According to his words, either one might have been fatal, but both together would in certainty have caused death.

The brain hemorrhages were in all probability spontaneous, due to the combination of meningitis and a prolonged period of apnea preceding the terminal hospitalization, as described above.

The baby had several features of "failure to thrive," including immaturity of the kidneys (renal lobulations, persistent attachment of renal tubules to the renal capsule), and a failure in real weight gain. He hypothesized that this may have been due to prolonged pneumonia and also a neonatal hypoxic event, as indicated by extensive nerve degeneration in the spinal column with extensive revascularization, the latter necessarily reflecting an old process and placing the timing around birth. (We now know that almost certainly such an event did take place around the neonatal period from a combination of severe hypoxia, severe hypoglycemia, and hyperbilirubinemia). Also, the defense witness's contention that there was brain/spinal injury near or at birth tends to be supported by the presence of nucleated red blood cells reported on blood counts for 3 days following birth(24) and elevated liver enzymes.(25)

**Trial Conclusion:** In spite of a brilliant presentation by the defense witness, in our opinion correct in every particular from birth to death of the baby, the jury found the defendant guilty of murder. As for the reasons for this verdict, the descriptions by the defense witness were highly technical, and the jury may have understood little of it. Also, the defense witness had only one appearance before the jury, while the state witnesses had a total of six. In this case it would appear that numbers did count.

Since the defendant had refused to plea-bargain, maintaining his innocence, the laws mandated a life sentence, and the court had no choice but to impose this sentence. In our view, the refusal of the father to plea-bargain for a lesser sentence was a courageous act, one which would have been made only by a person conscious of his own innocence.

**Review and Discussion:** Very clearly, the infant remained seriously ill following discharge from the hospital following his newborn period. Three serial chest X-rays in the hospital showed persistent pulmonary infiltrates, which were again found at postmortem examination, indicating a persistent, bilateral pneumonia, which had been present since birth. In addition, there were indications of brain damage from neonatal hypoxia and of failure to thrive, as pointed out and described by the defense witness. The baby was

born prematurely. Not to be dismissed were the mother's observations that the baby's chest congestion never did clear after being taken home from the hospital.

Under these and other severely compromised conditions the baby was administered a total of 6 vaccines, including the DPT, Hib, OPV, and hepatitis B. at approximately 8

weeks of life. A serious, possibly catastrophic reaction to the vaccines would have been predictable under these circumstances. Almost certainly a medical consensus would agree that vaccinations would have been contraindicated and should not have been given. In this regards, *The Physicians ' Desk Reference* provides warnings or precautions for all of these vaccines to inquire into the health of the recipient before their administration. For the DPT there is a warning that immunizations should be deferred during an acute infection, the clear implication being that there are heightened risks of reactions in the presence of infection or serious illness. Prematurity has also been listed as a contraindication to vaccines in early infancy.(26)

**Rationale that Baby Alan's Death was Vaccine-Related:** There are two possible mechanisms, either separately or in combination, by which the vaccines could have initiated a train of events culminating in death. The first would have been that of an "immune paralysis" from the vaccines, which could have resulted in a fulminating spread of the lung infections (pneumonia) to other parts of the body, including the brain. As the inflammatory cells were described to the court as lymphocytes, this would of necessity have been a viral infection, not bacterial.

The second mechanism would have been a vaccine-induced encephalomyelitis, of which the pertussis, hepatitis B. and Hemophilus influenza bacillus vaccines would have been prime suspects, either individually or in combination. This requires an acceptance of the validity of the 10 or 11 day latent period in the present case, the time period between the vaccines and the onset of signs of encephalitis and/or meningitis. It is freely admitted that this flies in the face of the 3 to 7 day limitations (depending on the vaccine), imposed by current guidelines of the Congressional Childhood Vaccine Injury Act of 1986, whereby symptoms of encephalitis must occur within these time periods for the vaccines to be recognized as a cause of the encephalitis. However, based on recent medical literature, some of which will be reviewed here, there are grounds for believing that these time limitations are outdated and unrealistic. It should also be noted that minor symptoms did appear the day following immunizations as noted by the parents and grandparents, including feverishness, irritability, and diarrhea as well as feeding problems.

However, there is one piece of information which outweighs all others and which carries the vaccine issue to a level of virtual certainty; that is, that the vaccines did cause the death of

baby Alan. Not discovered until December, 2000 because of careless and incorrect nurse’s notes in recording the diphtheria-pertussis-tetanus vaccine, as "DTP" rather than "DTaP," (acellular), the latter being the vaccine that was actually given to the baby, as confirmed by the doctor’s order sheet and also the mother’s vaccine records. Further investigation has revealed that this vaccine (Connaught Labs, DTaP 7H81507)

belongs to the "hottest" lot on record, according to VAERS files, ranking highest in infant deaths among more the 800 vaccine lots.

**"Immune Paralysis" from Vaccines, a Possible Role in Spread of Infection:** There is a small but firm body of medical literature that vaccines can bring about a form of immune paralysis, opening the way for invasion by micro-organisms which the body may be harboring, micro-organisms which otherwise might remain relatively harmless. One of the most intriguing of these was reported from Germany in 1986 in a little noted Letterto-the-Editor to the *New England Journal of Medicine.*(27) In the study, a significant though temporary drop of T-helper lymphocytes was reported in 11 healthy adults following routine tetanus vaccinations. Special concern rests in the fact that, among 4 of the subjects, the T-helper lymphocytes dropped to levels seen in active AIDS patients.

Parenthetically, if such results ensued from a single vaccine in healthy adults, it is frightening and sobering to think of the possible consequences of the multiple vaccines given to this vulnerable infant.

Although this study has never been repeated (as far as we are aware) a new text by Teddy H. Spence(28) provides 20 references of studies or case reports showing immune suppression following various vaccines, four of which are cited here.(29-32)

Historically, one of the earliest reports of spread of disease following vaccines is found in an older book, *The Hazards of Immunization,* by Sir Graham Wilson.(33) Although not necessarily opposed to vaccines, the author did give an extensive review of the potential side effects from the vaccines, including a chapter entitled, "Provocation Disease," in which he described certain complications, including paralysis from poliomyelitis in an arm into which vaccines had been given. Significantly, this was noted most frequently following the DPT vaccine.(34) In more recent times, a similar phenomenon was observed in Oman during a polio epidemic, in which it was found that a significantly higher proportion of the polio cases had received the DPT vaccine within 30 days before paralysis than did controls.(35)

It is known that the baby had a smoldering bilateral pneumonia at time of the vaccines, as well as failure to thrive. The defense witness, we believe correctly, testified that the baby had had neurologic damage from neonatal hypoxia. The immunological suppression from multiple vaccines into a highly vulnerable infant might well have resulted in a fulminating spread of the lung infection to other parts of the body, including the brain.

**Vaccines as a Potential Source for Cerebral Hemorrhage, Autoimmunity, and Vasculopathies:**

In a collection of abstracts from Med-line research from 1990 to October, 1997, on adverse reactions from the recombinant hepatitis B vaccine, Dr. Andrea Valeri of Italy catalogued a total of 45 different types of reactions in the world literature.(36) Among these were necrotizing vasculitis,(37) vaccine induced autoimmunity,(38) and segmentary occlusion of the central retinal vein.(39) In a report of 18 deaths of neonates following the hepatitis B vaccine by the Vaccine Adverse Event Reporting System, 1991-1998, hemorrhagic phenomena were common including 2 with cerebral hemorrhages, 4 with pulmonary bleeding, 1 with bloody diarrhea, and several with blood in upper airway passages.(40) A report in *PostGraduate Medicine* in 1973 on acute hemorrhagic encephalitis cites vaccines as one of the possible causes.(41)

As early as 1975 Urbaschek described the role of bacterial endotoxin (in this instance the pertussis endotoxin) in bleeding and coagulation disorders.(42) More recently McCuskey et al described the initial responses to endotoxemia as microvascular inflammation with activation of endothelium from its normal anticoagulation state to a procoagulation state.(43) However, in this instance blood coagulation tests may have been skewed by the administration of heparin soon after hospital admission.

In a study devised to provide an animal model for the systemic and neurological complications observed following the pertussis vaccine in children, Steinman and coworkers discovered a lethal shock-like syndrome in mice after immunization with B pertussis vaccine and sensitization to bovine serum albumin. Post-mortem examination of the brains revealed diffuse vascular congestion and **hemorrhages in both cortex and white matter.** (emphasis ours)(44)

In the case of Baby Alan, the encephalomyelopathy could have predisposed to hemorrhagic consequences due to (1) increased friability of the blood vessels, (2) brain edema with resultant shearing effects, and (3) slight but possibly significant prolongation of prothrombin time. Of passing interest, as related to retinal hemorrhages, was a study of 20 children resuscitated following events other than trauma such as near drowning, asthma, sudden infant death syndrome, and other causes in which it was found that 2 children (10%) were found to have retinal hemorrhages.(45)Thus, there are exceptions to the current belief that SBS is the sole cause of retinal hemorrhages.

**A New Syndrome Emerging from Tragedy?:** As yet based largely on observation and a limited but suggestive body of medical literature, in many cases thought to represent SBS it appears that we may be witnessing the adverse effects from interactions of highly potent vaccines given in combination, which potentially include: Hepatitis B (hemorrhagic vasculopathies, autoimmune reactions, neuropathies), Hemophilus influenza (Hib) (hypersensitization), tetanus (hypersensitization), and pertussis (hypersensitization, brain edema, and

hypercoagulability with vascular inflammation from endotoxin). A study by Terpstra found the Hib vaccine to exceed even the pertussis vaccine in the latter's sensitizing potencies.(46) Usually within a period of 12 days these interactions bring about a combination of brain edema, hypercoagulability of the blood, and inflammation of blood vessels, these in turn resulting in a shearing effect on subdural blood vessels and subdural hematomas, thus mimicking what is now thought to represent the SBS.

If this does in time prove to be a newly recognized syndrome, then it should have a name.

In our opinion, none could be more fitting than the "Yurko Syndrome," in honor of baby Alan Joe Yurko.

**Vaccines, Scurvy, and Hemorrhagic Diatheses:** In the 1970's a major contribution was made to medicine by the Australian, Archivides Kalokerinos, M.D. in his work among the Australian aborigines. After working a number of years among these people, Dr. Kalokerinos became appalled by the very high infant mortality rate, in some areas approaching 50%. Having observed cases of scurvy among the children, who were living on very poor diets of processed foods; and noting that infants frequently died following immunizations, especially if they had colds, he intuitively made a connection between vitamin C deficiency and deaths following vaccines. After improving nutrition and adding regular vitamin C supplementation, infant mortality was virtually abolished.(47,48) As a result of this work he was awarded the Australian Medal of Merit in 1978.

One of the primary roles of vitamin C being the production and maintenance of connective tissues in the body, Dr. Kalokerinos hypothesized that, in infants nutritionally deficient of vitamin C, with viral infections further depleting their limited reserves, the administration of the pertussis vaccine would often throw the children into fulminating scurvy with its hemorrhagic complications, with vitamin C being consumed at enormous rates in neutralizing the pertussis toxin.

In the present case, we have earlier reviewed the stormy course of the mother's pregnancy, with a total lack of weight gain from beginning to end of the pregnancy. This consideration, together with the fact that she was unable to take her vitamins, almost certainly would have resulted in gross nutrient deficiencies in the baby, especially vitamin C, resulting in heightened vulnerability to the vaccines. It could also have played a role in the rib fractures. Vitamin C deficiency may have contributed to inadequate connective tissue formation in the bones before birth, making them susceptible to "green stick" fractures during the stresses of the birth process. As well, vitamin C deficiencies are linked to anemic conditions.

In his writings, Dr. Kalokerinos referred to a case with which he was involved,(49) a case with uncanny similarity to that of Baby Alan including rib fractures, retinal and subdural hemorrhages. In referring to the case, Dr. Kalokerinos quoted from a text dealing with scurvy,(50) which described fractures at the costochondral junction, including those of the ribs to the spine. In his words: "Scurvy disrupts these areas (constochondral junctions), the bone breaks down and the ribs may 'override,' forming in typical cases 'beads.' Then healing commences with new bone formation looking just like true healing fractures. Furthermore, not all the ribs may be involved in this process and the changes will not all occur at the same time - giving the impression of multiple fractures of different ages.

Having heard about the case of Baby Alan and doing a review of the records, Dr. Kalokerinos offered to testify in the father's behalf, believing with a virtual certainty that the baby's death was vaccine related.

**The Controversy of the Latent Period following Immunizations:** As previously reviewed, there was a latent period of 10 or 11 days in Baby Alan between administration of the vaccines and the onset of signs of encephalitis and/or meningitis. Not to be discounted, though, were the minor reactions noted within 24 hours of vaccines, as previously mentioned.

If we think in terms of a vaccine-induced encephalomyelitis, most of the earlier literature deals with the pertussis vaccine. Flexner (1930) noted a strong tendency for the nervous system manifestations to declare themselves between the 10th to 13th days.(51) In a review of 108 cases recorded before 1929 by Gorter (1933), the onset of encephalitis as "strikingly constant," usually observed between the 10th and 12th days following vaccination, commonly with a febrile period on the 7th and 8th days, followed by recovery until onset of encephalitis.(52) In 1929 an editorial in the *Journal of the American Medical Association* reported on an increase in severe neurological complications following infections and inoculations, occurring on about the 11th day after vaccination.(53) Over 50 years later Munoz (1984), in a mice study of experimental encephalomyelitis elicited by injection of pertussigen, found the same latent period of 11 to 13 days.(54)

In contrast, literature since the 1970's has reported an entirely different pattern, with the onset of encephalopathy largely falling within a 3 day period following vaccines.(55-57) We can only speculate as to this changing pattern. Perhaps it could be attributed to the fact that, in those early years, children were given only the DPT vaccine or at most DPT with the oral polio vaccine, whereas in more recent years they have been receiving the hepatitis B and Hib vaccines in addition. As previously reviewed, the hepatitis B has been implicated in hemorrhagic diatheses, autoimmune

disorders and other complications; the Hib has been shown to have unusually high hypersensitizing qualities.

In the text, *Vaccinations and Behavior Disorders,* by Greg Wilson (publication pending), the author made the following comment in regards to the latent period:(58)

"Today the latent period is rarely mentioned in connection with neurological complications of immunization. . . Contemporary studies on the pertussis vaccine select an arbitrary time limit in which reactions have to occur to be considered as vaccine related. This time limit is usually from 3 to 7 days.

Perhaps the only study which explores the dynamics of post DPT reactions is an independent Australian study by Karlsson and Scheibner which, with a monitor which followed breathing volumes, found particular times of stress-induced breathing following DPT injections:"

'Of special importance (for stress) are days 2, 5, 6, and 8, 11, 13-16 and 18-21. (Scheibner, 1991).(59)

Dr. Scheibner's findings do have some support in two studies which showed a fairly high incidence of cardiorespiratory complications in premature infants following vaccinations.(60,61) Unfortunately, these studies were of limited duration (48 hours in one instance).

Another study throwing light on the latent period is one coming from Japan, from which it was found that increased histamine sensitivity in mice, brought about by the pertussis vaccine, showed two peaks, one on the 4th day following vaccination, and a second on the 12th day.(62)

In describing the mechanism of these cardiac and respiratory failures, Reisinger stated that the platelet injury by endotoxin may result in a dramatic rise in serotonin, which can initiate coronary chemoreflex causing bradycardia, hypotension and cardiac collapse.(63) Reisinger also commented that the hemorrhagic complications from the "black plague" of the Middle Ages were simply due to an unusually virulent form of endotoxemia from Pasteurella pestis, a property common to all disease-causing bacteria.(64)

In order to provide an overview of the latent period issue, there are two basic classes of immune systems, the immoral or antibody producing system, which tends to produce immediate-type reactions, and cellular immunity, in which reactions are delayed. Either class is capable of producing autoimmunity.(65) Obviously, the usual 3 or 7 day limitation, which now stands as a medical-legal standard, excludes a

recognition of the delayed-type autoimmune reactions and, by inference, even denies their existence. In an article by Cohen and Shoenfeld,(66) one dealing with questions of vaccine-induced autoimmunity, the authors pointed out that it is a subject about which relatively little is known, due to the comparatively little attention it has received in clinical and laboratory studies. In point of fact a more recent review on this subject cites a temporal relationship of 2 to 3 months between vaccines and autoimmune reactions.(67)

For this reason it is reasonable to assume that this is an area where large numbers of adverse vaccine reactions may be taking place, unrecognized and unreported because of this lack of study.

As a final comment about the latent period, in a letter to the *British Medical Journal*, Rosemary Fox, secretary of Parents of Vaccine Damaged Children, made the following comments:(68)

"Two years ago we started to collect details from parents of serious reactions suffered by their children to immunizations of all kinds. In 65% of the cases referred to us, reactions followed the triple vaccine (diphtheria-pertussis-tetanus). The children in this group total 182 to date; all are severely brain damaged, some are also paralyzed, and 5 have died. Approximately 60% of reactions. . . occurred within 24 hours of vaccination, 80% within 3 days, and all within 12 days."

It is of importance to point out that a significant number (20%) of reactions in this series did occur beyond the 3-day limit, which now serves as the medical-legal standard for identification of pertussis-vaccine reactions.

**Lymphocytosis and Brain Edema following Immunization:** Bringing back to mind the imflammatory cell (lymphocytic) infiltrations in the retinal and meningeal membranes in the present case, as described by the defense witness, it is of interest to review the literature on this subject.

Greg Wilson pointed out that that around the turn of the 20th century it was first noted that a marked leukocytosis and lymphocytosis occurred in the blood of children with pertussis, which has been a marker for the disease ever since. Cherry pointed out that the biologically active component in pertussis is known as the "lymphocytosis promoting factor."(69) Perhaps the most telling report concerning the present case is the previously reported case by Munoz,(70) in which an experimental encephalomyelitis was elicited in mice by the injection of pertussigen, a derivative of Bordetella pertussis, along with mouse spinal cord extract, from which there were histological findings of perivascular infiltrates, consisting largely of lymphocytes in the brain and spinal cord, findings reminiscent of the present case of Baby Alan.

Although Munoz mentioned nothing about the presence or absence of brain edema, the study of Iwasa stressed the finding of brain edema as a feature of pertussis-induced encephalopathy.(71) It is of interest to point out that there are human reports which support this finding: of infants which developed increased intracranial pressure with bulging fontanelles following DPT immunizations.(72-74)

With this information as a background, there is a basis for assuming the likelihood that the meningitis described from the pathological slides, with heavy infiltration of lymphcytes as well as brain edema, represents a vaccine-induced process.

**Allergic Sensitization Brought about by Vaccines:** The increasing incidence of allergic disorders in western nations is now universally recognized, with every third child in industrialized societies having an allergic disorder.(75) Since this trend coincides with vaccine programs, reports are now appearing which address the question of a possible relation between vaccines and increasing allergies. Among these are four controlled studies, from widely separated geographic areas, showing a marked increase in allergic disorders among fully immunized children as compared to those with limited or no vaccines.(76-79) Further indications of the propensities of vaccines, especially pertussis, to induce hypersensitivity reactions and/or encephalitis are to be found in laboratory studies, the natures of which are indicated by their titles:

"Pertussis adjuvant prolongs intestinal hypersensitivity.(80)

"Anaphylaxis or so-called encephalopathy in mice sensitized to an antigen with the aid of

pertussigen (pertussis toxin).(81)

"Immunoglobulin E and G responses to pertussis toxin after booster immunization in

relation to atopy, local reactions and aluminum content in the vaccines.(82)

"Comparison of vaccination of mice and rats with Haemophilus influenzas and

Bordetella pertussis as models of atopy.(83)

"Sensitization to thimerosal in atopic children.(84)

Regarding the Hemophilus influenza vaccine, possibly a result of its unusually high sensitization potential (85), it has been found that most children and adults experience a temporary decrease in the antibody to the capsule of the Hemophilus influenza bacillus following Hib vaccination. The authors cautioned that this decrease might

transiently increase the risk of invasive disease if it happened during an asymptomatic colonization with H influenza type b.

Finally, in a 1991 report by the National Institute of Medicine, the committee did find evidence of a causal relation between the DPT vaccine and anaphylaxis, a potentially lifethreatening allergic reaction.(86)

**Intriguing Studies in the Older Medical Literature:** Among other reasons, we must be thankful to Greg Wilson and his previously mentioned book for bringing to light some of the older studies on vaccine reactions, studies now largely forgotten.

As previously reviewed, studies of Flexner and Gorter reviewed the more prolonged latent periods between pertussis vaccine and onset of encephalitis observed in earlier decades than those reported in more recent times. Concerned reports by Byers and Moll(87) in 1948 and Toomey (88) in 1949 showed no reluctance to report on adverse reactions they observed from the pertussis vaccine, and to advise searches for greater safety in its use.

Of all the earlier reports, perhaps none is more intriguing than that of Low (Chicago, 1955), who reported a study in which he performed electroencephalograms on 83 children before and after pertussis vaccinations.(89) In 2 of these children the encephalograms turned abnormal following the vaccines without signs or symptoms of abnormal reactions. From these he concluded, "This study shows that mild but possibly significant cerebral reactions occur in addition to the reported very severe neurological changes."

The implications of this study are enormous. At a time when myriads of our children are suffering from minimal brain dysfunction or related disorders, it is possible that unrecognized vaccine reactions may be occurring on a large scale and may be contributing to this pool of unfortunate children. As Greg Wilson commented:

"studies such as Low's, which closely examine individual children, are extremely rare in the study of vaccine reactions and virtually non-existent in today's literature."

It is as if there has been a silent ban on studies which might reveal adverse side effects from the vaccines, and in the revealing raise questions as to whether or not, among some of the present vaccines, harmful effects may outweigh the benefits.

It is not quite true that there have no other similar studies since that of Low. There is a report from Japan in which 116 immunizations were given to 61 children with a history of febrile seizures or epilepsy, who had not had a seizure for one year. It was found that "epileptic spikes (among the children) reappeared after 10 and increased

among 10 out of 73 vaccine (administrations) given for DTP or DT or BCG vaccines."(90)

**Conclusion:** From all of the studies quoted above, especially the German study showing significant drops in T-helper lymphocytes in healthy adults following tetanus booster injections, and the study of Low just quoted (neither of which have had follow-up studies in the United States, as they should have had), a large number of adverse reactions may be taking place unsuspected and unrecognized. The adverse events from vaccines that have been reported may represent the tip of the iceberg, as compared with a much larger number that are actually taking place. All of this, we believe, has a direct bearing on the case of Baby Alan.

We have previously observed that the train of events in the present case, culminating in death, could be explained by the presence of pneumonia together with a viral meningitis and/or a vaccine-induce encephalitis. Shaken baby syndrome has never caused pneumonia and meningitis. Baby Alan died of a vaccine reaction.

Harold E. Buttram, M.D. & F. Edward Yazbak, M.D.

**Footnote:** For those who may wish to contact the wife of the prisoner or the prisoner himself, they may be reached as follows:

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[VaccineWebsite] [F. Edward Yazbak, MD] [Shaken Baby Syndrome]