Background
This meeting was convened by The Royal College of Pathologists, following a request from the Lay Advisory Committee of the College, in recognition that there had been disagreement between Fellows of the College on the appropriate interpretation of various aspects of the post-mortem findings in the cases of traumatic head injury in children (so-called ‘Shaken Baby Syndrome’).

The meeting was convened as a closed, invitation-only meeting, limited to pathologists, with the specific intention of discussing the pathological interpretation of relevant post-mortem findings rather than discussing wider areas of radiology, clinical paediatrics, child protection or legal aspects of the problem. The meeting did not attempt an in-depth or exhaustive discussion of all the relevant post-mortem features; nor did those present attempt to debate the possible patho-physiological mechanisms.

This report of the meeting was written by Professor Peter Furness, who is a histopathologist and is currently President of the RCPath but who has no special expertise in paediatric pathology, neuropathology, ophthalmic pathology or forensic pathology. The reason for this is set out in his Personal Comment at the end of the report. Three sequential draft versions of the report were circulated, by email to all the participants in the meeting, over December 2009 and January 2010; a number of amendments to the report were made during this process, and no dissent to the text of this final version was received during the week after its circulation on Wednesday 27th January 2010. However, not every participant responded to the requests for comment on the draft report so it is acknowledged that positive confirmation of the contents of this report have not been received from all the participants.
Attendees:

Prof. Peter Furness (Chair)
Dr Safa Al-Sarraj
Dr Richard Bonshek
Dr Nat Cary
Dr Marta Cohen
Dr Phil Cox
Prof. Jack Crane
Dr Daniel Duplessis
Mr Neil Formstone (Chair, RCPath Lay Advisory Committee)
Dr Allan Howatson
Dr Paul Johnson
Prof. Phil Luthert
Dr John McCarthy
Dr Chris Milroy
Prof. Michael Pollanen
Prof Tony Risdon
Dr Irene Scheimberg
Dr Colin Smith
Dr Waney Squier
Prof. Helen Whitwell

Introduction and Welcome, Aims of the Day

Professor Peter Furness (President of the College) welcomed the meeting participants. He acknowledged the fact that the subject had previously generated heated arguments. He set out the intention that the meeting should probe scientific aspects of the interpretation of the pathology and should therefore be conducted in a collegiate spirit of scientific investigation, putting aside the adversarial processes that might be more appropriate in other circumstances.

Ocular changes

A joint presentation from John McCarthy, Phil Luthert and Richard Bonshek was presented principally by John McCarthy.

Dr McCarthy explained that the three of them were of a fairly uniform opinion about the interpretation of ophthalmic changes in these circumstances.

He stressed that the age of the deceased is important in the interpretation of post-mortem findings.
**Retinal Folds**
Small folds can represent a post mortem artefact or fixation artefact. True retinal detachment is rare, but the retina can be lifted up by a sub-retinal bleed; it is debatable whether this should be regarded as a true detachment or not, but the term is widely used in the literature.

A perimacular retinal fold has a clear association with pre-mortem head trauma. Such a fold might be explained on the basis of local adherence of the vitreous to the macula, or it could be a consequence of retinal haemorrhage. Evaluation of the reliability of this feature is limited by limited experience of non-trauma post-mortem eyes, as they are very rarely examined in detail; but there is considerable clinical evidence from the living in support of this being a significant feature in relation to trauma. The larger/more severe the fold, the more reliable it would be as evidence of trauma. Such folds are normally seen in association with retinal haemorrhage; one should be suspicious of interpreting a fold that is not accompanied by haemorrhage.

**Haemorrhage**
Retinal haemorrhage may vary in its distribution around the globe, in its extent and in the layer of the retina in which the haemorrhage occurs. There are numerous non-traumatic causes of retinal haemorrhage, including meningitis and coagulation disorders, so it is essential to undertake a good post-mortem examination before attempting interpretation. It is known that birth injury can cause retinal haemorrhage. There is a problem in identifying a suitable control group for post-mortem examination, because the eye is not normally examined histologically unless there is a strong suspicion. This problem is mitigated to some extent by studies of eyes in living children, such as an ongoing (Great Ormond Street) study of the eyes of all children admitted to the intensive therapy unit. It is hoped that this will provide more reliable information around the circumstances under which retinal haemorrhages appear, though children in ITU obviously do not represent an ideal control group for normal children.

It is suspected that haemorrhages may continue to develop after the causal insult has ceased, but for how long is unknown.

**Optic Nerve Sheath Haemorrhage**
This relates to haemorrhage around the optic nerve, beneath and within the optic nerve sheath dura. This may occur at any point, but is most usually seen at the point of its attachment to the globe. This lesion can happen with or without associated retinal haemorrhage. The mechanism is not entirely understood, and the micro-anatomy of the central retinal vein anastomotic supply at this site is not well documented. Interpretation is again limited by the absence of appropriate controls where there is no suspicion of trauma, but there is nevertheless a firm belief of a strong association with head trauma. It is usually bilateral in cases of head trauma; unilateral optic nerve haemorrhage should be regarded as less reliable.
**Haemosiderin**

The presence of haemosiderin is usually indicative of old haemorrhage, though not invariably, because it is believed that it can sometimes mark the location of previous extramedullary haematopoiesis, or birth-related bleeding. Identifying its age is difficult; it can form within two days after haemorrhage, and the duration of its persistence is unclear, but persistence for months or years is possible. Haemosiderin deposits can presumably reflect old birth related trauma, so its presence alone is not a reliable marker of subsequent trauma.

**Subdural Haemorrhage**

Dr Waney Squier provided a detailed description of the anatomy of the dura and the venous plexuses around the intra-cerebral sinuses, stressing the considerable changes that occur in the first few months of life. She also discussed the circulation and resorption of the cerebrospinal fluid. She explained that a network of spongy ‘holes’ in tissue around the venous sinuses developed largely after three to six months, whereas intra-dural bleeding is commoner before six months. In such young children she believed that that intra-dural bleeding can be due to hypoxia plus other factors including increased intracranial or intravenous pressure; the age of the child is crucial to the interpretation. On radiology, it can be difficult to distinguish between blood in the venous plexuses adjacent to the cerebral sinuses and intradural haemorrhage.

The question of whether intradural haemorrhage can lead to subdural haemorrhage was discussed. It seemed likely that this mechanism can result in at least small subdural haemorrhages but radiology is not a reliable means to identify the distinction between intradural and subdural bleeding.

**Dr Safa Al-Sarraj** described his personal detailed analysis of 111 cases, divided on the basis of information prior to post-mortem examination into four groups:-

1. Hypoxia – 39 cases
2. Non accidental head injury with other associated injuries – 40 cases
3. Accidental head injury – 5 cases
4. Suspected non accidental head injury with no other injuries – 28 cases

(Note: These groups of cases, numbered 1 – 4, also feature in the subsequent presentations from Dr Al-Sarraj described below).

The age range was from birth to three years.
In relation to subdural haemorrhage, in Group 1 only one child had a small recent subdural haemorrhage; that child had suffered septicaemia. However, 75% had recent intradural haemorrhage, identified principally by microscopy. A few of these cases also had focal and unilateral retinal haemorrhage.

In Group 2, 27% of cases were associated with skull fracture. 97% had a recent subdural haemorrhage, the majority of which were small (less than 40ml). 66% had subdural haemorrhage in the spinal cord.

Group 3 (only 5 cases), 80% were associated with skull fracture, subdural haematoma was present in all cases (100%) and 40% of cases with subdural haematoma in the spinal cord. The interpretation of the haemorrhage in this group is not reliable because of the low number of cases.

Group 4: 100% associated with subdural haematoma. The majority are small (92.3%). In 30% of the cases there was subdural haematoma in the spinal cord.

Dr S. Sarraj suggested that statistically, Group 4 resembled Groups 2 and 3 in respect of subdural haemorrhage and they did not resemble Group 1. However, this does not preclude the possibility of individual unusual cases. It was suggested that age under 6-9 months was important and Dr Al Sarraj said he could provide this information for all groups.

Dr Irene Scheimberg provided illustrations of haemorrhage around the brain in numerous cases where the cause of death was clear and was not regarded as traumatic. Causes included placental abruption, intra-partum asphyxia, drowning, lung hypoplasia and choking on food by a baby which had been born prematurely. An agonal episode of hypoxia was common to all cases.

The majority of these cases were deaths in the first few days of life; it was agreed that the group represented a younger cohort of babies than those discussed by Dr Sarraj.

Dr Scheimberg presented the cases as evidence that mechanisms other than mechanical trauma, probably largely hypoxia in conjunction with other factors (increased ICP, infection, etc), can produce subdural haemorrhage, as all these cases had a history of pre-mortem hypoxia. It was pointed out in discussion that in this age group small subdural haemorrhages might be a relatively common (and usually unnoted) consequence of the trauma of birth, even normal birth, and that most deaths in this age group involve a period of agonal hypoxia. Dr Scheimberg countered the argument by pointing out that some of the babies had been delivered by caesarean section; others argued that emergency caesarean sections could still inflict mechanical trauma to the head of the baby. Dr Scheimberg acknowledged that SDH is seen in a proportion of babies, but not all traumatic births have SDH. The discussion was unable to generate agreement on whether the haemorrhages illustrated could reliably be interpreted as being due to hypoxia or not.
**Dr Marta Cohen** discussed the anatomy of the bridging veins, suggesting that one might expect a tear in a vein to cause subarachnoid rather than subdural haemorrhage, arguing that the subdural haemorrhages seen in cases of suspected traumatic head injury arose largely from bleeding from intradural vessels. She argued that subdural haemorrhage is commonly associated with diffuse intradural haemorrhage in very young children, arguing that the subdural haemorrhage could result from intradural haemorrhage in this group. She discussed the difficulties of radiological interpretation of these cases in view of the distribution of the venous plexuses within the skull. She suggested that intradural and subdural haemorrhage were common in babies under one week of age although she presented a few cases where children were above this age range (including 8 months, 24 months and up to 32 months). She presented post mortem MRI correlation and suggested that the MRI cannot identify with certainty the presence of an intradural haemorrhage (MRI is unable to say if the haemorrhage is intradural or adjacent to the dura). The possible mechanisms were discussed and Dr Cohen suggested that such intradural haemorrhage, possibly with subdural extension, could probably persist for a few months.

**Discussion**

The subsequent discussion of the interpretation of subdural haemorrhage considered its extent and the age of the child. It was agreed that in very young children, subdural haemorrhage must be interpreted with considerable caution because (whether due to hypoxia or direct mechanical trauma) it could be due to damage during birth. Deaths in very young children usually involve hypoxic/ischaemic changes, whatever the underlying cause of death, which made interpretation of the mechanisms very difficult. In older children it was agreed that macroscopically evident (thin film) subdural haemorrhage was considerably more suggestive of traumatic head injury, whereas microscopic haemorrhage, especially haemorrhage within the dura, was less impressive.

The discussion highlighted the fact that different pathologists present had considerably different experiences, both on the basis of the history of cases referred to them and also in relation to the age profile of those cases. This made the generation of valid comparisons and agreement somewhat difficult.

The meeting could not produce agreement on whether the source of the bleeding was the venous plexuses or bridging veins, though the majority of those present seemed to regard both sites as being possible. It was explained that the mechanism of haemorrhage could be important to courts where the involvement of trauma was accepted, but the court had to make a decision between charges of deliberate murder or manslaughter. However, in the context of deciding whether haemorrhage was due to trauma or some other pathological process,
understanding the mechanisms was probably less important than good documentation of associations in cases where the pathogenesis was known.

**Encephalopathy**

Colin Smith started the session by stating his belief that ischaemia causes encephalopathy, a point on which there was no dissent, although possible methods by which traumatic damage and hypoxic damage might be distinguished were later discussed by Dr Sarraj (below).

Marta Cohen discussed causes of acute collapse in young children. These are numerous and include cardiac dysrhythmias, some of them due to inherited abnormalities of ion channels, infections, obstructions to the upper respiratory tract, and possible genetic causes of an abnormal response to hypoxia.

She suggested that such causes of sudden death could result in agonal hypoxia and thereby spontaneously cause changes such as intradural/subdural haemorrhage, thereby mimicking traumatic head injury. All agreed the need for a thorough post mortem examination to exclude other causes of death before attempting to interpret intracerebral changes that might suggest traumatic head injury. Interpretation was made difficult by the observation that subdural haemorrhage could be caused by birth trauma.

Dr Safa Al-Sarraj discussed his four groups of cases described above, this time in relation to histological evidence of ischaemic injury or traumatic axonal damage (the latter defined by the histology supplemented by beta-APP immunohistochemistry).

Group 1 – all showed ischaemic damage with ischaemic axonal injury but no evidence of traumatic axonal injury. There was no haemorrhage.

Group 2: 37/40 cases (92.5%) showed ischaemic injury. 1/40 cases showed diffuse axonal injury (2.5%) and 12/40 cases showed focal traumatic axonal injury (30%). 12/40 cases showed traumatic axonal damage; 4/40 showed focal intracerebral haemorrhage (10%). Intraventricular haemorrhage was present in 3/40 cases (7.5%).

Group 3: 3/5 showed ischaemic injury, 2/5 showed traumatic axonal injury, 2/5 showed focal haemorrhage and 2/5 with intraventricular bleeding.

Group 4 – the suspected non accidental head injury, 96% showed ischaemic injury. 28% were reported to show multifocal traumatic axonal damage, but as this was not widespread and was difficult to interpret histologically, the finding was not accepted uncritically by other pathologists present. 3% showed small intracerebral haemorrhages.

Dr Al-Sarraj provided the following graph to illustrate these findings:
Dr Al-Sarraj went on to discuss damage to the medulla and nerve roots in the same set of cases.

Group 1 – No medullary or nerve root injury was found.

Group 2 – 35% showed traumatic injury into the corticospinal tracts in the medulla. 17% showed ischaemic injury. In the nerve roots, 42% showed axonal injury.

Group 3 – In the medulla 40% showed traumatic nerve injury and 80% show ischaemic injury. In the nerve roots axonal injury was present in 60%.

Group 4 - In the cases of suspected traumatic head injury without other injuries, 20% showed traumatic axonal injury in the medulla, 16% showed ischaemic pattern injury in the medulla. In the nerve roots 39% showed axonal injury.

Dr Al-Sarraj suggested that on this basis, the Group 4 statistically resembled Groups 2 and 3 far more than Group 1. However, it was clear that on an individual basis a significant number of babies in Group 4 had no injury detected.

Dr Sarraj explained that the nerve injury in the medulla was often patchy and could be difficult to find, but the intriguing possibility was raised that such injury, given its location, could induce apnoea.

It was agreed that extending these observations to other cases was as yet difficult, because spinal cords are not routinely examined in this way in non-trauma deaths.
Others present indicated that they regarded the histological distinction between traumatic and ischaemic nerve injury to be difficult to make and probably unreliable, even with the assistance of beta-APP immunohistochemistry. It was also pointed out that 70% did not have evidence of traumatic injury.

**Other pathological features of possible relevance to SBS**

Dr Waney Squier discussed subcortical damage after severe cerebral hypoxia/ischaemia with associated brain swelling and/or suspected traumatic injury. She illustrated this with subcortical changes after ischaemia due to severe pneumonia, cardiac arrest and seizure. Various terms had been used, including subcortical contusions, gliding contusions and subcortical tears. The evidence presented suggested that these changes were not necessarily attributable to trauma, as had been indicated by other authors, but that severe brain swelling could be responsible.

**Experiences from outside the UK**

Dr Michael Pollanen presented a retrospective review of cases from Ontario, around the ‘Goudge Inquiry’. This Inquiry reviewed possible miscarriages of justice and suggested review of certain cases where individuals had been convicted of causing damage to children.

The review was restricted in cases where conviction was based essentially on the presence of ‘the Triad’ of retinal haemorrhage, subdural haemorrhage and encephalopathy. Cases were subdivided into three groups, with more than ten cases in each category:

- **Group 1** had only the triad, with no other acute injuries.
- **Group 2** had the triad plus some head injury, but no extra cranial injuries.
- **Group 3** had multiple injuries.

Analysis of the circumstances around these three groups were described. Groups 1 and 2 presented at a similar age, with Group 3 tending to be slightly older. Study of the history and circumstances around cases in these three groups showed that admission of shaking the child or admission of head impact had little value, paradoxically being least frequent in cases where multiple injuries were present. A history of sudden death with no preceding events, or merely a history of non-specific distress in the child, was of similar frequency in the three groups. However, the numbers were relatively small, and statistical tests of trends or differences had not been undertaken.
Putting it Together

Dr Chris Milroy initiated a discussion by stressing that pathological evidence does not in isolation have to be ‘beyond reasonable doubt’ to secure conviction, as the court is charged with considering evidence from a number of sources. Expert witnesses had a duty to discuss possible contrary opinions and other interpretations of the findings, and should not be persuaded to express their opinions as certain unless there was no reasonable doubt.

Professor Furness attempted to ascertain the level of divergence of opinion on the probability of there being traumatic head injury as the underlying cause in cases where all three elements of the Triad were present in characteristic form, but with no other evidence of injury. There was widespread reluctance to participate in such an exercise, on the grounds that interpretation should invariably be influenced by other factors of the case. For example, all agreed that if the baby was one week of age then the possibility of such changes being a consequence of birth injury meant that they should be interpreted with far greater caution than in a child who was several months of age. It was agreed that for each individual element of the triad there is a differential diagnosis. Interpretation therefore depends on the elimination of as many of those possible diagnoses as possible. If that exercise is undertaken thoroughly, and all three elements are present in characteristic form, then the combination would generally be regarded as highly suspicious of traumatic head injury, if birth associated trauma could be excluded with reasonable confidence.
Conclusion

Areas of agreement and disagreement
The following list has been agreed by the participants at the meeting

**It was agreed** that when the following features are all present at a paediatric post mortem:

- widespread bilateral retinal haemorrhages and large macular folds
- thin-film subdural haemorrhage
- encephalopathy

(i.e. ‘the triad’ in characteristic form)

then, considering the case in the absence of other evidence, there should be a *prima facie* suspicion that the injuries are due to mechanical trauma, potentially including vigorous shaking.

**It was agreed** that all the individual elements of ‘the triad’ have a differential diagnosis, and that a thorough post-mortem examination is invariably needed to exclude, as far as possible, non-traumatic explanations of such changes.

**It was agreed** that in the current state of knowledge the presence of ‘the triad’, even in its ‘characteristic’ form, should not be regarded as absolute proof of traumatic head injury in the absence of any other corroborative evidence.

**It was agreed** that the following post-mortem findings would **lend support** to a suggestion of mechanical trauma to the head:

- Bilateral optic nerve haemorrhage at the point where the optic nerve enters the globe
- Histological changes in the brain indicating mechanical damage rather than ischaemic damage (though **there was not agreement** on how easy or reliable such histological assessment may be)
- Other post-mortem evidence of cranial trauma or extra-cranial trauma

**It was agreed** that the following post-mortem findings would **indicate a need for greater caution** in suggesting that there had been significant mechanical trauma to the head:

- One or more elements of ‘the triad’ being absent
- One or more elements of ‘the triad’ being present in a limited or atypical form (e.g. intradural haemorrhage without subdural haemorrhage; large, space-occupying or organising subdural haemorrhage; unilateral ocular changes)
- Young age at death (interpreted as under 3 months, with particular caution being needed in cases young enough for birth trauma or hypoxia to be a possible explanation for the post-mortem findings)

**It was agreed** that in some cases where death is undoubtedly due to head injury, some or all of the components of ‘the triad’ may be absent.
It was not agreed how the post-mortem findings should be integrated to suggest a level of probability of mechanical head injury for the benefit of a court, when considering an individual case. It is therefore anticipated that even where there is agreement on the description of the findings at post-mortem, pathologists could not be expected to agree on the precise probability of trauma being the underlying cause.

The meeting did not discuss mechanisms by which the changes of ‘the triad’ might develop, so areas of agreement and disagreement concerning mechanisms cannot be stated.

Suggested future steps
The meeting had not achieved complete consensus and it had only considered the pathological interpretation, excluding many other areas of relevance to these cases. Possible next steps were briefly discussed.

A further meeting?
There was agreement that it would be beneficial to follow up this closed, pathology-only meeting with a larger, open meeting that involved specialists in other relevant fields, including radiology, paediatrics, child protection, lawyers, experts in biomechanics etc. However, Professor Furness (as President of the RCPPath) stressed that although the current meeting had been funded by the College any further meeting would be dependent on a different source of funding becoming available.

Research to resolve disagreements?
There was consensus that the only way in which the disagreements discussed at the meeting would be resolved was by undertaking systematic research. This would demand large studies of paediatric deaths in a variety of circumstances spanning the areas of practice of all those present at the meeting; it would therefore demand collaboration between different units, and it would demand substantial funding.

Those present, including those whose opinions currently differ considerably on the interpretation of post-mortem changes, agreed that they would all be willing to collaborate in such a project.

However, barriers to such a research project were discussed.

The main barriers were not perceived as interpersonal disagreements between pathologists, nor even the need to secure funding (though that would not be easy).

The main problem is with the regulatory environment, especially with the regulatory changes that had been put in place since the inappropriate unconsented use of paediatric post-mortem samples at Alder Hey and Bristol prior to 2000. It was reported that even for observational studies, research ethics committees and NHS R&D staff were often extremely difficult to satisfy.
before material could be published. Confidentiality requirements were often raised by regulators, with conventional anonymisation processes being regarded as insufficient for these unusual and high-profile cases, even (or especially) after the details of the case had been discussed in court. The difficulty of obtaining appropriate control material was discussed; while the paediatric brain would be examined at most paediatric post-mortem examinations, removal and detailed examination of the eyes would be a research-specific process for which specific consent would be necessary. It was anticipated that it would be difficult for staff to ask for, or for the bereaved parents to provide, such consent.

There was agreement that poor drafting of the Human Tissue Act 2004 had made the difficulty of conducting this research considerably more severe. It was pointed out that to study tissue samples for research in these cases, appropriate consent was invariably needed. But consent had to be provided by a parent, so in many cases consent had to be requested from a person who stood accused or convicted of murder. Putting aside the practical difficulty of obtaining access to such a person to request consent, the circumstances would effectively guarantee that, from a scientific perspective, any group of cases where consent was obtained would have to be regarded as a biased sample.

Several representations had been made to the Government to the effect that this was not an intended outcome of the Human Tissue Act 2004 and that the law therefore needed to be changed. These representations notably included the Joint Parliamentary Select Committee on the Human Tissue and Embryos (Draft) Bill in 2009 and amendments to the Coroners and Justice Bill 2009, proposed by Baroness Finlay in the House of Lords. Others, including the Coroners’ Society, have also recommended that the 2004 Act be changed. All such approaches had been rebutted by the Government, apparently without giving any justification beyond the observation that Parliament had already debated the Human Tissue Act prior to its passage in 2004.

The Human Tissue Authority had acknowledged the problem in undertaking research in this context but had refused to recommend any change to the legislation, again without giving reason.

Those present deplored this attitude of the Government and the Human Tissue Authority.
Personal comment by Professor Peter Furness, President of the Royal College of Pathologists

I convened and Chaired this meeting despite having no specialist expertise in the area, because I was aware that Fellows of the College of which I am President were expressing different opinions, in and out of the context of Court hearings. I was informed that this was producing problems for the courts as well as generating a regrettable level of discord amongst Fellows of the RCPPath.

I did not expect the meeting to produce complete agreement and it did not. However, it was held in a considerably more cordial atmosphere than I had been led to expect. This led me to suspect that some of the antagonism that had developed between participants had been exacerbated by their previous experiences in the adversarial context of court hearings.

Another factor that I believe contributed to the level of disagreement is the very different types of practice of the pathologists who were present. Some see only forensic cases, the majority being deaths at more than 3 months of age; others see a caseload predominantly of deaths in hospital, predominantly less than 3 months of age. This results in practitioners having radically different personal experiences, and it is natural that over many years of such experience they form firm but different opinions on the appropriate interpretation of the post-mortem findings.

I attempted to listen to the debate with an open mind. If I had any preconceived suspicions, it was that a cohort of very experienced forensic practitioners were being challenged in their interpretation by new evidence and they were reluctant to admit that the opinions they had expressed in court over many years might be incorrect. After the meeting I was convinced that this is not an accurate representation of the true position.

I left the meeting convinced that where ‘the triad’ is present in typical form in an infant of 3 months or older it represents strong evidence of mechanical injury, consistent with ‘shaken baby syndrome’. However, I was reassured that no-one present regarded such a finding as absolute proof. It seemed to me that in all such cases a court should seek some further corroborative evidence.

I was also convinced that there are circumstances where all the individual elements of ‘the triad’ can be produced by insults other than mechanical trauma. For example, it seems to me probable that the natural events around a ‘difficult’ birth may induce these lesions. It follows from this that all three might be present in a case where intentional mechanical trauma is not the underlying cause.

Consequently, in cases where the dead child is very young, or where one or more elements of ‘the triad’ are absent or are present in some atypical form, the need for other corroborative
evidence is proportionately greater before a court should decide that the evidence justifies a
decision ‘beyond reasonable doubt’. Pathologists cannot be expected to agree on precise
probabilities in this situation. This problem can only be alleviated by research, not by further
discussion. Unfortunately the barriers to undertaking such research are considerable, and have
become much greater in recent years.

The meeting did not consider mechanisms by which shaking might generate the post-mortem
changes that were discussed. This is an area of considerable debate, and some of those present
at the meeting regarded this omission as a serious defect in our meeting. I was less concerned
by this omission, observing that the absence of a known mechanism does not prove that a
mechanism does not exist. At risk of trivialising the matter I drew the attention of those
present to reports some years ago that an aerodynamic study of bumble bees ‘proved’ beyond
scientific doubt that they cannot fly. Personal observation is, of course, to the contrary. A
more accurate statement would have been that current aerodynamic theory was unable to
explain how they fly. Subsequently, more refined scientific theories took account of the fact
that, for structures as small as a bee, air has a significant viscosity, and the flight of the bumble
bee now has a scientific explanation.

When the science improved, the explanation became apparent. Until that happened, the
observation that bees can fly showed the scientific theory to be inadequate. The same might be
ture of the problems in explaining precisely how vigorous shaking can generate the lesions we
have been discussing.