Shaken Babies

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Introduction

Shaken Baby Syndrome was originally defined to include
1. Fractures
2. Bruising
3. Haemorrhages
   a) Intracranial
   b) Retinal
   c) Elsewhere
4. Other injuries not explained by a **clear and witnessed** (by so-called 'reliable witnesses') history of accidental injury. That is; what is regarded as 'nonaccidental injury' (NAI).

It was later defined by intracranial and/or retinal haemorrhages alone.
Then it was claimed that **gentle shaking alone** could initiate the problem.

Later still, the definition was extended to include some cases of the '*Sudden Infant Death Syndrome'*. Reasons for this included the idea that shaking a baby could upset

the brain stem and result in cessation of breathing. Support for this was generated by the fact that, sometimes in the brain stem ‘diffuse axonal injury’ is found, and cervical spinal ‘injuries’, such as haemorrhages, are sometimes found during autopsies. Note that diffuse axonal injury does not necessarily mean that an inflicted injury is always the cause. The term is misleading because cerebral anoxia can result in similar pathology – and this, obviously, is not always precipitated by inflicted injuries.

When so-called ‘diffuse axonal injury’ was added to the list of ‘baddies,’ paediatricians and forensic scientists became totally overcome by what they saw as the ultimate crime - someone loosing control, grasping an infant firmly around the chest, and shaking it violently for several seconds. Animated videos were produced to demonstrate how the brain moved inside the skull and ‘tore’ the axons apart. Animal experiments were performed and the results appeared to support the shaking hypothesis. The matter was therefore declared closed. Those who were accused appeared to have no defence. In America several individuals are on death row.

But, like so many other aspects of medicine, it was not simple. There are reasons to believe that shaking is not always the cause of the pathologies found. That is: there are explanations that do not involve inflicted trauma.

THE HUNT FOR THE REAL CRIMINAL
A true ‘whodunit’ detective story

The search began some years ago when I was asked to investigate one particular shaken baby case. There was a mountain of paperwork. To make matters difficult, I was not involved in the case until a Thursday and the long trial was due to end a few days later.

At the time I was also investigating another case. Deep within myself I sensed that I was missing something, but I could not determine what it was. This obsessed me, it bothered me, and I could not rest. Then suddenly, it appeared like a gift from heaven. In both cases there were ‘fractures’ of the costochondral junctions—caused, according to the prosecutors, when the infants were roughly gripped around the chest. These, I realised, were not inflicted injuries. They were similar to the changes that occur in the costochondral junctions in scurvy!

All this happened on the Friday night. Excited, almost beyond measure, I faxed the information to the lawyers. On the Monday I contacted them by phone. But that is where I came down to earth. A ‘confession’ had been extracted—as part of a ‘plea bargain’. The case was closed.

However, I was not closed. With the help of Dr. Ian Dettman, an extremely extensive literature search, and my own clinical experience, I was able to piece together a picture that was remarkable in its clarity and significance. Words and terms that previously had no meaning to me now became a part of me. I had, from the medical libraries of the world unearthed a treasure of medical knowledge and understanding. And to think that it had been there, documented by many medical researchers, for many years. It was also exploding in size every day.

I was left to wonder why so many of my colleagues could not see the glitter of the gems that so obviously confronted me.

AN ALTERNATIVE EXPLANATION FOR THE PATHOLOGIES FOUND IN 'SHAKEN BABIES' THAT DOES NOT INVOLVE INFLICTED TRAUMA

Because of the complexities of the issues, it is best to initially consider, separately, the various pathologies found:
1. Bruises
2. Haemorrhages, including intracranial, and retinal haemorrhages
3. Fractures.

There are some important common causes and features in each of these pathologies. However, because of differences in physiology and biochemistry, responses and, therefore, pathologies, will vary in many ways according to which organ is involved. For example, a haemorrhage in the brain, can progress towards complications that cannot occur in a skin haemorrhage.

The fundamental cause of all the pathologies

There is an increased utilization of Vitamin C precipitated (mostly) by endotoxin or other bacterial toxins. This, in turn, results in two pathologies:
1. Due to Vitamin C deficiency
2. Due to endotoxaemia.

Usually there is a combination of these two factors, with special complications that can occur in the brain because of its unique physiology. Taking this one step further, one can then move on to understand how:
1. Vitamin C deficiency can, in itself, cause haemorrhages.
2. Vitamin C deficiency can, in itself, cause spontaneous fractures.
3. Endotoxin can in itself, cause coagulation/bleeding disorders.
4. Endotoxin uses vast quantities of Vitamin C while being 'detoxified' (involving free radical reactions).

Endotoxin can specifically target the brain by:
1. specifically, and selectively, damaging the endothelial linings of cerebral blood vessels
2. breaking the blood-brain barrier
3. seeping into the cerebral tissue
4. causing anoxia by a direct effect on the respiratory centre
5. initiating a rapid series of biochemical disturbances, including free radical reactions, leading to an extremely rapid onset of cerebral oedema—with a host of possible complications
6. causing coagulation/bleeding disturbances.

Lacroix, *Brain Pathol* 1998 Oct:8(4):625-40 states:
" …a direct role of endotoxin on specific cell populations of the central nervous system, which is likely to be responsible for the transcription of proinflammatory cytokines, first within accessible structures from the blood vessels and thereafter through scattered cells."

Mayer, *Medicina* (B Aires) 1998;58(4):377-85, states:
" Lipopolysaccharide affects the permeability of the blood-brain barrier…"

The cerebral capillaries are particularly sensitive to endotoxin damage. Because of this the cerebral circulation can quickly cease—either in a localised area, or totally. Because the respiratory centre is damaged, respiration may cease—either forever, or for a period. This has the same effect as anoxia due to cardiac or respiratory arrest. In other words, there is no need to have cardiac or respiratory arrest for the initiation of cerebral oedema, although cardiac and/or respiratory arrest may sometimes occur. Failure to understand this is the reason why, in some very high profile cases, the prosecution has attempted to add an element involving suffocation, even though no evidence existed.

Thus, there can be two separate, but sometimes related, mechanisms responsible. Both involve a breakdown of the blood-brain barrier. The first involves a direct effect of endotoxin on the respiratory centre. The second involves a breakdown of cerebral circulation and, indirectly, a cessation of function of the respiratory centre.

The rapid effects of hypoxia on certain blood coagulation factors and fibrinolysis is well known. A coagulation/bleeding disorder follows on from this. Immediately, one can understand that there are features in the developing pathology of so-called ‘shaken babies’ that are similar to what is found following obvious cerebral trauma—for example, disseminated intravascular coagulopathy complicating cerebral trauma.

How does one separate the ‘spontaneous’ causes (via endotoxin/ excessive vitamin C utilization) from somewhat similar pathologies found in trauma?
The answer lies in:
1. the case history – sometimes a difficult issue
2. the presence of pathologies (outside the brain, such as scurvy type bone changes) suggestive of increased utilization of Vitamin C
3. factors that can lead to, or cause, endotoxaemia.

AN INCREASED UTILIZATION OF VITAMIN C

It is known that requirement for Vitamin C may vary from one individual to another by a factor of 1,000 - or even more. Furthermore, there are many conditions that may respond to Vitamin C only when this is administered, in a large dose, by injection.

Dr Sherry Lewin, in *Vitamin C. Its Molecular Biology and Medical Potential*, pages 182-183, states:
"It follows that the variation in vitamin C requirements by different individuals allowing for the various parameters noted, is of the order of a hundred – to a thousand fold…the range is likely to lie between the very approximate limits of 0.2 to 10 g daily."
Scurvy can occur when an infant is supplemented with the recommended daily allowance of Vitamin C – or more.

Hess, page 228-229, states:
"We have met two cases of recurrences in infants, one of which is of particular interest as it happened in spite of giving lemon juice in the intervening period…It improved, but during the winter had bronchitis, otitis, enteritis, and later furunculosis. In spite of the fact that it had been receiving an antiscorbutic for almost this entire period it developed scurvy once more."

This matches my own experience – infants, under my care, developed scurvy (usually following infections) despite being supplemented with more than the recommended daily allowances of Vitamin C. The use of Vitamin C, administered by injection, was a dramatic ‘discovery’ by myself in 1967 – or so I thought, because I later found that other practitioners, in other parts of the world, had beaten me to it by many years.

Endotoxin is often the precipitating factor for an increased utilization/need for Vitamin C. Furthermore, an intramuscular or intravenous injection of Vitamin C can almost instantly ‘detoxify’ endotoxin; and free radical reactions, set in train, are quickly quenched.

There is no standard, typical, presentation of scurvy. Certainly, some presentations have been highlighted, but the absence of all the signs of typical scurvy does not negate a diagnosis.
Furthermore because of:
1. The administration of antibiotics.
2. The administration of vaccines
3. Failure to exclusively breast-feed
4. The role played by endotoxin
infantile scurvy is presenting at an earlier age than it did 75-100 years ago.
And the mode of presentation and the nature of the pathologies, are different, in many respects, to what is usually described in the literature as 'classical' infantile scurvy.

INFANTILE SCURVY

Present-day infantile scurvy is mainly a mixture of scurvy and endotoxaemia. Scurvy and endotoxaemia cause haemorrhages by different mechanism, although the two, more often than not, combine.

From a purist's point of view, scurvy is a disease caused by Vitamin C deficiency and affecting collagen formation. Certainly, Vitamin C is necessary for more than one of the stages involved in the synthesis of the complex triple helix that is a feature of the four types of collagen.

Faulty collagen formation—the feature of scurvy. This affects many tissues, including bone and blood vessels. The result is:
1. Bruises and haemorrhages.
2. Some complex bone pathologies that, to the uninitiated, look like trauma-induced fractures.

Bruises and haemorrhages can occur anywhere – including:
1. Subdural.
2. Other intracranial areas
3. Retina
4. Spine.
5. Skin.
There is no typical area for bruising and haemorrhaging. And there is no typical distribution.

Bone pathologies include:
1. Periosteal elevations with underlying haemorrhages that quickly ossify in a manner similar to ossification that occurs in haemorrhages that surround fracture sites.
2. Epiphyseal disorders, primarily due to breakdown of collagen formation,

complicated by haemorrhages. There may be separation of the epiphyses – resulting in 'pathological' fractures.
3. Weakening of bone structure, due to faulty collagen formation.

A 'favourite' site for epiphyseal changes is the costochondral junctions—the so-called 'scorbutic rosary', or 'beading'. This, in individual cases, may involve only one, or several ribs. The areas involved quickly heal with callus formation. The bone pathologies can occur in any bone, in one bone (or area of a bone) at a particular time, and in another area at a different time. This 'reinforces' the impression that an infant has been physically abused on multiple occasions. One overseas case that I am investigating at the moment had 32 'fractures' of various ages.

The fractures may be painless and not detected by a clinical examination. Much depends on how quickly the scorbutic process proceeds. Relatively slow onset of a scorbutic bone pathology may not present with signs typical of inflicted injury (pain or tenderness). Yet, X-rays may show many fractures. The so-called 'frog-leg posture', with pain and tenderness (due to haemorrhages) does exist. However, mechanisms involved are complex, and the posture can present, together with pain and tenderness, without haemorrhages – and be relieved quickly, and dramatically, in a matter of minutes, by administering Vitamin C by injection.

I do not understand the mechanisms involved. But there is a striking resemblance to some of the features found in acute poliomyelitis. Because of the dramatic response to injections of Vitamin C I suspect that endotoxin is involved.

COAGULATION/BLEEDING DISTURBANCES

It is necessary to highlight several details.

1. Coagulation factors are not fully understood – despite the presence of an enormous amount of knowledge that has been instrumental in the saving of many lives.
2. Standard coagulation profiles may be normal but the patient may bleed to death.
3. Extensive coagulation profiles may reveal a problem not revealed by standard profiles.
4. Platelet function tests should always be a part of extensive profiles despite known difficulties that exist with infants..
5. In typical scurvy, standard coagulation profiles can be normal.
6. In typical scurvy, capillary fragility tests are abnormal.
7. Endotoxaemia (a 'cause' of scurvy) can disturb coagulation/bleeding factors and result in bleeding.
8. In shaken baby cases a diagnosis of 'shaking' is usually made at an early stage, and it is then not considered necessary to search for signs of endotoxaemia, or anything

else.
9. Strangely, cost has been advanced as a reason for failure to thoroughly investigate.
10. One ‘authority’ demanded that I produce retinal haemorrhages ‘with retinal haemorrhages due to scurvy written all over them’ [my words]. What he really asked for were features of scurvy-induced retinal haemorrhages that differentiated them from other retinal haemorrhages. This was quite absurd. Retinal haemorrhages due to scurvy can occur anywhere in the retina. They are, simply, haemorrhages, and have no specific features – unless one uses technologies like electron microscope studies.
11. It is of interest to note that during autopsies on SIDS cases liquid (uncoagulated blood) is sometimes found. This demonstrates the presence of a coagulation disturbance – confirmed by extremely high D-dimer levels.
12. Platelet functions may be abnormal in some cases of scurvy, but it is difficult to separate scurvy and endotoxin as causes. The two interact together in many cases.

Sushkevick et al, *Vopr Pitan,* 1969 Sept-Oct;28(5):23-7. state:
" Experiments were staged on 25 healthy and 38 guinea pigs with vitamin C deficiency. The blood platelets count, their adhesiveness, thrombocytogram, factor X111 activity, thrombotic test, bleeding time and the volume of blood lost were determination the 18-22nd day of keeping the animals on a scorbutogenic diet, parallel with analogous determinations in controls. In animals suffering from C-avitaminosis the number of blood platelets dropped, thrombocytograms demonstrated a decrement of mature forms of blood platelets and an increase in degenerative forms. This was attended by reduced adhesiveness of blood platelets and factor X111 activity and also by deranged structure and properties of the fibrin clot. Hence, disrupted formation of full-fledged thrombocyte plugs and fibrin clots with impaired vessels which in scurvy have reduced mechanical strength, this resulting in lengthening of the bleeding time and in increased volume of blood lost."

This does not mean that every case of infantile scurvy presents with all these features. However, it is a very significant report. Obviously, coagulation factors should be intensely investigated in all shaken baby cases. The extreme example of haemorrhage complicating a viral infection is seen in Ebola fever. With an extremely short time of presentation, most patients ‘bleed from everywhere’. I never cease to wonder why intravenous Vitamin C is not used to treat this awful disease—and used early.

DIFFUSE AXONAL ‘INJURY’

The use of italics for the word ‘injury’ is deliberate because the pathology involved is not always caused by an ‘inflicted injury’.
Kaur et al, *J Clin Pathol.* 1999;52::203-209, state:
" Conclusions – Axonal bulbs staining positively for _APP 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."

Rosomoff et al, Crit Care Med, 1996, Feb;24(2 Suppl):S48-56, state:
"Severe traumatic brain injuries are extremely heterogenous. At least seven of the secondary derangements in that have been identified as occurring after most traumatic brain injuries also occur after cardiac arrest."

Geddes et al, *Neuropathol Appl Neurobiol* 2000 Apr:26(2):105-16, state:
"The lack of correlation between well-documented histories and neuropathological findings means that in the interpretation of assault cases at least, a diagnosis of traumatic axonal injury or diffuse axonal injury is likely to be of limited use for medicolegal purposes."

Little more needs to be said about this except to state that, in many cases, diffuse axonal injury is presented as clear evidence of shaking. The animated video showing how the brain is supposed to move inside the skull when a baby is shaken, and 'tear' the axons apart, certainly impresses the judge and the jury. The effect of that video is, indeed, dramatic. Attempting to counter that, even with extensive references from the best medical literature, is difficult—particularly when the video is presented by authorities with impeccable qualifications.

ENDOTOXIN

Endotoxins can have extremely rapid actions – and, often, this is virtually instantaneous. Although, in this paper, I refer to 'endotoxin', other toxins of bacterial origin can have somewhat similar actions. It is possible to estimate the amount of endotoxin in blood and CSF – during life and during an autopsy. Endotoxin can specifically 'target' the brain via mechanisms involving specific sensitivity of the cerebral capillaries to endotoxic damage. This allows endotoxin to seep into the brain tissue. Cerebral anoxia can follow. Anoxia can, of course, by itself cause a breakdown of the blood-brain barrier. Free radical reactions are immediately initiated and accelerate violently. The result is cerebral haemorrhage, retinal haemorrhage and/or cerebral oedema that can begin and accelerate with dramatic rapidity.

How to look for endotoxaemia

1. Can be measured directly.
2. Look for 'toxic' strains of intestinal bacteria.
3. The administration of vaccines can result in changes in the nature of intestinal bacteria and excessive endotoxin formation.

4. The administration of antibiotics can result in excessive endotoxin formation.
5. Failure to exclusively breast-feed has already been mentioned.
6. Culture blood and CSF, during life and during the autopsy. Cultures should include viral cultures, which can, by indirect mechanisms result in excessive endotoxin production by gut bacteria.
7. Check for otitis media – which is sometimes associated with endotoxaemia. In the Australian Nanny case there was no sign of otitis media when the baby was first admitted to hospital following the final collapse. Yet during the autopsy , a few days later, 'muco-purulent' material was found in both middle ears.
8. Check liver and kidney functions – which can be disturbed by endotoxin.
9. Check liver and kidney histology – including electron microscope studies.
10. Electron microscope studies of several parts of the gastrointestinal tract may show signs of endotoxin damage or abnormal adhesion of bacteria to the gut wall.
11. Electron microscope, and light microscope, studies of blood vessels (including capillaries in the brain) may reveal signs of endotoxin damage.
* Note that it is not necessary to have a raised leucocyte count to have raised levels of endotoxin.

THE ROLE PLAYED BY VACCINE ADMINISTRATION

I would like to avoid this subject but cannot do so.
It is not a matter of whether vaccines should or should not be used.
It is a matter of – "Is there a role for vaccines in the pathogenesis of the Shaken Baby Syndrome?"

In several cases (probably a significant number) the final collapse followed within a very short period of a vaccine administration. In the Sally Clarke case, this happened with her two babies. She refused to have her third baby (born after she was charged) vaccinated.

There is no doubt, in my mind (and this is based on long experience) that despite advice to the contrary it is not wise to administer vaccines to sick infants—including infants with 'colds'. This is because, with infections (including 'colds'), endotoxin is likely to be produced in the gut in excessive amounts, and liver detoxification processes are likely to be stressed. Immediately, some practitioners are going to state that in many situations some infants 'always have colds'. This applies particularly to groups such as Australian Aborigines. The answer to that is to supplement, first, with Vitamin C and zinc. Risks will then be reduced enormously (but not completely).

Mechanisms involved with vaccine administration include excessive endotoxin formation. Knowing this allows one to follow the remainder of the pathway towards the development of the pathologies found in so-called 'shaken babies'.

STRANGE PSYCHOLOGY THAT LEADS DOCTORS TO IGNORE IMPORTANT DETAILS IN ORDER TO MAKE A GUILTY VERDICT APPEAR MORE LIKELY

There are several aspects to this:

1. Infants are said to have been 'healthy and normal' before the final collapse despite a long history of medical problems, such as repeated infections and the administration of multiple courses of antibiotics.
2. Many infants were hospitalised several times before the final collapse.
One infant, for example, was on an intravenous drip for several days, for what was called (by specialists) 'a trivial illness'.
3. The inappropriate administration of medications.
One case, being investigated at the moment, was given (inappropriately) frequent doses of promethazine.

Parry EW. *Inflamm Res* 1996 Jul;45(7):354-6, states:
" The three drugs (dexamethasone, promethazine and nordihydroguauaretic acid) have the ability to inhibit powerfully the synthesis of tumour necrosis factor-alpha in response to lipopolysaccharide [endotoxin] …it is proposed that
the three drugs act by impairing the hepatic mechanism which normally removes portal vein-borne endogenous lipopolysaccharide leading to systemic distribution of lipopolysaccharide."

One problem, demonstrated here, is that many practitioners are too busy and cannot find the time to study everything. I have a 'bone to pick' with this issue, because, for 30 years I have been trying to interest my colleagues about the potential dangers involved when antihistamines are administered to infants. The response has been one of extreme hostility.

MOVE THE GOALPOSTS AND IT IS POSSIBLE TO PROVE ANYTHING

With shaken babies this is done in several ways:

1. By 'declaring' that certain pathologies are 'diagnostic', when there are serious doubts about the logic involved.
2. By accepting that 'convictions' are certain evidence of guilt, and the features found in these cases are therefore diagnostic of the shaken baby syndrome.
3. By the process of 'plea bargaining' – where it is made clear to the defendant that to plead innocent will result in a charge of murder and a long jail term (or even execution), and a guilty plea will result in a lesser charge (such as involuntary manslaughter) and a short sentence.

4. Many of those charged have limited financial resources and cannot afford good lawyers.
5. Publishing what are called ‘position papers’ and ‘a consensus’, that generates an impression of ‘finality’. That is; the matter is settled and there is no need to examine it further.
6. By deliberately withholding important information from the defence in order to strengthen the case for the prosecution.

The ‘Australian Nanny’ (Louise Sullivan) case was one of the first where lack of knowledge (and an absolute determination to achieve a conviction) generated disturbing responses. It was felt that Louise had ‘killed’ the child, but lacking was an important piece of evidence. The prosecution could not understand what initiated the rapid onset of cerebral oedema. It was, of course, cerebral anoxia, caused by endotoxin, but the prosecution did not know this. So it was deduced that Louise not only shook the infant but suffocated it as well.

In order to win a conviction, it was decided that if Louise was 'psychologically softened' first, then hit with an invented piece of evidence, she would ‘confess.' I will not detail how the ‘softening’ was carried out—but it was not nice. Then Scotland Yard said to Louise, "We have re-examined the lungs and can prove that you suffocated the infant as well as shook it. Plead guilty to a lesser charge of involuntary manslaughter and your sentence will be light. Plead innocent and you will be charged with murder and spend many years in jail." Louise accepted the plea in order to save her sanity. She was allowed to go home.

I then asked to see the reports of the lung examination. Clearly stated was: "The lungs are normal. No evidence of anoxia." Unfortunately, that ‘little’ detail was omitted from the book that was later written about the case.

The recently finalised Sally Clarke case (England) demonstrated the existence of the strange psychology involved in the last (number 6) factor, in a dramatic manner—after Sally had spent more than 3 years in jail for ‘killing’ two of her babies.

The first died, and an autopsy resulted in a diagnosis of ‘cot death’. Later, a second baby died, and the authorities claimed that this was because it was ‘shaken’. Then the first death was ‘reconsidered’ and the authorities said that this, also, was ‘shaken’. Paediatrician, Professor Sir Roy Meadow, stated that the likelihood of having two cot deaths in one family were one in 73 million. This certainly impressed the court. Then the Home Office Pathologist, Dr Alan Williams, withheld vital evidence from the court. This concerned the fact that cultures were taken from the blood and CSF from the second baby—and these showed that the baby was ‘riddled with potentially lethal organisms’ (staph. aureus).

After more than 3 years, Sally's husband was finally able to obtain the full medical records to which the defence had previously been denied access. And a 'proper statistician' showed that the chances of having two cot deaths in one family could be as low as one hundred to one. Sally was freed.

Now the two 'experts' who gave false/wrong evidence are in real trouble. The question that should be asked is - Why did the eminent doctors behave in such a fashion? It appears that they were hell-bent on achieving a prosecution and cared little about how this was achieved.

In another case an eminent paediatrician was asked to comment about a detail in my report to the court, where I stated that the infant concerned 'had endotoxaemia'. He declared something to the effect that this could not be so because 'the infant was not sick'. Some time later he was asked why he did not transfer the infant to another hospital (as recommended by an ophthalmologist) in order to have the retinas photographed for evidence. He answered with something like 'We could not, because he was too sick'.

I have no doubt that this 'shaken baby' business will eventually be recorded as one of the worst pages in the history of paediatrics. And the saddest part of it all concerns the fact that, while important doctors are busy collecting 'evidence' for the prosecution, vital issues that can save many lives are being not only ignored but destroyed with intense hostility.

During one trial, the prosecution stated that infantile scurvy was no longer seen. I replied with 'Yes it is. But it is not called 'scurvy' it is called the 'shaken baby syndrome'.

Failure to properly consider the case history.

Usually, the prosecution attributes all problems suffered by an infant previous to the final collapse as caused by abuse. For example, if an infant has 'colic' this is diagnosed as being due to abuse. Crying is also considered to be due to abuse. Of particular concern is the tendency to disregard concern expressed by parents, and then claim that the parents were negligent. Frequent admissions to hospital (for infections or undiagnosed problems), and frequent examinations by doctors (including paediatricians) are usually, in retrospect, after the final collapse, left unexplained or considered to be part of the chronic abuse syndrome.

If a premature infant, while still in hospital after birth, develops intracranial haemorrhages or periosteal haemorrhages the diagnosis is usually considered to be 'a normal risk that all premature babies are exposed to'. However, if one of these infants is diagnosed with one or more of these disorders soon after being allowed home the

chances are that someone will be charged with abuse. Radiologists report the presence of periosteal elevations in premature infants as ‘a normal variant’. Mechanisms responsible are rarely considered.

When the findings, either before or after death, cannot be explained by a carer, this is regarded as ‘an inconsistency’ consistent with abuse. It is considered by authorities to be a diagnostic feature of abuse.

In many ways this is like the ‘witch hunts’ of old.

Because of knowledge that is now available, prosecutors (including doctors) who provide evidence suggestive of guilt, without considering the factors stated in this lecture, possibly, later, may be charged with criminal negligence. Furthermore, the issues involved are extremely complex. No single doctor can possibly claim to be an ‘authority’ on every aspect of every factor involved in the genesis of the pathologies found in shaken babies. However, sufficient knowledge exists to enable one to at least cast serious doubt on the inflicted trauma hypothesis of the pathologies found in some so-called ‘shaken babies’.

For those of you who want to investigate the subject of shaken babies in greater detail I suggest that you connect onto PubMed, and Google. Type in endotoxin, vitamin C, blood-brain barrier, coagulation/bleeding disorders, platelets, Factor X111, axonal injury, shaken babies, collagen, and free radicals. Be prepared to spend a few years doing so, because there is an enormous amount of literature available.

I do not doubt that it is possible to shake a baby to death. However, in the 35 cases I have extensively investigated, there were substantial reasons to conclude that shaking was not the cause of the pathologies found.

http://www.freeyurko.bizland.com/kaloksb1.html