**CANNINGS 2004 2 Cr APP R 7**

*“The unavoidable reality is that some infant deaths remain “unexplained” or “unascertained” (para 8)…We cannot avoid the thought that some of the honest views expressed with reasonable confidence (in that case as to cause of death) will have to be revised in years to come when the fruits of continuing medical research both here and internationally become available. What may be unexplained today may be perfectly well understood tomorrow. Until then any tendency to dogmatise should be met with an answering challenge” (para 22…) “Experts in many fields will acknowledge that later research may undermine the accepted wisdom of today “never say never” is a phrase which we have heard in many different contexts from expert witnesses. That does not normally provide a basis for rejecting the expert evidence or indeed for conjuring up fanciful doubts about the possible impact of later research. With unexplained infant deaths however…in many important respects we are still at the frontiers of knowledge. Necessarily further research is needed and fortunately thanks to the dedication of the medical profession it is continuing”* (para 178)

**R V HENDERSON 2010 EWCA CRIM 1269**

*“There are few types of case which arouse greater anxiety and controversy than those in which it is alleged that a baby has died as a result of being shaken. It is of note that when the Attorney General undertook a review of 297 cases over a ten-year period following the case of R v Cannings [2004] EWCA Crim 1, [2004] 1 All ER 725, 97 were cases of what is known as ‘shaken baby syndrome’. The controversy to which such cases gives rise should come as no surprise. A young baby dies whilst under the sole care of a parent or childminder. That child can give no clue to clinicians as to what has happened. Experts, prosecuting authorities and juries must reconstruct as best they can what has happened. There remains a temptation to believe that it is always possible to identify the cause of injury to a child. Where the prosecution is able, by advancing an array of experts, to identify a non-accidental injury and the defence can identify no alternative cause, it is tempting to conclude that the prosecution has proved its case. Such a temptation must be resisted. In this, as in so many fields of medicine, the evidence may be insufficient to exclude, beyond reasonable doubt, an unknown cause. As Cannings at [177] teaches, even where on examination of all the evidence, every possible known cause has been excluded, the cause may still remain unknown.”*

**RE R (CARE PROCEEDINGS: CAUSATION) 2011 EWCH FAM 1715**

*”I have been impressed over the years by the willingness of the best paediatricians and those who practise in the specialities of paediatric medicine to recognise how much we do not know about the growth patterns and what goes wrong in them, particularly in infants. Since they grow at a remarkable speed and cannot themselves give any clue as to what is happening inside them, and since research using control samples is self-evidently impossible in many areas, perhaps we should not be surprised. In my judgment, a conclusion of unknown aetiology in respect of an infant represents neither professional nor forensic failure. It simply recognises that we still have much to learn and it also recognises that it is dangerous and wrong to infer non-accidental injury merely from the absence of any other understood mechanism. Maybe it simply represents a general acknowledgement that we are fearfully and wonderfully made”.*

FOR INFORMATION RE THAT CASE:

*(“ ….. I am deeply unwilling to make a finding of culpable conduct against these parents, unless entirely compelled by the medical evidence to do so. In my view, the court faces four options: first, that the child sustained the fracture whilst in the baby-walker, notwithstanding that explanation’s innate unlikelihood; secondly, which emerged in the course of argument, that the child sustained the fracture when, having in some way hurt himself in the baby-walker, he was yanked from it by the mother in such a way as to cause a twisting injury, but in circumstances where she could not be expected to know that. That is a possible, if inherently implausible, mechanism. Thirdly, that this was an inflicted injury in a momentary loss of control, perhaps whilst changing the baby; and fourthly, that the cause of this fracture is simply unknown. It seems to me that the explanation has to be found in one of these options, but all four options pose serious difficulties. The first two are inherently unlikely, though of course not impossible. Inflicted injury raises the difficulties that I have already referred to. Moreover, there is simply no evidence from which the court could draw any inference of pressure, tiredness, frustration or bad temper at the relevant time in either of the parents, nor indeed have any such circumstances been suggested to them. An unknown cause is very unlikely in circumstances where, quite unlike the head injury, the mechanism and causation of these fractures are generally well understood. An unknown cause must, I think, be rejected in this case. I have given long and anxious consideration to this matter, deeply aware, as Baroness Hale of Richmond has reminded us,that a mistake either way can have serious consequences. As she says, however: ‘It is a task which we are paid to perform to the best of our ability’, and that is all that I seek to do.* ***[32]*** *In the end, I have concluded that the local authority has not satisfied me that this injury has come about as a result of the culpable conduct of the parties….”)*

**WALKER V HM ADVOCATE 2011 HCJAC 51**

*“48 Cases involving the deaths of infants allegedly at the hands of a parent or other carer are amongst the most difficult, and potentially the most complex, of all cases coming before the criminal courts. In many such cases, and the present is such, there will be no direct evidence of criminal conduct by the accused towards the child. The case will largely, if not exclusively, depend on inferences to be drawn from medical testimony. In this field, while knowledge advances, there remain many uncertainties. Establishing the cause of a sudden infant death may be very difficult and in some cases may not be possible. If criminal liability is to be brought home to the accused it will be necessary to exclude not only any natural explanations for the death suggested in the evidence, but also any realistic possibility of there being an unknown cause. In R v Henderson (a series of cases concerned with “shaken baby syndrome”) Moses LJ said at para 217:*

*“ … a realistic possibility of an unknown cause must not be overlooked. In cases where that possibility is realistic, the jury should be reminded of that possibility. They should be instructed that unless the evidence leads them to exclude any realistic possibility of an unknown cause they cannot convict. In cases where it is relevant to do so, they should be reminded that medical science develops and that which was previously thought unknown may subsequently be recognised and acknowledged. As it was put by Toulson LJ, ‘today's orthodoxy may become tomorrow's outdated learning’ (*[*R v Houldsworth [2008] EWCA Crim 971*](http://login.westlaw.co.uk/maf/wluk/ext/app/document?src=doc&linktype=ref&context=144&crumb-action=replace&docguid=I743CCE8018CC11DDA249804644D629C2) *at [57]). In cases where developing medical science is relevant, the jury should be reminded that special caution is needed where expert opinion evidence is fundamental to the prosecution.”*

**A LOCAL AUTHORITY V T, F 2015 WL 1310691**

*“77 I was struck in this case by the particularly cautious manner in which all the instructed experts expressed their view, but especially Mr Richards in relation to the neurological findings. Mr Brown (on behalf of M) reminded Mr Richards of his evidence three years ago in the case of Re JS [2012] EWCA 1370 (Fam) recorded at paragraph 59 of Baker J's judgment in that case. Mr Richards said: ‘We have enormous gaps in our knowledge. Anything anyone says is informed speculation, not scientifically proven fact, including what I say in the reports.’ Mr Richards confirmed in his evidence before me that this remained his view.”*

**DR CAROL JENNY (2011**) wrote that the “*triad is a myth”.* **GREELEY** has recently written *“the complex features of AHT are often disparagingly distilling simply to “the triad”; a term devoid of any real clinical meaning and not used at all in practice”.*

**COWLEY (2015)** noted *“increasing emphasis on going beyond the simple triad of injuries that is often cited when arriving at a diagnosis of AHT, namely retinal haemorrhage, subdural haemorrhage and encephalopathy”*

**SHAKEN BABY SYNDROME**

1. **IN 1972 CAFFEY PUBLISHED A PAPER ENTITLED “THE PARENT-INFANT TRAUMATIC STRESS SYNDROME; CAFFEY – KEMPE SYNDROME”,** (“Battered baby Syndrome” based on a lecture delivered to the Society for Pediatric Radiology. During the lecture “shaking” was first suggested as a mechanism for abuse. He said “*These deaths and injuries suggest that shaking of the young infants is probably an important cause of serious cerebral and skeletal lesions and of deaths especially when the shakings are repeated frequently”.* CAFFEY went on to suggest that milder *“but still vigorous*“ shaking may be an important cause of mental retardation and cerebral palsies giving examples of common practices such as *“dangling on the knee” “tossing in the air” “slapping on the back after feeding” and “burping”.* All, he felt capable of inducing dangerous acceleration – deceleration forces leading to “*stretchings and compressions”* of the intra cranial blood vessels and the brain.
2. At the tenth annual Abraham Jacobi Award Address published as *“On the Theory and Practice of Shaking Infants”* he introduced the term “*whiplash shaking”* and said that the accumulated evidence suggested that whiplash shaking and jerking of abused infants are common causes of the skeletal as well as cerebro vascular lesions.
3. He felt that the condition could occur in the most banal of situations such as tossing the baby in the air in play. He questioned whether baby bouncers, infant jumpers, seesaws, swings, bicycles and motor cars driven rapidly and habitually over rough roads might cause the condition. Repeated convulsions in tetanus or epilepsy may be associated with whiplash brain damage. He suggested that the baby might be a danger to himself by reason of *“rhythmic whiplash habits of the infant himself during the first months of life such as head rolling, body rocking, and head banging”* which may cause damage to the brain and veins. He concluded that indirect acceleration- deceleration traction of whiplash shaking was the cause of bilateral SDH and frequent bilateral RH with lack of impact to the head and usually no bruises to the face or scalp and no skull fractures.
4. BY 1974 CAFFEY included as the essential elements of the whiplash shaken baby syndrome *“bilateral subdural haematomas and bilateral intraocular haemorrhages”” in the absence of external trauma to the head or fractures of the calvaria (skull cap)”.* He was clear that the presence of this constellation of findings in the absence of external signs of trauma presented an “*extraordinary diagnostic contradiction”.* He described rapid repeated acceleration- deceleration flexions of the head followed by reverse extensions of the head. He thought that the movement was associated with the chin striking the chest and the occiput of the head striking the back. In the same paper he referred to *“latent whiplash shaken infant infant syndrome”* causing mild mental retardation resulting from *“habitual moderate casual manual whiplash shaking”* and opined:

*“The exact frequency violence and pathogenicity of this type of infantile mild assault have never been studied and are not known even approximately. However in view of the high vulnerability of all normal infantile brains to whiplash stresses and the usual repetition of these causal milder shakings over protracted periods it seems reasonable to hypothecate that habitual whiplash shakings are pathogenic to some degree in many such cases. It follows that whiplash shaking may be responsible for repeated small but cumulative intracranial and intraocular bleedings which slowly engender progressive cumulative permanent disorders of the brain and eyes….” “These facts being true it is highly probable that routine examinations of the ocular fundi in all, even apparently healthy babies would detect the residues of retinal haemorrhages and make possible the early stoppage of habitual causal shaking”.*

1. **GEDDES (2001)** undertook a microscopical study of a large series of infants thought to have suffered inflicted brain damage. It was previously thought that irritability, lethargy, loss of consciousness depended on the mechanical shearing of the nerve fibres of the white matter of the brain (diffuse axonal injury). GEDDES found that most babies had brain swelling together with findings indicating deprivation of blood or oxygen supply (hypo ischaemic injury). Only a small minority of babies had axonal injury restricted to the area where the brain is attached to the upper spinal cord. Whereas shearing of nerve fibres results in immediate loss of function, the pace at which swelling occurs varies considerably from case to case taking as much as 48 hours to reach a maximum. This allows for a “*lucid interval*” to occur where a baby may be only mildly symptomatic or even seem quite well.
2. In **2003 GEDDES** in her paper *“Dural haemorrhage in non traumatic infant deaths: does it explain the bleeding in shaken baby syndrome*” described dural bleeding in 50 infants without trauma and proposed that in some infants subdural bleeding may be due to a combination of severe deprivation of oxygen (hypoxia) and raised central venous pressure. The combination would allow blood to leak from blood vessels in the dura causing SDH and from the retinal blood vessels causing RH. These conditions would be exacerbated by additional haemodynamic forces such as increased pressure in the venous system, the effects of sustained increased blood pressure and episodic surges in blood pressure. These are conditions seen in babies who suffer a period of hypoxia and later cardiopulmonary resuscitation and mechanical ventilation.
3. GEDDES suggested that in these circumstances subdural and retinal bleeding were not the result of traumatic rupture of bridging vein but a phenomenon of immaturity.
4. **R V HARRIS 2006 1 CR APP 5** referred to **GEDDES** who (57) *“produced three papers setting out the result of their research into the triad. In the third paper “GEDDES III” the team put forward a new hypothesis “the unified hypothesis” which challenged the supposed infallibility of the triad. It was called the unified hypothesis because it relied on the proposal that there was one unified cause of three intracranial injuries constituting the triad; that cause was not necessarily trauma. It is important to note that the new hypothesis did not seek to show that the triad was inconsistent with NAHI. It did however seek to show that it was not diagnostic. When GEDDES III was published it was and still is very controversial. It is not overstating the position to say that this paper generated fierce debate in the medical profession both nationally and internationally….early on in the hearing it became apparent that substantial parts of the basis of the unified hypothesis could no longer stand. Dr Geddes …accepted that the unified hypothesis was never advanced with a view to being proved in court…it was meant to stimulate debate (para 56-57)…The criticism of GEDDES III is that it is not hypoxia and/or brain swelling which causes sub dural haemorrhages but trauma….As a result of critical papers published in the medical journals…Dr Geddes ....frankly admitted that the unified hypothesis could no longer credibly be put forward. …she accepted that she could no longer support the hypothesis that that brain swelling was the cause of sub dural haemorrhages and retinal haemorrhages. She did however state that she believed that raised intra cranial pressure might prove to be an independent cause of both lesions….she had not (published a paper on this hypothesis) …her research was still incomplete…It was clear that this work was still in its early stages….(68/9)…In our judgment ..the unified hypothesis can no longer be regarded as a credible or alternative cause of the triad of injuries*

The Court set out the salient features of the triad *“in an effort to inform those involved in future trials as to the current accepted state of medical science as we understand it from the evidence before us on some of the very difficult issues which are raised in criminal and civil trials involving allegations of NAHI*” (59). “*Whilst a strong pointer to NAHI on its own we do not think that it is possible to find that (the triad) must automatically and necessarily lead to a diagnosis of NAHI. All the circumstances including the clinical picture must be taken into account…”* (70)

1. The research and debate continue.
2. **(KEMP (2003)** confirmed on the basis of imaging that there was a high incidence of diffuse brain swelling and hypoxic ischaemic damage on brain scans in babies thought to have suffered inflicted injury. **OEHMICHEN (2008)** studied the neuropathology of 18 infants and confirmed GEDDES observation that most of the babies died from failure of cerebral perfusion rather than traumatic brain damage. **VINCHON (2002, 2010)** observed that thin film SDH is likely to be a feature of young age and does not indicate the cause of bleeding. **WHITBY 2004; LOONEY 2007; ROOKS 2008** imaging studies demonstrate that almost half of healthy symptomatic babies have small amounts of subdural bleeding in the same areas as those described in babies thought to have been shaken. **GILES and NELSON 2012** Intradural and subdural bleeding is common in neonates, even those who lack evidence of trauma or overt hypoxia. The fact that intradural bleeding always accompanies SDH in non traumatic cases and if extensive can leak into the subdural compartment (**COHEN and SCHEMBERG 2009; SCHEIMBERG 2013)** is evidence that the dura rather than the torn bridging veins is the source of mild thin film bleeding. **BROWDER 1975; MACK 2009** Blood vessels in the immature dura are larger and more complex at birth than at any other time and **ROOKS 2008; HYMEL 1997; DUHAIME 1998** are particularly extensive in the sites where SDH are most commonly described in both healthy babies and those diagnosed with SBS. Thus **SLOVIS 2012** new research has changed our understanding and SDH can occur from bleeding dural veins and not just from bridging veins. **MATSHES 2010** produced evidence supporting the GEDDES hypothesis with regard to RH by demonstrating that retinal and optic nerve sheath haemorrhages were significantly related to prolonged hypoxia, reperfusion, brain swelling and cardio pulmonary resuscitation).

**BIOMECHANICS AND FORCE**

1. **In 1987 DUHAIME** a neurosurgeonpublished her study of pathological, clinical and biomechanical aspects of SBS. She took as her starting point the clinicopathological entity characterised by RH, subdural and/or subarachnoid haemorrhages and minimal or no signs of external trauma. She acknowledged that a history of shaking was usually absent and that shaking had been assumed on the basis of intra cranial bleeding. She acknowledged that whiplash injury resulted in tearing of the bridging veins leading to subdural and subarachnoid bleeding. She reviewed cases of SBS in the Childrens’ Hospital of Philadelphia over some 7 years with the Department of Bioengineering at the University of Philadelphia. She concluded that the results of the studies relating to impact fell within the ranges for concussion, SDH and axonal injury whilst those relating to shaking fell well below the injury ranges. She concluded that the results were consistent with the observation that all fatal cases of SBS in the series were associated with evidence of impact to the head and *“based on these observations we believe that shaking alone does not produce the shaken baby syndrome”.* She further concluded that SBS *“at least in its most severe form is not usually caused by shaking alone. Although shaking may be part of the process, it is more likely that such infants suffer blunt impact*” and “*unless a child has predisposition factors ….fatal cases of the shaken baby syndrome are not likely to occur from the shaking that occurs during play, feeding or in a swing or even from the more vigorous shaking given by a caretaker as a means of discipline”.* She speculated that *“the most common scenario may be a child who is shaken then thrown against a crib or other surface striking the back of the head and thus undergoing a large brief deceleration*”.
2. **CORY and JONES (2003)** comment that DUHAIME did not take into account any response characteristics of infants in building her models; that the study did not take into account repeated shaking as opposed to a single event or shakings in different directions. CORY and JONES conclude that it cannot be stated categorically from the DUHAIME study that *“pure shaking”* cannot cause fatal injuries in an infant.
3. **CORY and JONES** created models to replicate DUHAIMES biomechanical study. The models had a variety of head, neck and body designs and were shaken to allow impact of chin to chest or occiput to back. All volunteers showed severe fatigue after 10 seconds of violent shaking. The authors suggested that the shaking durations postulated to cause brain injury might be physically impossible to achieve. They selected the thresholds of acceleration at which concussion and SDH would be expected and found that the angular acceleration they generated by shaking in all but one test crossed two of the three thresholds for concussion. None of the tests approached the required threshold for SDH.
4. **JONES (2015)** has since indicated that occiput to back contacts are limited by the neck anatomy. The authors expressed reservations about the models used in the earlier DUHAIME study and concluded that on the basis of DUHAIMEs work it cannot be categorically stated that “*pure shaking”* cannot cause fatal head injuries in an infant. They comment that the DUHAIME modelling thresholds were based on a series of primate experiments which prevented the possibility of the head striking the chest and back during the whiplash motion.
5. **DUHAIME** is criticised in that she relied on injury tolerance levels scaled from adult primates to adult humans and then further scaled from adult humans to infants. There are significant differences in how the human toddler and infant brain respond to injury beyond what would be expected through simple mass scaling. Mass scaling is ineffective within the human species much less between humans and primates (**IBRAHIM 2010**. “Physiological and Pathological Responses to Head Rotations in Toddler Piglets” 27J Neurotrauma 1021, 1022).
6. Traditional biomechanical scaling between infants and older children or adults fails to account for differences in myelination and tissue development and may not reflect the infant brains increased susceptibility to rotational injury (**IBRAHIM and CASE** “Abusive Head Injuries in Infants and Young Children 9 St Louis U Med. Center Forensic Pathology 83-87).
7. **In 1989 BRUCE and ZIMMERMANN** suggested “Shaken Impact Syndrome” although the mechanism of shake and throw were speculative.
8. **PLUNKETT (2001)** in discussion states: “*The extent of injury depends not only on the level and duration of force but also on the specific mechanical and geometric properties of the cranial system under loading. Different parts of the skull and brain have distinct biophysical characteristics and calculating deformation and stress is complex….”* **PLUNKETT** in relation to the same study “*”The brain, scalp and skull of a new born are not the same as the head and neck unit of an adult. Scaling commonly used for the automotive industry may not apply to the neonate or infant. The developmental anatomy and physiology are certainly different but where is the cut off and where is the biophysical evidence to support it? There is only one published study of the biomechanical failure characteristics of an infant skull* (**MARGULIES THIBAULT** *“Infant skull and suture properties: Measurements and implications for biomechanics of Paediatric brain Injuries J Biomech. Engin. 2000 122: 364-371* ). *There are only three published studies of experimentally produced infant skull fractures* (**TAYLOR** “*Marks of Violence on the Head; Fractures of the Skull* Medical Jurisprudence Philadelphia: Blanchard and Lea 1856:364; **WEBER** “*Experimental Studies of skull fractures in Infants”*. Rechtsmed 1984 94, 93-102; **WEBER** *“Biomechanical fragility of the infant skull” Rechtsmed 1985 94 93-101).**There is one published reference concerning the concussion threshold in the paediatric age group. There are no other studies evaluating the failure thresholds or the differential biophysics of infant, toddler, or paediatric injury whether for a 3 month old, 3 year old or 13 year old”.*
9. **OMMAYA (2002)** addressed causation of traumatic brain injury *“with special reference to critical differences in the biomechanical properties and responses of the skulls and brains of infants and brains of infants, children and adults”.* The authors state “*The brain is not a solid component but rather a complex rhetorical substance which is permeated by vascular structures and is contact with cerebro spinal fluid….Due to the vast number of neural elements and current lack of knowledge of their interaction in spite of substantial local in homogeneities and anisotropies the brain has been approximated as a uniform component whose principal properties consist of its density and viscous characteristics subject to its extensive and pervasive vasculature. However recent studies have identified differences in properties of various regions of the brain….The prenatal or infant skull can be regarded as a series of nearly elastic/brittle interlocking plates capable of resisting compression and shear but incapable of transmitting bending across the sutures or fontanelle….the resistance to fracture for an adult is 11 times greater than for a neonate”.* Referring to **WEBER** *“The requisite forces (necessary to fracture) are reduced as a function of the skull thickness and the moduli of the bone. Weber provided experimental confirmation of the presence of skull fractures in each of 15 (average age 8.2 months) perused infant cadavers. Three groups of five cadavers were dropped on stone or tile, carpeted and linoleum covered floor. The floor heights were consistently 82 cm with the cadaver in in a horizontal position with head strike at the parieto occipital zone. This produced an impact velocity of approximately 4 m/s (13 ft/s). Fractures were found in every case….Weber reports (on the basis of falls from parents arms, tables, strollers, beds and sofas) that linear skull fracture does occur in low falls without specific symptoms. He concluded that asymptomatic linear skull fractures can occur and not be recognised by lay caretakers as well as by professional staff…”*
10. **PRANGE (2003)** considered the effects of shaking on the infant neck. He found that shaking generated loads on the infant neck which would be expected to cause neck damage. Support for the damaging effects of whiplash on the infants and child’s neck comes from descriptive reports of injuries seen in infants involved in motor vehicle whiplash injuries where the child has suffered fractures and dislocations of the bones of the neck but subdural and retinal bleeding are not described (**WINTER 2003, FUCHS 1989, JOHNSTON 2004).** The studies (and others such as **BANDAK** which is severely criticised as to methodology) indicate that the loading induced by shaking or whiplash would cause injuries to the infant neck well below those needed to cause retinal and subdural haemorrhage. **MATSHES (2011) and BRENNAN (2006**) suggest that any neck injuries may be subtle.
11. As to the degree of force necessary to cause injury **R V HARRIS**:

*“56 This leads on to a very important issue which arises in these appeals and will no doubt arise in many cases where the triad of injuries are present. It is the question of how much force is necessary to cause those injuries. There is a measure of common ground between the doctors on this issue. Generally it is agreed that there is no scientific method of correlating the amount of force used and the severity of the damage caused. To state the obvious, it is not possible to carry out experiments on living children. Further, experience shows that the human frame reacts differently in different infants to the same degree of force. However the medical opinion on this issue appears to be divided into those who maintain that severe injuries can confidently be ascribed to a traumatic cause, for example (but not only) Dr Rorke-Adams, a very experienced paediatric neuropathologist, and those who maintain that very little force may cause very serious injuries, for example Dr John Plunkett, a distinguished anatomical, clinical and forensic pathologist.*

*57 It is quite impossible for this court to make any finding on this issue beyond referring to some general propositions with which both counsel agreed. First, common sense suggests that the more severe the injuries the more probable they will have been caused by greater force than mere ‘‘rough handling’’. We note that the most recent Update from the Ophthalmology Child Abuse Working Party; Royal College of Ophthalmologists (2004) concludes:*

*‘‘It is highly unlikely that the forces required to produce retinal haemorrhage in a child less than 2 years of age would be generated by a reasonable person during the course of (even rough) play or an attempt to arouse a sleeping or apparently unconscious child.’’*

*58 Secondly, as Mr Peter Richards, a very experienced neurosurgeon with a speciality in paediatrics, pointed out, if rough handling of an infant or something less than rough handling, commonly caused the sort of injuries which resulted in death, the hospitals would be full of such cases. In our view this points to the fact that cases of serious injuries caused by very minor force such as might occur in normal handling or rough handling of an infant, are likely to be rare or even extremely rare.*

*59 But, thirdly, as Dr Plunkett demonstrated by his research and in particular by reference to an amateur video of a child falling from a three foot high railing, described as part of a play tree-house, which resulted in catastrophic injuries, there will be cases where a small degree of force or a minor fall will cause very severe injuries. We shall have more to say about Dr Plunkett’s research later in this judgment, but at this stage we repeat that the evidence suggests that cases where this occurs are likely to be very rare.*

*60 Fourthly, although the younger the infant or child, the more vulnerable it is likely to be, it is not possible to conclude that age is necessarily a factor in deciding whether injuries are caused by strong force or a minimal degree of force or impact. The balance of the evidence is that, although an infant’s skull is more pliable than that of an older child, the internal organs and vessels are as robust as those of an older child. The vulnerability of an infant arises from the fact that its head is generally larger in proportion to its body than in an older child and its neck muscles are weaker and not as well developed as in older children, hence the significance of injuries at the site of the craniocervical junction.”*

1. **RANGARAJAN (2009**) studied the effect of AHT on the eye. There are limitations on the methods because *“model predictions of tissue deformations and stresses are influenced by the geometry, anatomy, tissue material properties, tissue - tissue interactions and applied loads used in the model, one should be aware that inaccuracy of these inputs will have little or no similarity to a real life response”.* “*Because the material properties and tissue interactions of paediatric ocular tissues have not been measured or published it is currently impossible for a finite element model to mimic the response of the paediatric eye. Furthermore it is unknown what values of retinal and ocular stresses produce injury so these models cannot be used to predict the occurrence of retinal haemorrhages or other ocular injuries”* **MARGULIES 2009.**
2. Force is referred to in **HENDERSON 2010 2 CR APP R 24**

*“The issue of necessary force is difficult. Dr Stoodley said that the conventional view was that to produce these symptoms the force required would be such that any reasonable person seeing what was happening would recognise it as dangerous. That view is essentially based on two assumptions: first that it cannot be much more than that as many shaking injuries do not involve other damage eg to the neck or spinal cord; second, that it cannot be much less than that or there would be far more of those injuries than in fact there are. Dr Anslow, for the purposes of this case, did not dissent from that view but asserted than a re-bleed would require lesser force, though how much less was not known, and he was of the view that the mother’s explanation of the buggy ride might indeed account for what had happened. Dr Stoodley accepted that lesser force might be required to trigger a re-bleed (though of course in his view there was no such thing in this case) but he regarded as deeply unlikely the explanation of the buggy ride.”*

1. Force was again referred to in **LONDON BOROUGH OF ISLINGTON V AL ALAS 2012 EWCH 865 (FAM)** a 4 month old baby with skull fracture SDH and RH subsequently found to have rickets due to vitamin D deficiency. Referring to the paediatric radiologist Dr Barnes

“*176 His view was that there is little evidential base or scientific study for radiologists to be able to time fractures in an infant under six months of age, particularly when there is a bone fragility disorder. In relation to the healing process he said there is not a substantial evidence base to be able to provide any kind of parameters other than a general impression that it takes longer, particularly in babies who continue to be handled which may impact on healing. He made clear this view was given from the perspective of imaging examination……*

*Turning to the strength of the bones he agreed that was not possible to predict from x-rays. He said it is known from the scientific literature that rickets not only affects the growth centres but also the shaft of the long bone or shaft of a rib where those can be weak. No one has done the work or the research, either with normal bones in this age group or with abnormal bones to know what the minimum force is that can cause a fracture; such work, he said, was desperately needed in this age group”*

1. **FORENSIC NEUROPATHOLOGY THIRD EDITION JAN LEESTMA (2014)**

The following quotes come from the book which will be available in full shortly.

*“The immature skull is equipped to undergo potentially large deformations associated with vaginal childbirth and thus possess flexible cartilaginous bones and hinge like membranous joints between the bones formed by the periosteum. The mechanical properties of the bones and sutures vary greatly over the first few years of life as the infant brain grows and develops. With growth and development the bones of the skull increase in thickness and differentiate into their sandwich construction of the dense inner and outer tables surrounding the diploe.*

***The parietal bone for example increases in thickness from approximately 1-2 mm at birth to approximately 10 mm at maturity. The prominences of the parietal bones (parietal eminences) are the growth centres from which the bone growth emanates radially forming a mechanical structure whose properties vary according to the anatomical loading direction (loading oriented parallel to the radial fibres versus across the radial fibres). This mechanical behaviour is analogous to the behaviour of corrugated card board, a structure that carries bending loads more effectively along the direction of the corrugations than in the direction across the corrugations…(to be supplied).”***

*“In contrast the sandwich composite into which the bones typically develop at maturity provides a stiff light weight structure that is capable of carrying external eg impact crush loads effectively in bending and shear. The cortical inner and outer tables of the bones provide bending and shear strength to the structure, whereas the diploe core provides space for intracranial channels as well as a lightweight energy absorbing cancellous bone core, much like engineered sandwich structures as in aircraft wing panels, architectural building skins etc. The skull bones are a structural composite that achieves an optimum balance of weight, stiffness and energy absorbing ability.*

***The curved sandwich structure of the skull acts like an architectural dome receiving an external load at a point along the curvature and distributing that load across the bone. The load is carried by the bone to its margins, where the load is shared and transmitted to the other skull bones via the sutures. In the immature skull the membranes are incapable of supporting a bending load and possess little ability to absorb energy (eg from an impact). As the skull matures the joints between the bony plates of the skull begin to achieve their typical interdigitated conformation with the joint between bones possessing a network of collagenous connective tissue. The interdigitation of the bones provides a large surface area over which the joints form and the connective tissue present in the joint in concert with the increased surface area, forms an effective means of absorbing energy transmitted between bones during impact loading of the skull for example”.***

1. **IBRAHIM “PHYSIOLOGICAL AND PATHOLOGICAL RESPONSES TO HEAD ROTATIONS IN TODDLER PIGLETS J NEUROTRAUMA 2010 JUNE 27(6) 1021-1035**

The introduction re myelination and “*Clinical evidence suggests that even within the pediatric population age significantly affects the response of the immature brain to trauma…Overall children less than 4 years of age exhibit worse outcomes compared to older children and adults with head injuries (****KOSKIMIEMI 1995; LUERSSEN 1988)*** *Even among children < 4 years of age we demonstrated differences in head injuries between infants and toddlers after the same event (****IBRAHIM)****. We presented a cohort study of infants and toddlers with accidental head injuries and found that infants sustain significantly more skull fractures after accidents whereas toddlers presented with significantly more neurological signs suggesting that development plays a role in the head injury response of the immature brain”*

**THIBAULT and MARGULIES** 31J Biomechanics 1119. *“The toddler brain can withstand over three times greater strains than the infant before axonal injury results”.*

An infant brain is much less myelinated than an adult brain (**BRENNAN** “Neck Injuries in Young Pediatric Homicide Victims Clinical article 3J Neurosurgery Paediatrics 232-239 (2009)).

Thus infant brains may be more susceptible to injury due to anatomical and chemical differences from toddler and adult brains.

1. **MORITZ “THE PATHOLOGY OF TRAUMA 2ND ED PHILADELPHIA LEA& FEBIGER 1954 REFERRED TO IN KRANIOTI “FORENSIC INVESTIGATION OF CRANIAL INJURIES DIE TO BLUNT FOCE TRAUMA: CURRENT BEST PRACTICE”**

 *“Moritz notes that if the head is free to move with the impact the fractures tend to be linear or completely depressed whereas if the head is immobilised (ie against a solid surface) heavy blows will result in comminuted fractures with inward displacement”*

1. There is no biomechanical evidence of the force exerted by an elbow moving backwards. **PEDOWITZ and JOHNSON** “Practical Orthopaedic Sports medicine and Arthroscopy” analyses the throwing mechanics in baseball and the elbow including the “wind up” “stride” “cocking” “acceleration” and “follow through”. The acceleration phase *“is the shortest phase of the pitching motion but one of the most intense for the elbow and shoulder. This phase begins at maximal shoulder external rotation (the initiation of forward ball motion) and ends at ball release. Sometimes this phase is also divided into early and late phases. The early acceleration phase is the first 25% where the greatest forces are generated, the initiation of forward motion ball and the last 75% the late acceleration phase….”*

**REBLEEDS**

1. It is recognised that birth related bleeding may persist and develop into a chronic collection around the brain which may later present with symptoms. Old subdural bleeding is the most common birth related pathology identified at routine post mortems of SIDS victims **ROGERS 1998; KEELING 2009.**
2. Spontaneous fresh SDH can be seen in the context of chronic extra cerebral collections possibly triggered by illness and dehydration **VINCHON 2010**
3. Small fresh bleeds seen after collapse and resuscitation are often assumed to be the cause of collapse but may be the result of collapse, the bleeding coming from the large delicate vessels in the membrane as the result of hypoxia and the intra vascular changes during resuscitation.
4. Reference is made to **HYMEL** “*Intra cranial haemorrhage and Rebleeding in Suspected Victims of Abusive head Trauma; Addressing the Forensic Controversies“* 7 Child Maltreatment 329-248 **2002** to support the proposition that a traumatic SDH may rebleed. In the study both children were over 11 months of age. One fell down several concrete stairs and suffered a fractured skull and epidural haematoma. Three months later he hit his head on a window sill and suffered a rebleed. Because the second trauma was minor, symptoms were limited to irritability and loss of appetite. Thus a SDH may rebleed but if new symptoms are seen there must have been a significant new brain injury.
5. Subdural bleeds in new borns are small and lie primarily in the posterior fossa (back part of the brain) and resolve without rebleeding or becoming symptomatic (**ROOKS 2008).** Rooks concluded that haemorrhages seen in asymptomatic neonates are limited in size and duration. SDH after one month of age is unlikely to be birth related.

**LOW FALLS AND “LUCID INTERVAL”**

1. The potential for low falls to cause severe intra cranial injury or death was recognised by **CAFFEY** and is supported by other studies and debated in others **(PLUNKETT 2001; STEINBOK 2007; VAN EE 2009; LANTZ and COUTURE 2011; CHADWICK 1991; WILLIAMS 1991; RUDDICK 2010; HELFER SLOVIS BLACK 1977** “*Injuries Resulting when Small Children Fall out of Bed* “Pediatrics 60: 533-535; **LYONS and OATES** *“Falling out of bed: A Relatively Benign Occurrence”* 92 Pediatrics 125-127. Among 85 children who had fallen out of bed, crib or examination table no injury (57), small cuts (17), bump or bruise (20), one skull fracture with no apparent sequelae. In **LANTZ and COUTURE** reference is made to a review by HALL of 18 children who died from accidental falls of less than 0.9 m (8 witnessed by two or more people in public places with 5 linear skull fractures. Falls consisted of two dropped on ice, 5 while playing, 8 off an object and three down steps.
2. In **R V SHAKEEN CLARKE 2006 EWCA CRIM 2667** para 31 “*Nobody challenges that there is now some evidence in the literature to the effect that significant injury can be caused by low level falls”*
3. **GREENES SCHUTZMAN “***Occult intracranial injury in infants” Am Emerg Med 1998 32: 680*referred to in OMMAYA at page 230 “*Greenes & Scutz have recently reported a retrospective review of 101 infants less than two years old who were admitted for suspected TBI to a tertiary care paediatric hospital over the course of 6.5 years. 19 of these infants all less than 1 year old were asymptomatic but harbouring occult intracranial injuries. They had no loss of consciousness, no mental or behavioural changes, no seizures, irritability or vomiting , without retinal haemorrhages or a bulging fontanelle and with normal neurological examination....all were alert and five were playful.....18 had skull fractures..."*
4. **HUNTINGTON 2002** describes a 13 month old Hispanic girl admitted in the morning complaining of vomiting for 24 hours, irritable and sleepy. Described as “*fussy and clingy*” by the doctor. Deterioration the following morning when decreased respiration and unresponsive. Died. At post mortem severe intra cranial injury although no skull fracture.
5. The single case study documented by **SCOTT DENTON 2003** *“Delayed Sudden Death in an Infant Following an Accidental Fall*” 24 Am J Forensic Med pathology 371 concerns a nine month old child reported by the grandmother to have fallen off a bed, acted normally for the next several days and was then found dead with a posterior parietal skull fracture and SDH *“He immediately began crying and the grandmother placed ice on a knot on the back of his head. He stopped crying and was consolable within a few minutes. The child was taken to the babysitters residence…When the mother picked the child up at the babysitters in the afternoon (the fall having occurred in the morning) he appeared well. The baby sitter reported no problems and that he acted, ate and behaved as usual. For the next two days the grandmother, mother and baby sitter did not notice any abnormalities in either behaviour or appearance of the child”*. Symptoms set in 72 hours later. At PM he was found to have a linear non displaced posterior right parietal skull fracture…”
6. **WILLIAM KRISTA (1997)** 95 cases of fatal head injury in children found only two lucid interval cases involving epidural and not SDH.

**STARLING 2004** suggest that in most case of confessed AHT injuries there is no significant lucid interval. Lucid intervals are associated with epidural haematomas.

**GILLIAND 1998** studies the time between the initial injury and the appearance of severe symptoms defined as *“the time when an external event occurred or the caretaker called for medical assistance*”. The symptoms are unresponsiveness, difficulty or cardiovascular collapse. Where an independent observer witnessed the incident the child was immediately symptomatic. “*It remains unclear if fatal head injuries in young children are characterised by immediate rapid deterioration or can present after an initial period of lucidity”* (referring to inter alia WILLIAM, PLUNKETT, DENTON)

**PLUNKETT (2001)** did not involve infants. The falls were between 0.6 to 3 metres (2-10 feet). 12 of the 18 children experience lucid intervals of between 5 minutes and 48 hours. No child was under the age of one year. All lucid intervals were less than 15 minutes for the children under 4 years. One 3 year old fell 0.6metres/2 feet with a lucid interval of 15 minutes and complex calvarial fracture, contusions, cerebral oedema with herniation.

See also the Court of Appeal in **R V HARRIS 2006 paras 196-202**

The **ARBOGAST** study (2005) assessed children using the Glasgow Coma scale. Two per cent were above 12 which was determined to be *“lucid*”. The GCS is based on motor and verbal skills and cannot be used for children under 36 months. The GCS does not address vomiting, irritability and a score above 12 does not imply that the children were completely asymptomatic.