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Shaken Baby Syndrome Diagnosis On Shaky Ground

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Abstract

An epidemic of accusations against parents and baby sitters of shaken baby syndrome is sweeping the developed world. The United States and the United Kingdom are in the forefront of such questionable practice. Brain (mainly subdural, less often subarachnoid) and retinal haemorrhages, retinal detachments, and rib and other bone 'fractures' are considered pathognomonic. However, the reality of these injuries is very different and well documented: the vast majority occur after the administration of childhood vaccines and a minority of cases are due to documented birth injuries and pre-eclamptic and eclamptic states of the mothers.

Evidence that vaccines cause brain and retinal haemorrhages and increased fragility of bones, has been published in refereed medical journals. That this has been to a great extent due to the depletion of vitamin C reserves resulting in acute scurvy, has also been published. I refer to such articles and demonstrate that there is a viable differential diagnosis available explaining the observed injuries in what is called the Shaken Baby Syndrome (SBS) as non-traumatic injuries, and that the diagnosis of SBS is an incorrect evaluation of the cause of such injuries; it has resulted in unspeakable injustices and suffering for the affected individuals and their families, and deprived the surviving babies of their parental care by replacing it with foster care. It does not reflect well on the justice and medical systems in the developed world which are, sadly, characterised by blindness to the most obvious and victimisation of the innocent. Those who inject babies with great numbers of vaccines within short periods of time in the first months of life, often ignoring the observed serious reactions to the previous lots of vaccines, are not only the accusers of innocent carers, but are not prosecuted or brought to justice; quite to the contrary, they continue injecting babies with toxic cocktails of vaccines and creating further innumerable cases of grievous bodily harm and death.

Introduction

A great number of parents and other carers are being accused of shaking their small babies and causing grievous bodily harm and death. Most USA, but also other countries', hospitals have SBS squads who get hold of distraught carers rushing their seriously ill or dying babies to get help literally on entering the emergency units, often before any tests are done. In Australia, the affected babies are taken away from their parents, who are not always charged with any criminal offence, but have a hard time to prove that they did not harm their babies and have difficulty getting them back into their lawful custody.

Nobody seems to listen to the carers' stories, which are remarkably similar in the lack of evidence of any trauma and in that they are at a loss to understand what happened to their precious babies. Even though the administration of vaccines is recorded, their possible role in the observed injuries is not considered, or is deliberately ignored. The distraught carers are, as a rule, pressured into admitting that they shook their babies and some may 'confess' under duress or under a promise of leniency. It is only thanks to the Internet that a great number of carers subsequently learn that the vast majority of the affected babies developed their symptoms after and not before they were subjected to the routine vaccine injections.

The most worrying element in this misplaced eagerness to 'protect' babies against abuse, is the ignorance of the medical 'experts' who adamantly, and under oath in court, will testify that there is no evidence (published or otherwise) or "no reputable evidence" that the observed injuries, considered pathognomonic of SBS, have other, viable, non-traumatic causes. In my experience, such experts adamantly reject any suggestion that the administered vaccines had anything to do with the observed injuries.

I dare not say that eagerness to deliberately victimise carers is the motive in their unforgivable behaviour, even though this phenomenon has been described in refereed medical journals. Kirschner and Stein[9] warned about mistaken diagnoses of child abuse based on the failure of treating physicians to make a correct diagnosis and that they mistake life-threatening illness or postmortem artifacts for inflicted injury. They wrote: "Not only lack of experience with severe childhood illness and death but also an attitude of suspicion and/or hostility probably contributed to these misdiagnoses".[9]

I have also witnessed the 'experts' admitting that, of course, vaccines are not 100% safe or effective and can cause injuries, but not in the case under scrutiny!

There are a number of benchmark articles referred to by the proponents of the SBS, Caffey's 1972 article[2] being the most quoted. Since other authors essentially repeat what was published by Caffey, I shall only elaborate on the data contained in the above publication of Caffey's.

Analysis and comparison of the published data

Caffey[1] originally described six infants, 13 months or younger, with the combination of subdural haematomas and what he considered characteristic "bone lesions of battering". In 1972, Caffey talked about the theory and practice of shaking infants as part of his Abraham Jacobi Awards Address. He ascertained that during the last 25 years substantial evidence, direct and circumstantial, "has gradually accumulated suggesting that the whiplash-shaking and jerking of abused infants are common causes of the skeletal as well as the cerebrovascular lesions". He also wrote that "potentially pathogenic whip-lash shaking is commonly practised in a wide variety of ways, under a wide variety of circumstances, by a wide variety of persons, for a wide variety of reasons".

He considered that the most common motive for such action was an attempt to correct minor misbehaviour. However, he also wrote that the line of demarcation between pathogenic and nonpathogenic shaking is often vague. He maintained that the interpretations of such injuries must be done from the radiographic changes exclusively due to the lack of systematic studies of either surgical exploration or necropsy.

Metaphyseal avulsions in the form of small fragments of cortical bone torn off the external edge of the cortical wall at the metaphyseal levels where the periosteum is most tightly bound down to the cortex were most common. In most cases they appeared to be small chunks of calcified cartilage which have been broken off the edges of the provisional zones of calcification at or near the sites of the attachments of the articular capsules. Bones on both the proximal and distal sides of a single joint were affected, especially at the knee.

Then in 1972[2] Caffey proceeded to speculate that "all of these metaphyseal avulsions appeared to result from indirect traction, stretching, and shearing, acceleration-deceleration stresses on the

periosteum and articular capsules, rather than direct, impact stresses such as smashing blows on the bone itself". Then, without further evidence, he called these findings "traumatic involucra" which commonly accompany the metaphyseal avulsions and involve the same terminal segment of the same shaft. He thought that such injuries develop due to traction-rupture of the abundant normal perforating blood vessels, which occur between the periosteum and the medullary cavity and which are severed at the junction of the internal edge of the periosteum with the external edge of the cortex. The accumulated blood then lifts the periosteum off the wall for variable distances and forms subperiosteal haematomas of variable sizes and shapes.

The position of such haematomas varies from either being symmetrical in analogous bones, or asymmetrical, affecting bones in one arm or leg only, or sometimes occurring only on the thighs and shanks. Frequently, they involve bones on both the distal and proximal sides of a single joint, especially the knees. First they appear as masses of water density superimposed on the shaft, but after four to ten days, a thin opaque shell of new fibrous bone forms around the external edge of the haematoma. The entrapped blood then gradually resorbs.

Importantly, Caffey quoted several observers who noted associated diffuse sclerosis of the shafts of some of the affected bones of some 'abused' infants.[2] Importantly, he wrote that some radiologists described them as excessively fragile, brittle, chalk-like bones. He then continued:

> "In recent biopsies, however, the microscopic examination disclosed the lamellae to be laid down in an irregular woven pattern. This, in my opinion, indicates that the sclerosis is caused by excessive newly-formed primitive fibrous or woven bone, which forms regularly under the periosteum following traumatic subperiosteal oedema or bleeding or both. Traumatic thickenings of the external subperiosteal edge of the cortical wall are the cause of the sclerosis. The epiphyseal ossification centers and round bones are probably stronger than normal shafts. Traumatic metaphyseal cupping is due to traumatic obstructive injury to the epiphyseal arterioles in the neighbouring cartilage plate; and the metaphyseal 'loop' deformities are due to stretching and extension of the traumatic involucra terminally. Both of these lesions are best explained on the causal basis of the grabbing, squeezing the extremities by the assailant's hands, and whiplash-shaking the infant's head".[2]

It is obvious from these quotations, that Caffey was preoccupied with presumed but unsubstantiated, let alone proved, traumatic origin of such injuries and he ignored the available evidence to the contrary.

Indeed, Caffey's contemporary, Hiller, published a very important article in 1972 ("Battered Or Not – A Reappraisal Of Metaphyseal Fragility")[6] in which he demonstrated that the 'typical' epiphyseal plate fractures – usually involving a flake of metaphysis, with or without displacement of the epiphysis, and considered virtually diagnostic of battering – are something else: a sign of acute scurvy. Hiller wrote that such fractures often produce periosteal stripping up of the shafts of the bones, with added subperiosteal haematoma formation, which later shows extensive ossification.

Hiller also mentioned that this type of fracture was originally described by Caffey in 1946, who noted the occurrence of such fractures in infants with subdural haematomas, but drew no conclusion at that time. Indeed, Caffey at that time even coined the term "metaphyseal fragility" and for a long time afterwards infants presenting with these types of bone fractures were fully investigated to exclude blood dyscrasias, clotting abnormalities and abnormalities of calcium metabolism. However, nothing ever came of these investigations and subsequent authors simply accepted such fractures as being the result of inflicted trauma. By 1965, Silverman published that such bizarre fractures should now be accepted generally as strong evidence of battering.[19]

Hiller maintained his reservations about the validity of such hypothesis. He had many reasons for such hesitation:

1. the inability on many occasions to elicit, even by most careful and thorough cross-questioning, any evidence of maltreatment (indeed one of such infants was a doctor's son);
2. the type of trauma (when any had occurred) reported by proponents of the SBS was so minimal, that it could occur in a high percentage of normal home environments, and we should definitely see more of it; in actual fact, this did not occur;
3. in a number of infants with multiple epiphyseal plate fractures, all bones, including those in no way involved with the fractures, show a dense chalky appearance on the x-ray, which, in fact suggests a degree of osteopetrosis.

   Both Caffey and Silverman[19] recorded these findings but did not draw the correct conclusion from them;
4. Hiller drew attention to the occurrence of epiphyseal plate fractures on both sides of a joint – often appearing at the same time on x-rays and therefore almost certainly being sustained at the same time. If any twisting or torsion had occurred, as Caffey and his followers hypothesised, such a fracture might occur at one side of the joint, but "how could it possibly occur at both sides?" asks Hiller.

Hiller then discussed the occurrence of other fractures accompanying the above typical fractures of scurvy, such as multiple rib fractures and skull vault fractures. He wrote that the occurrence on more than one occasion of a fracture of the acromion and of spinous processes, causes some difficulty in accepting the trauma-alone theory. Also, greenstick fractures of a number of metatarsals in a 4-month-old infant are difficult to explain as being caused by inflicted trauma.

Indeed, Hiller instigated a two year retrospective study at the Royal Children's Hospital, Melbourne, of all long bone fractures seen in infants and children under the age of 3 years, in whom no stigmata of battering were found. The study included three groups of children: those with no known injury, those who sustained severe falls from heights or had been involved in automobile accidents, and those who had less severe falls, being dropped or having been subjected to the playful actions of parents. The results showed that out of a total of 145 fractures reviewed, not one was of the epiphyseal plate type. They were invariably greenstick, oblique or spinal shaft fractures.

At the same time it was decided to make a 12-month survey at the Royal Children's Hospital, Melbourne, of all children and infants suspected of having been battered, and to ascertain how many of these demonstrated the typical epiphyseal plate fractures. Out of the total of 25 children, 5 were found to have these typical fractures. All 5 showed the chalky bone structure to a greater or a lesser extent and all showed multiple fractures not only of the epiphyseal plates, but also shaft fractures and, in one case, a linear skull fracture. In 2 of the 5 patients, a bone biopsy was performed which showed an abnormal trabecular structure. "The lamellae were found to be laid down in an irregular woven pattern, i.e. the collagen framework showed a criss-cross basket weave of bundles". Hiller stated that this irregular collagen pattern resembled that of immature woven bone, and contrasted with normal controls which showed a more regular lamellar pattern.

Long before both Caffey and Hiller, Hess published a book *Scurvy, Past and Present*[7] in which he elaborated on many typical signs of scurvy involving many types of haemorrhages which may take place in any organ and vary from small petechiae to very extensive extravasations. The hair follicles and sweat glands are particularly susceptible to such bleeding, as some authors noticed in inmates of French prisons.

Relatively small trauma may result in bleeding into the skin, the lower extremities being the commonest sites, between the knee and ankle, and in children the inner aspect of the thighs due to trauma of the nappies. The deeper haemorrhages may be very extensive and tend to follow the connective-tissue strata. The blood surrounds the muscle fibres, which appear quite intact. The neighbouring blood vessels are congested and may contain thrombi, both venous and arterial. Such thrombi are found also in areas where extravasation has not taken place, and conversely, haemorrhages occur where no thrombi are demonstrable. Brownish pigment (no doubt haemosiderin, my comment) is frequently found in the neighbourhood of the haemorrhagic areas.

In the healing areas a marked formation of the scar tissue will be found. Bizarre forms of haemorrhage may occur in the right lower abdomen and in the region of the transverse colon. Certain organs are more and others less predisposed to bleeding. Haemorrhages are commonly seen in the adrenals, (mainly the medullary portion), bladder and urethra. Haemorrhages may occur into the brain substance, into the cord or into the membranes surrounding them. What Hess called "*pachymeningitis haemorrhagica interna*" has been described frequently, and may give rise to the symptoms of meningitis. The optic nerve and peripheral nerves may also be affected.

With modern technology, such haemorrhages are now identified as subdural or subarachnoid and can be described in great detail. Even though they may occur separately from brain haemorrhages, retinal haemorrhages occur frequently when the brain haemorrhages are present, because of the close anatomical connection between the eyes and the brain.

Bones are affected by subperiosteal haemorrhages, especially in the distal end of the femur or of the tibia, which may be evident and surrounded by unusually large calluses. The blood may extend along the long bones under the periosteum. The clot forms, readily demonstrating that the nature of the haemorrhage is not a defect in coagulation and the callus constitutes more or less firm connective tissue containing fibrin, pigment and granulation tissue. Epiphyses may be entirely separated from the bones. In some cases the cartilage is telescoped into the crushed end of the bone. Typically, 'beading' of the ribs occurs, the counterpart of the 'rhachitic rosary'. There may be fractures at the costochondral junctions, or a separation of the cartilage from the sternum, as described by Lind and many others in soldiers suffering scurvy.

The subperiosteal haemorrhage has long been recognised as a lesion characteristic of scurvy and may involve almost any of the bones, such as scapula, cranial vault, orbital plate of the frontal bone, ribs, etc. The most frequent sites of fracture or separation in the epiphysis is the lower end of the femur. On sectioning the bones longitudinally, the cortex is noted to be exceedingly thin (a mere shell) and very brittle. The trabeculae are so thin and reduced in number that the bone has become a very fragile structure. The marrow is no longer deep red at the ends of the long bones, but yellowish, frequently presenting a patchy appearance. It has a gelatinous consistency. The bone structure is irregular with osseous trabeculae few in number and those remaining are slender and irregular and frequently appear as isolated islets. The line of junction with the cartilage becomes zigzag.

Retinal haemorrhages and pallor of the optic disc, found in SBS, were also considered pathognomonic of scurvy.

Enlargement of the heart was cited by Hess[7] as one of the symptoms of acute scurvy. Pericarditis, hydrothorax, pleurisy with effusion, diarrhoea, bleeding into the gums and pneumonia are common complications of scurvy.

In babies, the signs of scurvy may be overlooked even though it is indicated by a large number of symptoms such as irritability, tachycardia and tachypnea, slight weight gain, pale complexion and slight oedema of the eyelids and periorbital area. Petechial haemorrhages on the face, around the eyes and on the upper torso are one of the typical signs of scurvy in infants. In this connection, it is appropriate to mention petechial haemorrhages into the thymus, pericardium, lungs, and other organs as the most typical (and often only) pathology found in the babies who die suddenly (SIDS). These may well be signs of acute scurvy precipitated by the administration of a multitude of vaccines containing a number of toxins. The petechiae were well-described in 1978 by Hans Selye[16] (and elsewhere), as part of the pathology of his non-specific stress syndrome (or general adaptation syndrome) in rats injected with formaldehyde, and also in 1959 by Pekarek and Rezabek in rats after the administration of the DPT vaccines.[11]

All examples of what Caffey considered "typical battered baby" fractures and periosteal bleedings in his papers, are in fact typical scurvy fractures and bleedings.

These days, people generally think that nobody suffers scurvy, which used to be identified with long sea voyages during which the sailors were deprived of any fresh fruit and vegetables. The reality is far from such idealised perceptions. Most people probably have only marginal reserves of vitamin C and this applies particularly to babies and small children. Administration of vaccines depletes the marginal vitamin C reserves very quickly and this results in acute scurvy.

Vaccines of the kind given to babies as early as at birth and then one month later (hepatitis B vaccine) and DPT, Polio and Hib at 6 to 8 weeks of age, contain a number of toxins. The DPT (three in one vaccine), being the toxoid vaccine, contains pertussis, diphtheria and tetanus toxins which are treated with formaldehyde to decrease their toxicity. However, all of these treated toxins (toxoids) have the ability to revert back to their original toxicity by passage in the injected individuals, as demonstrated by Samore and Siber.[13] These toxins are capable of causing, and they demonstrably cause, serious immunological, vascular and metabolic injuries, of which scurvy is one of many documented mechanisms.

Weiss and Hewlett[17] elaborated on virulence factors of *Bordetella pertussis*, the causative organism in the disease pertussis and the active ingredient in all pertussis vaccine, whether the whole-cell or acellular. They enumerated the following virulence factors of Bordetella pertussis: agglutinogens, adenylate cyclase toxin, dermonecrotic toxin, filamentous haemagglutinin, haemolysin, lipopolysaccharide, pertussis toxin and tracheal cytotoxin. Importantly, dermonecrotic toxin causes necrotic lesions and elicits vasoconstriction of peripheral blood vessels, followed by ischemia, diapedesis of leucocytes and petechial haemorrhage. The lipopolysaccharide is pyrogenic and is comparable to endotoxin from *Salmonella* or *Escherichia coli* in the Limulus amaebocyte lysate essay and in promoting hypersensitivity to histamine.

Pertussis toxin is the most extensively studied product of *B. pertussis* and is undoubtedly a major virulence factor. It is known under a number of names such as histamine-sensitising factor, lymphocytosis-promoting factor, islet-activating protein and pertussigen. Pertussis toxin is not cytolytic but rather alters cellular responsiveness to regulatory molecules. It blocks the stimulation of phosphatyl inositol hydrolysis, arachidonate release, and calcium mobilisation by some mediator cells, such as immune effector cells, including neutrophils, monocytes, macrophages, basophils, bone marrow stem cells, and natural killer lymphocytes. This explains the range of reactions to the pertussis vaccines.

Other vaccines contain toxins, such as the diphtheria and tetanus vaccines, which may and do cause injuries of the kind seen in the SBS babies. However, even vaccines which are not toxin-based, such as the hepatitis B vaccine, cause reactions seen in the SBS babies, particularly retinal haemorrhages (Devin et al.[3]). Perhaps the most important effect of such toxins is arteritis affecting medium and small arteries, particularly at the point of bifurcation and branching. Segmental

inflammation, infiltration with fibrinoid and necrosis of the blood vessel lining and walls leads to diminished blood flow to the areas normally supplied by these arteries (Taber's Cyclopedic Medical Dictionary, 1981).

There is another aspect to the observed brain and retinal haemorrhages in a great number of particularly newborn babies: the iatrogenic effect of inductions. Schoenfeld et al. (1985)[20] studied the retinal haemorrhages following labour induced by oxytocin or dinoprostone. They observed retinal haemorrhages in 40% of neonates in the dinoprostone treatment group, compared with 28% in the oxytocin group. They concluded that accumulation of prostaglandins in the foetal circulation may be responsible for the haemorrhages. They wrote that other organ systems must be carefully examined in the neonates to detect other possible untoward effects.

Considering that large numbers of neonates are now not only induced, but are also injected with hepatitis B vaccine within hours or a few days of birth, it is not surprising that so many of them are diagnosed with extensive brain and retinal haemorrhages; the haemorrhagic birth injuries caused by the overload of prostaglandins used to induce or speed up so many births, are aggravated by the vasculopathic toxic effect of these vaccines. The haemorrhages have hardly had time to start healing and the next dose of multiple vaccines is administered at 6 to 8 weeks of age. Tragically, the presence of haemosiderin, indicating old haemorrhages, is not appropriately correlated with the vaccine administration, but is incorrectly used as further 'evidence' that the child was assaulted repeatedly. Gilliland et al. warned that such interpretation represents an opportunity for medicolegal confusion and miscarriage of justice.[5]

Leadbeater et al.[10], in a letter to the editor of *BMJ*, commenting on Carty and Ratcliffe's article ("The Shaken Infant Syndrome"),[18] warned about the unproven concept of the SBS as a result of violent shaking and quoted Duhaime et al.,[4] who raised the question whether the forces generated by shaking are sufficient to cause brain damage. Leadbeater et al.[10] also justly criticised the way a lack of precision in citation by some authors (proponents of the SBS) is raising false hopes of an objective account of an unbiased witness, "but on studying the cited reference one finds only an unsubstantiated statement of belief that describes the act of 'shaking/slamming'". Leadbeater et al. concluded that "It seems premature to warn against an act of violence when its precise mechanism of action is not clearly defined, its potential for serious trauma in the absence of concomitant impact is not supported by existing experimental data, and the clinical findings said to result from it are not in themselves specific".

This agrees with my experience of SBS from court cases in which the unfortunate parents become victims of such unsubstantiated 'beliefs'. The accused carers are presumed guilty when they are steadfast in proclaiming their innocence and presumed guilty when they 'confess' to shaking, without the court establishing just how strong and when the alleged shaking should have occurred. The courts are often deaf to the fact that the accused only admitted to slightly shaking the baby *after* finding it unconscious or fitting, in their legitimate effort to revive the baby. "You just don't stand there and watch your baby die."

Hess'[7] and Hiller's[6] findings cast very serious and considerable doubt on the acceptance of Caffey's multiple epiphyseal plate, rib and skull fractures as definite radiological evidence of battering. Both authors ascertained that such fractures are common in scurvy without undue trauma to the child beyond normal handling and that greenstick fractures are equally common in rickets. Both conditions result in increased temporary bone fragility, which may result in fractures due to normal handling. It has been amply demonstrated that administration of vaccines, such as DPT, results in depletion of vitamin C reserves, leading to acute scurvy.

Pittman, in her landmark paper on "The Concept of Pertussis as a Toxin-Mediated Disease",[12] quoted Pekarek & Rezabek[11] who reported that toxic pertussis vaccine, as reflected by the mouse weight gain test, causes a temporary decrease in the level of ascorbic acid in the adrenals of the rat. Considering that in contrast to rats, the human species does not produce its own vitamin C, which must daily be replenished by oral intake, the administration of vaccines may result in serious long-term depletion of vitamin C, unless large doses are administered before and after such vaccines are given. Proper investigation of the vitamin C status is imperative in establishing the cause of the observed bone fragility.

Conclusions

The above brief review of the perceived benchmark publications dealing with issues directly related to the diagnosis of Shaken Baby Syndrome, demonstrates that the SBS diagnosis is on very shaky ground indeed. The pathology, considered currently to be foolproof evidence of inflicted trauma, may be caused by inductions and other birth injuries, temporary increased fragility of the bones due to acute scurvy caused by the toxic effect of vaccines and the observed brain and retinal haemorrhages may also be a result of vascular injuries due to the toxic effect of the administered vaccines. Indeed, the only documented facts in the vast majority of cases of SBS are the administered routine vaccines, while the evidence of any shaking, other than slight shaking as part of resuscitation efforts by the carers who found the affected infants in distress, is missing.

There are more plausible mechanisms than shaking which explain the increased bleeding tendency without the standard tests revealing the usual blood clotting disorder due to low platelet count. Hans Selye[16] postulated the presence of liquid unclotting blood due to decreased viscosity of blood as one of the characteristics of the second stage of his non-specific stress syndrome which is caused by the stress dynamics of retention of water rather than changed platelet count.

Indeed, shaking is the most unlikely cause of such injuries.

The practice of accusing innocent carers of injuring vaccine-damaged children should cease forthwith.

All past cases of SBS should be revised and the victims released from prison and compensated for their mental suffering, financial losses and emotional trauma.

The practice of administering toxic substances such as vaccines should be looked into and there must be an independent enquiry, which should include the critics of vaccines, and which should investigate vaccines' proven dangers.

And last but not least: the unjustifiable accusations of innocent parties and harassment of the vaccine victims should serve as a serious warning about the shortcomings of the Western medical and legal systems and their susceptibility to serious errors.

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