

IN THE HIGH COURT OF JUSTICE
COURT OF APPEAL (CRIMINAL DIVISION)
ON APPEAL FROM READING CROWN COURT
The Hon. Mr Justice Keith
T20067156

Royal Courts of Justice
Strand, London, WC2A 2LL

Date: 17/06/2010

Before:

LORD JUSTICE MOSES
MRS JUSTICE RAFFERTY
and
MR JUSTICE HEDLEY

Between:

Keran Louise Henderson
- and -
The Crown

Appellant

Respondent

Mr M Topolski QC and Mr A Scott (instructed by William Bache & Co) for the
Appellant
Miss J Glynn QC and Miss S Campbell (instructed by the Crown Prosecution Service)
for the Respondent

Hearing dates: 3rd-5th March 2010

ON APPEAL FROM CROYDON CROWN COURT
His Honour Judge TMF Stow QC
T20077598

Royal Courts of Justice
Strand, London, WC2A 2LL

Date: 17/06/2010

Before:

LORD JUSTICE MOSES
MRS JUSTICE RAFFERTY
and
MR JUSTICE HEDLEY

Between:

Ben Butler
- and -
The Crown

Appellant

Respondent

Miss S Wass QC and Miss K Thorne (instructed by Mark Williams Associates) for the
Appellant
Mr E Brown QC (who did not appear below) and Miss N Tahta (instructed by the Crown
Prosecution Service) for the Respondent

Hearing dates: 10th-11th March 2010

IN THE HIGH COURT OF JUSTICE
COURT OF APPEAL (CRIMINAL DIVISION)
ON APPEAL FROM THE CENTRAL CRIMINAL COURT
His Honour Judge Focke QC
T20057588

Royal Courts of Justice
Strand, London, WC2A 2LL

Date: 17/06/2010

Before:

LORD JUSTICE MOSES
MRS JUSTICE RAFFERTY
and
MRS JUSTICE SHARP

Between:

Oladapo Oyediran
- and -
The Crown

Appellant

Respondent

Mr N P Valios QC and Miss K Arden (instructed by **Mackesys Solicitors**) for the
Appellant
Miss S M Howes QC and Mr B P J Kelleher (instructed by the **Crown Prosecution**
Service) for the **Respondent**

Hearing dates: 24th-25th March 2010

Judgment Approved by the court
for handing down
(subject to editorial corrections)

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Lord Justice Moses :

Introduction

1. 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] 2 Cr App R 63, 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* [177] teaches, even where on examination of all the evidence, every possible known cause has been excluded, the cause may still remain unknown.

2. This court has heard, over a period of three weeks, three appeals concerning three babies, two of whom died, whilst in the care of a single adult. During the course of the trials a large number of medical experts were called. In two of the appeals what was asserted to be ‘fresh’ medical expert evidence was called. These three cases highlight a particular feature of cases where it is alleged a baby has been shaken in the care of a single adult. The evidence to prove guilt may consist only of expert evidence. It must never be forgotten that that expert evidence is relied upon to prove that the individual defendant is lying in the account he gives, either at the time or at trial. The correct management of such evidence is, therefore, of crucial importance in cases such as these. The correct approach to such evidence must be identified. If a conviction is to be based merely on the evidence of experts then that conviction can only be regarded as safe if the case proceeds on a logically justifiable basis. That entails a logically justifiable basis for accepting or rejecting the expert evidence (see *R v Kai-Whitewind* [2005] 2 Cr App R 31 [90]). Hearing these three appeals in succession affords an opportunity to make observations on the correct approach and the management of such expert evidence.

3. We should draw attention to the principles we have applied in relation to the admission of fresh evidence pursuant to s.23 of the Criminal Appeal Act 1968. In all three appeals the appellants sought to adduce fresh expert medical witnesses, although it was not necessary to hear the expert evidence in Butler. As is apparent in a trilogy of cases (*R v Stephen Jones* [1997] 1 Cr App R 86, *R v Meechan* [2009] EWCA Crim 1701 and *Kai-Whitewind*) it is difficult to apply the provisions of that section to expert evidence. Where medical evidence is adduced before the Court of Appeal by an appellant from witnesses who were not called at trial and that evidence appears cogent

and relevant, it is difficult for this court to exclude it on the basis that that evidence should have been called at trial. There is a danger, therefore, of overlooking the importance of the principle identified by Lord Bingham CJ in *Stephen Jones* and repeated by Sir Anthony May P in *Meechan* that it would subvert the trial process if a defendant were to be generally free to mount on appeal an expert case which, if sound, could and should have been advanced before the jury (*Stephen Jones* at [93], *Meechan* at [1] and [23]). In *Kai-Whitewind* Judge LJ observed that the court would only in the rarest of circumstances permit repetition or near-repetition of “evidence of the same effect by some other expert”. Trials should not be a “dry run” for experts. Hearings of appeals should not present an opportunity to call new experts in the hope that they might do better than those whose evidence had previously been rejected [97]. We have sought to apply those principles, particularly in the light of the fact that expert reports were obtained for the trial by the defence in Henderson and the defence chose not to call those experts because, in part, they assisted the prosecution. In such a case, an appellant should not be in any better position than an appellant who had called evidence at trial.

4. The important observation of Lord Bingham CJ in *R v Pendleton* [2002] 1 Cr App R 441 [17] that trial by jury does not mean trial by jury in the first instance and trial by judges in the Court of Appeal in the second, applies no less to cases which depend upon expert evidence than to those which do not. But the difficulty of applying s.23 in cases which depend entirely on expert evidence is more acute.
5. Since the appeal depends upon an assessment of the expert evidence, just as at trial, the preparation and marshalling of that expert evidence is of the utmost importance in achieving just resolution. The appeal requires presentation by counsel experienced and expert in the field of what is contended to be the unexplained death of or injury to a child. Such counsel need to be able to identify focussed issues upon which this court can concentrate and to identify the evidence, whether it be evidence at trial or which it is sought to call, on which resolution of those issues will depend. All counsel we heard in these cases were able to assist due to their experience in cases such as these and to the skill with which they deployed that experience. It is no criticism of other counsel if we highlight the manner in which counsel for the prosecution, Joanna Glynn QC and Sarah Campbell, and for the defence, Mr Topolski QC and Andrew Scott, in Henderson, prepared their appeal. The skeleton arguments were focussed upon the particular medical evidence. Different features of that evidence were clearly identified and when any medical proposition was advanced, it was explained and its source clearly identified. A number of different disciplines were involved, all of which were clearly distinguished by separate files, separately coloured and with the underlying evidence and literature upon which that evidence was based, identified and collated. A core literature file, prepared by Mr Topolski, enabled the court to find and weigh the underlying literature upon which controversial evidence was based. The Vice-President conducted a detailed case management hearing providing timetables and giving directions as to how the evidence was to be prepared. Importantly, meetings were held between the experts so as to identify clearly those issues upon which agreement had been reached and those issues which remained a matter of debate. Without such preparation and obedience to the directions given by the

Vice-President it would have been difficult properly to resolve the appeal. The example of the preparation in that case should, we suggest, be followed in future appeals. We shall return to the theme of management and preparation in relation to the conduct of trials when we have considered the individual appeals before this court.

6. There is a further problem to which we should draw attention. Cases such as *R v Harris and Others* [2006] 1 Cr App R 5 contain detailed analysis of medical evidence and the conclusions of this court on that evidence. In particular, in that case, the court commented upon the cogency of the “triad”: widespread bilateral retinal haemorrhages, thin film subdural haemorrhage and encephalopathy [63] and [69]. The court rejected the unified hypothesis that the same triad of injuries could be caused by lack of oxygen in the tissues leading to brain swelling, a hypothesis which Dr Geddes herself accepted could no longer credibly be put forward [66-68]. These conclusions have inevitably informed the basis of the approach taken by the police and prosecution and have been relied on in argument either at trial or before this court. But it is trite to observe that the conclusion of any court as to the medical evidence, whether at first instance or on appeal, is dependent upon the evidence before that court. No appellate jurisprudence could provide authority for a medical proposition. The strength of a proposition in medicine depends upon the strength of the medical evidence on which it is based. The quality and extent of the evidence will inevitably vary from case to case. Whilst it is now commonly accepted that the triad is strong prima facie evidence of shaking, that depends upon the common acceptance of experts in the field and not upon the conclusion of courts which are only able to weigh the evidence presented before them. Previous legal authority cannot determine whether the conclusion of a medical report should be accepted or rejected. The most legal authority can do is present an accurate record of what was or was not accepted or propounded.

7. We stress this problem because we feared that the medical profession may have looked to the courts to resolve medical controversy. But the difficulty the courts face, in an area of medical controversy, was demonstrated by the course of these appeals. It had been hoped to hear the appeals of Henderson and Butler before the end of 2009. However, renewed controversy in relation to post-mortem findings in cases of traumatic head injury in children led to a meeting at the Royal College of Pathologists on 10 December 2009. The controversy was a matter of concern to the courts as the President, Professor Furness, recognised. At the meeting a number of those recognised to have expertise in the field, who advanced opposing opinions, attended. Certain areas of agreement and disagreement were recorded. But they give rise to two difficulties. Those areas of agreement and disagreement should inform future opinions. It is difficult to see how any expert could advance an opinion to which those conclusions were relevant without at least reference to them. But the record of those areas of agreement and disagreement is not itself evidence. Further, whilst the report had been circulated for amendment, positive confirmation of its content was not received by the President from all the participants. We have not been able to deploy that report save insofar as it was adopted in evidence before us. We should say, however, that, in the evidence before us in these appeals, no expert sought to

undermine the proposition that the triad was, as described in *Harris*, “a strong pointer to non-accidental head injury”. But we should emphasise that none of these three cases is concerned solely with the triad: in Henderson the issue was whether there was evidence of injury independent of and additional to the triad, in Butler one of the issues was whether the unusual fact of the baby’s recovery after the discovery of retinal haemorrhage indicated a cause other than shaking, and in Oyediran the prosecution alleged a distinct and separate injury, namely, fracture of the baby’s arm. For those reasons, we must emphasise that this judgment constitutes legal, not medical, authority and neither adds to nor subtracts from the strength of the evidence afforded by the triad.

8. Two of the appeals, Henderson and Butler, were heard by the same constitution. But in the third appeal, Hedley J was replaced by Sharp J. Counsel in all appeals had the opportunity to study a careful and comprehensive note, for which we are most grateful, made by counsel Gwawr Thomas. All judges have made substantial contribution to the individual appeals but, of course, neither Hedley J nor Sharp J has commented upon the appeal which they did not hear. The appeals were heard in succession, with a week between the second and third, because it was thought that it would be unfortunate if different constitutions wrote different judgments on different occasions when the appeals were heard so closely together. We turn, then, to those individual appeals.

Henderson

9. Maeve Shepherd was born on 7 April 2004. On 2 March 2005, when she was just under 11 months old, she suddenly collapsed whilst in the care of the appellant and was taken to hospital. She remained in a critical condition for two days but, tragically, died on 4 March 2005.
10. The appellant was a well-respected childminder of whom witnesses spoke highly. She had been a registered childminder since November 2000 and was permitted to care for up to five children under the age of 8 at any given time. She had, in addition, her own two sons. The prosecution alleged that, on 2 March 2005, she had shaken Maeve, or, in shaking, had caused an impact to her head on a soft surface leading to the baby’s sudden collapse and subsequent death. The appellant was charged with manslaughter. After a six week trial at which the prosecution called eleven expert medical witnesses and the defence one, a jury at Reading Crown Court, on 13 November, 2007, convicted the appellant of manslaughter by a majority of ten to two. Strictly, unless and until the appellant’s application to call fresh evidence is granted, no permission should be given. But the case is of sufficient importance and complexity to grant permission before reaching any conclusion as to the admissibility of that evidence.
11. The appellant, supported by her character witnesses, gave evidence that she had not shaken the baby and was not responsible for her death. The only evidence on which the prosecution could rely to make the jury sure that the appellant was lying was the

medical evidence of fact and opinion. This appeal is concerned with fresh evidence from two experts which, it is contended, casts doubt on the reliability of the conclusion that this appellant unlawfully killed Maeve Shepherd. The appeal was focussed on two particular aspects: ophthalmological and neuropathological evidence. But it is necessary to place those particular features within the context of the facts and of the expert evidence at trial.

Evidence at Trial

12. Until she returned to work, Maeve's mother looked after her. A family friend then cared for her until the appellant was recommended to Maeve's parents by a friend. From 31 January Maeve became unwell with a number of infections. The appellant conscientiously kept a diary which travelled home with the baby each evening. From the outset she found difficulty in helping Maeve to feed. On 2 February 2005 Maeve had a cough and runny nose, she vomited three times and the appellant took her to a GP who diagnosed a viral illness. She was sick the following two days whilst in the appellant's care. Two days later, on 4 February 2005, she spent the day with her mother, appeared very unwell, and was taken to see the GP who diagnosed a chest infection and prescribed antibiotics. Following the weekend, she visited the GP again. On 8 February 2005 when her condition had improved she spent the day with the appellant. She was sick when she was fed. No problems were recorded during the following two days, 9 and 10 February, whilst Maeve was with the appellant. Between 11-13 February, when she was with her parents, her condition improved.
13. On 15 February 2005 events took a more serious turn. Whilst in the care of the appellant, Maeve was sick and the appellant rang Maeve's mother to tell her that Maeve had banged her head and that the appellant was very worried about her. With Mrs Shepherd's permission, the appellant took Maeve to the GP and told the doctor that Maeve had rolled over on a hard floor and hit her head. The appellant then, on the doctor's advice, took Maeve to a local hospital where she was diagnosed with an upper respiratory tract infection. During the next five days Maeve was cared for by her parents; she started to vomit on 17 February 2005. A doctor diagnosed viral illness. But Maeve continued to be unwell and returned to hospital on 19 February 2005 where she was kept under observation for several hours. Upper respiratory tract infection was again diagnosed and she was discharged until the following day, 20 February 2005, when at a paediatric assessment clinic she was noted to be better. The appellant looked after her on 22 February without incident but on 24 February 2005 Maeve was unable to feed, vomited in the morning, and was sick after lunch. Maeve's father collected her and she was taken to the GP who again diagnosed upper respiratory tract infection and prescribed a course of antibiotics. Maeve remained unwell when she was with her parents on 25 February 2005 but appeared to improve during the following two days. The appellant looked after Maeve on 28 and 29 February 2005 without incident although Maeve vomited on both days.
14. On 2 March 2005 the appellant looked after Maeve from about 7.30 a.m. The appellant took her to a "mother and toddler" group, returning at approximately 11.00

a.m. After Maeve's collapse a friend who drove Maeve's mother to the appellant's house advised the appellant to write down exactly what had happened before she forgot the details. The appellant followed this advice, scrupulously. She recorded that at about 11.10 a.m. Maeve was awake, happy, and was "whizzing around in the baby-walker" but at about 12.00 p.m. she would not eat and spat out the mouthful she was offered. She attempted to change her nappy and noted that Maeve was "grizzling like normal". She then described the baby having a seizure, screaming, and that she "just went floppy". The appellant tried to feed her with juice but Maeve was not swallowing at all. The appellant rang 999 and checked her airways and throat but found nothing.

15. The harrowing record of the 999 call demonstrates the appellant's concern. Particularly, she described Maeve's difficulty in breathing:-

"She keeps losing consciousness...she's just gone into like a seizure sort of thing. She keeps taking little breaths but she's not getting anything inside of her. She hasn't took a breath now for ages. She's completely rag doll. She's comatose. She looks dead. She keeps trying to take a breath."

The ambulance arrived at 12.11 p.m. and the paramedics' report describes a reduced respiratory rate which improved as they attempted to restore the oxygen supply. The Glasgow Coma Scale showed the minimum motor ability (1). She was, in effect, found to be moribund. There was no sign of external injury.

16. Maeve was taken to Wexham Park Hospital. It is of significance to note that retinal haemorrhages were seen at 4.10 p.m., within four hours of admission. She was taken that evening to John Radcliffe Hospital, Oxford. At 5.30 a.m. the following day, 3 March, retinal folds, which had been suggested at 2.00 a.m., were confirmed.
17. Maeve remained on life support but died two and a half days later on the evening of 4 March 2005.
18. There were three medical witnesses who described the symptoms they found whilst Maeve remained alive. Dr Connell, Consultant Paediatrician at Wexham Park Hospital, spoke of the CT scan taken shortly after her arrival, which showed severe and widespread hypoxic-ischaemic damage to her brain. He and three other clinicians saw retinal haemorrhages. He regarded those as signs of inflicted injury and called for various investigations to see whether Maeve's collapse and subsequent death had been caused by natural causes or trauma. Dr Pike, Consultant Paediatric Neurologist at the John Radcliffe Hospital, initially suspected either a non-specific infection or trauma. He observed retinal haemorrhages and, in the absence of signs of disturbance to Maeve's blood clotting system, suspected that she had been shaken. Dr Ali, a Consultant in paediatric anaesthesia and paediatric intensive care, gave evidence to similar effect.

The Issues

19. The essential issue at trial was whether the prosecution could prove that the appellant's denial that any traumatic event had occurred on 2 March 2005 was untrue and that the true explanation for the baby's death was trauma inflicted deliberately by the appellant. The prosecution contended that the expert evidence demonstrated not only all three features of "the triad" but significant and clear characteristics within those features, all of which, taken together, demonstrated injury caused by shaking in a manner which was obviously dangerous. In addition, the prosecution relied on what they contended were two additional features consistent only with trauma: first, traumatic axonal injury in the cortico-spinal tracts and second, retinal folds. The prosecution relied upon eight experts who excluded "natural" causes of death such as infection, metabolic disorder, genetic disorder and malformation of the brain.
20. The defence case was that either Maeve suffered a seizure, possibly triggered by what had happened two weeks earlier on 15 February 2005 or that the cause was unknown and that, in the light of the appellant's own evidence and the respect in which she was held by others, the prosecution could not prove that she was responsible for injuring the child.

Expert Evidence at Trial

21. We can deal comparatively shortly with the issue of whether the prosecution disproved any known "natural" cause of death, such as infection. We emphasise that we are dealing with natural causes of death within the purview of up-to-date medical knowledge because in this appeal we were properly reminded that at no stage can knowledge in a field such as this be regarded as complete and comprehensive. There are limits to the extent of knowledge and no conclusion should be reached without acknowledging the possibility of an unknown cause emerging into the light of medical perception and that the mere exclusion of every possible known cause does not prove the deliberate infliction of violence (see *Canning passim* and [177]).
22. The evidence of known natural causes of death at trial was comprehensive. Mr Peter Richards, a Consultant Paediatric Neurosurgeon, gave evidence as to the thorough nature of Dr Pike's investigation as to natural disease. All natural diseases that could cause subdural haemorrhages were excluded. At the trial the appellant relied upon Dr Anslow, a Consultant Paediatric Neuroradiologist. It was his evidence that Maeve might have suffered a seizure brought about spontaneously which resulted in hypoxic-ischaemic brain damage leading to swelling of the brain and raised intra-cranial pressure. As the defence now stress, the evidence of the triad and the retinal folds and axonal damage (the triad with those two additional features) was never challenged, nor was the proposition that they demonstrated violent trauma. The issues of retinal folds and axonal damage were topics outwith the expertise of Dr Anslow. The appellant was unable to explain the two critical features additional to

the triad which formed the basis of the prosecution case against her. This appeal is concerned with her attempts to do so now.

23. We turn to the evidence at trial of those two additional features. Professor Risdon, a Consultant Paediatric Histopathologist, explained to the jury the three features of the triad which he said were highly indicative of inflicted head injury by the violent rotation of the head backwards or forwards, such as might be expected if the baby was shaken or the head came to an abrupt halt when in contact with a soft surface. The three features of the triad, present in this case, were encephalopathy, specifically hypoxic-ischaemic brain damage, second, retinal haemorrhages and third, subdural haemorrhages. Whilst he accepted that any of those three individual components could be found in cases other than inflicted injury, it was very unusual to find them together, save in cases of inflicted head injury. He emphasised the importance of the characteristics of each of those components and the importance of considering them as a whole. He acknowledged that there were other individuals who took a contrary view but suggested that they tended to be experts not involved in the day-to-day care and management of abused children.
24. The importance of considering the evidence as a whole and the particular characteristics of the three individual components was emphasised by Dr Stoodley, Consultant Neuroradiologist and Dr Cary, a Forensic Pathologist. None suggested that the presence of the triad, even with the characteristics demonstrated in this case, was dispositive or provided a certain diagnosis. But all emphasised those characteristics indicate what this court described, in *R v Harris*, as “a strong pointer” to a non-accidental head injury (see [70]).
25. In this appeal, it is unnecessary to dwell on the extent to which a case which merely contains the features of a triad will be sufficient to prove non-accidental injury. This is not a case, we emphasise, on which the prosecution relied merely on the triad. This case is concerned with whether the triad and what the prosecution describe as two additional features were and remain sufficient to prove the appellant’s guilt.

Ophthalmological Evidence

26. Whilst Maeve was alive, her eyes were examined by clinicians, Dr Gibson and by Mr Elston, Consultant Ophthalmic Surgeon at the Oxford Eye Hospital, the John Radcliffe Hospital, specialising in paediatric and neuro-ophthalmology. His particular clinical paediatric experience is concerned with examining children’s eyes in a clinical setting. Whilst Maeve was alive, he found retinal haemorrhages in all the different layers of the retina extending throughout the whole of the retina. Second, he found that both eyes had a 360° fold, with an optic nerve sheath haemorrhage in the right eye. As we have recalled, the haemorrhages were first seen at Wexham Park Hospital by non-specialist doctors four hours after Maeve’s collapse.

27. The distribution of these retinal haemorrhages was, so Mr Elston told the jury, indicative of trauma. They were consistent with shaking or a shaking and impact but not with accidental trauma or non-traumatic raised intra-cranial pressure. Raised intra-cranial pressure was eliminated for reasons which need not now detain us (the two possible causes, retinal haemorrhages secondary to persistently elevated intra-cranial pressure causing papilloedema and cases of Terson's syndrome were excluded). The time when these haemorrhages were first seen was particularly important in excluding non-traumatic raised intra-cranial pressure as a cause. The next feature of the retinal haemorrhages which is of importance is their effect. They would have rendered Maeve blind or almost blind as soon as they were sustained. The eye injuries must, accordingly, have been sustained shortly before midday on 2 March 2005 since she was awake and happy, "whizzing around in her baby-walker" after about 11.10 a.m. The other important feature was the discovery of retinal folds. Mr Elston told the jury that he had only seen such perimacular retinal folds in cases of trauma. That may include accidental trauma, accompanied by external signs of injury, but most commonly caused by shaking or a combination of shaking and impact. Mr Elston told the jury that in 25 years of clinical paediatric ophthalmology he had only seen retinal folds cases of shaking or shaking and impact.
28. At the trial the defence legal team had obtained a report from a Consultant Histopathologist and Ophthalmic Pathologist, Dr John McCarthy. The report was disclosed to the prosecution in August 2007: his evidence of the extent of the haemorrhages was consistent with that of Mr Elston. In those circumstances he was not called. But the defence now rely upon an expert of the same discipline as Dr McCarthy to cast doubt on the safety of Mr Elston's conclusions. That expert is Professor Luthert.
29. Professor Luthert is a Professor of Pathology and Consultant Ophthalmologist at the UCL Institute of Ophthalmology. His distinguished career and qualifications were not the subject of any challenge by the prosecution and so it is unnecessary for us to give their detail. The prosecution merely point out that he is not a clinician but a pathologist of similar standing to Dr McCarthy, whom the defence at trial chose not to call. Professor Luthert produced three reports dated 8 July 2008, 23 February 2009 and a consolidated report dated 5 October 2009. There was a joint conference between him and Mr Elston from which a document was produced, signed by both. In his first report Professor Luthert said that whilst shaking could be the cause of the retinal haemorrhages and retinal folds, "the evidence base is not as strong as one might hope for". He referred to raised intra-cranial pressure. This can be excluded for the reasons we have given [26]. He also referred to a case of very extensive retinal haemorrhages caused by meningitis. Meningitis has been excluded in this case. He concludes:
- "I think it entirely reasonable to consider it likely that Maeve collapsed due to inflicted trauma. There is no known, well-documented alternate explanation that I am aware of that fits with what I have read of the clinical and pathological findings in this case. Nevertheless, for reasons I have outlined

above, I consider it problematic to assume that the ‘triad’ is diagnostic of trauma and I think this view is increasingly widely held. I am concerned that the findings in the eyes should not be interpreted as direct evidence of trauma.”

30. After Professor Luthert had received the ophthalmic pathology slides he composed a second report dated 23 February 2009 in which he repeats that he has read nothing incompatible with inflicted head injury and adds that he knows of no other “witnessed and validated cause of the above triad other than, rarely, accidental injury”. He accepts in that report that there is a strong association between trauma and perimacular folds. In his final, summary note, he reiterates that he believes that the triad can occur as a result of trauma and that nothing in the eyes or in the documentation which he read was “strongly suggestive of an alternative diagnosis”. But he repeats that the findings are not diagnostic of trauma.
31. In his first report Professor Luthert says that the cause of retinal haemorrhages in cases of alleged head injury in infants is not known. He refers to hypotheses including trauma-induced tractional forces between the vitreous and retina, increased retinal intra-vascular pressure, secondary to increased intra-cranial pressure, splinting of the chest during shaking and loss of normal control of vascular tone (loss of auto regulation). Additionally, the mechanism of retinal fold formation is not known with certainty. Professor Luthert accepts that such folds are more commonly seen in more severe cases of retinal haemorrhage but he does not believe that there is “compelling evidence that they arise from trauma-related tractional forces from the retina”. He advances what he describes as an equally tenable alternate hypothesis, namely:-
- “They arise from profound expansion of the retina due to haemorrhage and oedema (increased tissue fluid following injury).”
32. In this second report dated 23 February 2009 he concludes:-
- “I cannot be certain that an admittedly unknown non-traumatic aetiology can lead to the triad” (*we think can should read cannot*).
33. Professor Luthert also referred to some experimental data supporting a non-traumatic cause as the mechanism for retinal fold formation. This is a reference to correspondence from Dr Gardiner commenting on a paper by Dr Sturm in the American Journal of Ophthalmology of April 2008. Dr Sturm had suggested that a process, known as OCT, revealed morphological changes, missed by clinical examination, which provided data in favour of the theory of vitreo-retinal traction as a direct mechanical effect attributable to rapid head movements which occur when a baby is shaken. That view was criticised by Dr Gardiner who suggested that the

haemorrhage itself might raise the fold. Dr Sturm disagreed, not least because the vitreous of young children is much more adherent to the retina than it is in adults.

34. Mr Elston rejected Dr Gardiner's challenge to Dr Sturm's paper on the basis of his clinical observation. Dr Gardiner had suggested that haemorrhaging itself might cause a raise in the fold. Mr Elston said that the hypothesis was not relevant in Maeve's case because Dr Gardiner's hypothesis requires a cavity posterior to the apices of the folds to be full of blood. Maeve's were not (see Elston's report 10 March 2009). Mr Elston rejected Professor Luthert's reliance upon animal experiments on kittens (*Troll* 1999) and hamsters (*Khalifa* 1991).
35. The reports of Professor Luthert led, as we have said, to a meeting. The joint report which emerged and was signed by both Mr Elston and Professor Luthert is dated 20 January 2010. The clinical and pathological findings were agreed. The report records:-

“The experts further agreed that:-

- i) The focal brain stem/spinal cord injury is understood to be of traumatic origin and provides evidence of focal injury to the central nervous system, contemporary with the retinal injury (this is, in fact, controversial but depends on a dispute between non-ophthalmological experts: we deal with this below).
 - ii) The known causes of injuries to the central nervous system and the eye listed in sections 1 and 2 above, when seen together, are all traumatic in origin (see below). The mechanism by which such trauma leads to the physical signs in the eye is not known, but the fundamental aetiology is traumatic.
 - iii) The presence of prior subdural haemorrhage does not pre-dispose the eye to development of the clinical and pathological findings noted in this case.”
36. Under the heading “Causes of the Ophthalmological Clinical and Pathological Findings” known and verifiable causes of the very severe haemorrhagic retinopathy and the perimacular fold were agreed to be inflicted injury in the form of shaking or shaking and impact. Other causes, such as a fatal crush head injury or a single impact head injury or Terson Syndrome, were excluded. Under the heading “Mechanism of Eye Signs” it was accepted that the mechanism of the formation of retinal folds with pan-retinal haemorrhagic retinopathy was not known with certainty. The joint report refers to the hypothesis that the findings were due to trauma related to acute tractional forces or an alternative hypothesis that the retinal folds can be caused by “acute

expansion of the retina with haemorrhagic retinopathy due to failure of retinal vascular auto regulation and folding of the ischaemic retina”.

37. Professor Luthert gave oral evidence before us which was consistent with the evidence in his reports. In his oral evidence he emphasised that, absent certainty as to how folds are caused, they do not, in his opinion, provide evidence distinct from the evidence of the triad. Accordingly, very extensive retinal haemorrhages could themselves be reasonably expected to cause retinal folds. His view was that such folds were not independent evidence of trauma but merely a manifestation of very severe retinal haemorrhage. In cross-examination he repeated that neither the folds nor the optic nerve sheath haemorrhage were independent of the retinal haemorrhages themselves.
38. During the course of his evidence Professor Luthert referred to some form of ALTE (acute life-threatening event) which could result in disturbance to circulation, inadequate supply of oxygen to the retina and associated changes in intra-cranial pressure.
39. There was a striking development since trial. At trial it was accepted that the only known cause of retinal folds was trauma. Between the time of the reports and Professor Luthert’s evidence, one case emerged of perimacular folds associated with extensive retinal haemorrhages not due to trauma but rather to acute myeloid leukaemia suffered by a 14 year-old (Bhatnagar and others ARCH Ophthalmol Vol 127 November 2009). The paper demonstrated that perimacular folds could be caused other than by trauma although this was unknown both at the time of trial and at the time the reports were prepared for the purposes of this appeal. It was not suggested that Maeve suffered from leukaemia. But the report powerfully demonstrated, so it was contended, a cause which was previously unknown, unrelated to trauma and unforeseen and unexpected. It demonstrated the limits of knowledge at any given time in the field of medical science. The previously accepted proposition that *only* trauma can cause retinal folds was shown to be incorrect.
40. Professor Luthert accepted that it is reasonable to conclude that Maeve suffered from inflicted trauma and was himself unable to provide any alternative to a traumatic cause to the eye injuries. He accepted that hypoxic-ischaemic injury to the brain is not a recognised cause of extensive retinal haemorrhages with folds (second report, dated 23 February 2009).
41. We accept, not least because the experts were agreed, that the mechanism of retinal fold formation is not known with certainty. But we reject Professor Luthert’s suggestion that the existence of retinal folds is not a feature additional to the triad. The triad affords strong support, whilst not being conclusive, of shaking or shaking and impact injury absent the presence of retinal folds. Since it is not known how retinal folds are formed there is no sound evidential basis for saying that they occur merely as a result of a haemorrhage. Mr Elston’s evidence, as a clinician, that in 25

years of paediatric ophthalmology he has only ever seen retinal folds in shaking or shaking and impact cases (his evidence at trial, repeated in his report dated 10 March 2009) persuades us that the folds are features pointing towards shaking or shaking with impact over and above the appearance of the haemorrhage.

42. Professor Luthert's evidence does not challenge the association between retinal folds and trauma. No doubt that is the reason why he reached the agreement we have set out above at [36]. In effect, he is urging more caution in reaching any diagnostic conclusion. But he does not seek to dispute Mr Elston's clinical experience and, indeed, he is in no position to do so since he is a pathologist and not a clinician. Moreover, in his evidence he is unable to suggest any alternative reason for the retinal haemorrhages and in particular their appearance, extent and location. Nor is he able to put forward any alternative cause for the folds, other than that they are to be regarded as attributable to the haemorrhages. In particular, the evidence obtained from other paediatric clinicians since the trial excludes infection as a realistic cause of the retinal haemorrhages. Professor Pollard, Professor of Paediatric Infection and Immunity at Oxford University, excludes systemic infection as a cause of the haemorrhages in the absence of any coagulation disturbance associated with shock. Professor Klein, Professor of Infectious Diseases and Immunology at the Institute of Child Health and an Honorary Consultant in Great Ormond Street Hospital, gives, in his final report dated 11 November 2009, similar evidence. He and Dr Peters, Senior Lecturer in Paediatric Intensive Care at UCL, and Consultant Paediatric Neo-Natal Intensivist at Great Ormond Street Hospital, reject resuscitation as a cause of the retinal haemorrhages.
43. In those circumstances, Professor's Luthert's evidence amounts to no more than an expression of greater doubt and greater caution than that to which Mr Elston would subscribe. The discovery of a case of retinal folds due to leukaemia, and, thus, without any traumatic cause, emphasises the importance of recognising the limits of medical knowledge at any given time and the need to appreciate that that which has never previously been contemplated may nonetheless occur.
44. We must recognise the limits of medical science and in particular that there may be events, deaths or symptoms which are unexplained and unforeseen. Further, any conclusion must acknowledge the importance of the burden of proof in the context of cases such as these. It is not for the defence to provide any explanation; the mere fact that it is unable to do so is not of itself a sound basis for concluding that the prosecution's evidence is correct.
45. But we must also recall that we are dealing with an appeal and not with a trial. The essential question is whether Professor Luthert's evidence casts doubt on the safety of the verdict. We remind ourselves that the evidence comes, like the evidence from Dr McCarthy, whom the defence chose not to call, from an expert pathologist of the same discipline as Dr McCarthy. For the reasons we have given, Professor's Luthert's evidence is, at its heart, no different from the evidence of Dr McCarthy save as to expressions of greater caution and doubt. In the absence of any new explanation as to

the cause of the haemorrhages or folds, it does not undermine the evidence given by Mr Elston.

46. The joint report appeared to demonstrate the consensus between Professor Luthert and Mr Elston that known causes of the constellation of injuries, particularly the recent acute onset of haemorrhage and retinal folds, were all traumatic. Perhaps, on first reading, we failed to appreciate the emphasis upon “known” causes. Essentially, Professor Luthert’s evidence amounted to no more than that the injuries were probably due to shaking or shaking with some impact but that he could not be certain. That in our view is insufficient to undermine the safety of Mr Elston’s conclusion.
47. There is one further aspect of Professor Luthert’s evidence to which we ought to draw attention. The evidence of any expert in a particular field is inevitably limited to the field in which he professes expertise. That, of course, is why no expert can be in the position of a jury or, for that matter, of this court, able to put particular evidence in the context of the totality. Professor Luthert acknowledged this in referring to the evidence of traumatic injury to the nerve fibres (“traumatic axonal injury”). He accepted that that would be additional evidence of trauma, outside his field of expertise, and would diminish the relevance of his discussion as to the interpretation of what he called “the triad alone” (see his note dated 5 October 2009). It is, accordingly, necessary to turn to the evidence of the damage to the nerve fibres.

Axonal Trauma: Neuropathological Evidence

48. The second factor on which the prosecution relied as additional to the triad was founded on the evidence of Dr Al-Sarraj, a Consultant Neuropathologist, that he had identified traumatic axonal injury in the cortico-spinal tracts by the use of beta-app staining. He distinguished that type of injury from diffuse axonal injury. As Professor Luthert accepted, if that evidence was accepted, it was a powerful indication, when added to the evidence of the triad, of a shaking or shaking and impact injury.
49. At trial, the two neuropathologists called by the Crown, Dr Squier and Dr Al-Sarraj, both asserted that they could distinguish between diffuse axonal injury and traumatic axonal injury. Dr Al-Sarraj asserted that he could determine that the traumatic axonal injury was about two to three days old, consistent with Maeve sustaining that injury on 2 March 2005. The defence challenged Dr Al-Sarraj’s ability to make the distinction. But Dr Al-Sarraj relied in part on slides taken by Dr Geddes as part of her research in papers which have become known as Geddes I and II, and on Reichard “The Significance of Beta App Immunoreactivity in Forensic Practice”. Dr Al-Sarraj also relied upon his own research, published literature and experience. A critical feature for distinction was the location of the axonal damage in the cortico-spinal tracts and the appearance of the severed axons, the ends of which pick up the stain and show up as round globules rather than a diffuse cloud or “granular staining”, as Dr Al-Sarraj described it. The distinction was important in demonstrating that the cause

was not ischemia which will affect the whole area in which there is a lack of oxygen. Traumatic injury will cut individual axons causing a leak of protein which will show on the beta app stain (this particular evidence became clear in response to a pointed jury question at the end of Dr Al-Sarraj's cross-examination).

50. In Geddes I, "Neuropathology of Inflicted Head Injury in Children" (*Brain* [2001] 124 (1290-1298) and II (1299-1306), Dr Geddes demonstrated that the brain damage caused by non-accidental head injury suffered by children was due not to the trauma but to oxygen starvation, i.e., hypoxic-ischaemic damage. But for the purposes of the neuropathology in the instant case what is important about both Dr Geddes's papers is the reference to axonal damage at the cranio-cervical junction. Dr Geddes sought to distinguish between axonal damage caused by trauma and axonal damage secondary to hypoxia-ischaemia, raised intra-cranial pressure and/or brain shift (1297). Localised axonal damage demonstrated in cortico-spinal tracts was regarded as significant. In Geddes II the authors identified foci of ischaemic-type staining in the dorsal brain stem but remarked:-

"However, the staining in the cortico-spinal tracts was quite distinct, affecting variable numbers of axons in these fibre bundles bilaterally and appeared to represent localised traumatic axonal injury at the cranio-cervical junction. We believe that this pattern results from non-disruptive stretch injury to the neuraxis." (1305)

In Reichard, (*Neuropathology and Applied Neurobiology* [2005] 31) Dr Reichard reviewed seventy-three cases and assessed them "blind" to the clinical history. His assessment endorsed the value of beta app staining in assessing the extent of axonal injury and in particular that it was of "the greatest utility" in the assessment of traumatic brain injury. The evidence of Dr Al-Sarraj and Dr Squier on this aspect, coupled with the literature on which it was based, was not met by any contrary evidence called on behalf of the defence. The defence had instructed Dr Colin Smith, a highly experienced expert in "neuropathology and baby-shaking" who was a co-author of the Reichard paper to which we have already referred. It chose not to call him although his evidence was served on the prosecution; it did not assist the defence. The defence also obtained a report from Professor Milroy, Professor of Forensic Pathology at the University of Sheffield on which, since it supported the prosecution, the defence did not rely.

51. The defence now seek to rely upon Dr Leestma and we received his reports and heard oral evidence from him. Dr Leestma is a neuropathologist from Chicago. He was Professor of Pathology and Neurology at the University of Chicago between 1986 and 1987 and Neuropathologist and Associate Medical Director for the Chicago Institute of Neurosurgery and Neuro Research between 1987 and 2002 and a Neuropathology Consultant for the Children's Memorial Hospital of North Western University Medical Centre Chicago between 2003 and 2005. He has been consulted in matters of forensic neuropathology in private practice for the past thirty years. He advanced a number of propositions in reports dated 10 July 2008, 2 February 2009 and 24 June

2009. Not all of them survived by the time he gave oral evidence before us. In particular, he had previously raised the possibility that the axonal pathology may have been due to artefacts produced by removal of the brain and cord post-mortem. After discussion with Dr Al-Sarraj and after a meeting between the two doctors, he no longer pursued that possibility. When he wrote his first report dated 10 July 2008 Dr Leestma suggested that whilst subdural haematomas in infancy are more likely to be due to physical forces, inflicted or accidental, “there are a host of natural disease states that can cause them”. Now that the appellant has expressly abandoned infection as a cause of the constellation of symptoms from which Maeve suffered and that it is accepted that those symptoms are not attributable to any known infection, Dr Leestma’s proposition, that there are many rational explanations for Maeve’s symptoms other than abuse, was no longer relied upon.

52. Dr Leestma also suggested, in his first report, an accidental “short” fall followed by a lucid interval. This suggestion is no longer pursued and was abandoned by those now acting for the appellant during the course of the preparation of the appeal. It was, in any event, the subject of undisputed evidence at trial. Particular reliance was placed upon the fact that no space-occupying subdural haematoma was found.
53. The absence of a space-occupying subdural haematoma is also of importance in relation to a further possibility raised by Dr Leestma: that the recent haemorrhage was caused by a re-bleed from a subdural haematoma sustained two weeks earlier; it was part of the natural process of the chronic subdural haematoma. The undisputed evidence at trial was that any re-bleed would not have caused the catastrophic injury and death of Maeve. Dr Al-Sarraj accepted that recent bleeding could be a re-bleed from an old haematoma but there was only a small amount of subdural haematoma found and that would not explain Maeve’s death. The refutation of Dr Leestma’s written suggestion as to the cause of the subdural haemorrhage is summed up by the clinician Dr Peters. He said that in his sixteen years of practice in Great Ormond Street:-

“I have never seen a clinically detectable subdural haemorrhage in the absence of trauma, severe central nervous system injury such as a stroke, or an abnormal bleeding tendency.”

The last two have been eliminated. At trial, Dr Cary (a Forensic Pathologist) regarded the signs of fresh subdural bleeding as a marker for what had happened in the brain, showing the application of angular rotational force.

54. The most significant part of Dr Leestma’s evidence challenged the prosecution’s assertions that it was possible to attribute the appearance of axonal damage within the cortico-spinal tracts to trauma. In his report dated 24 June 2009 he stated that the staining did not necessarily reflect any form of physical injury and that:-

“In the presence of obvious “respirator” brain changes from perfusion failure of the brain and possibly parts of the spinal

cord it is probably impossible to differentiate beta app reaction products due to true axonal injury (physical forces) from those due to many complex processes such as circulation failure, hypoxia, oedema and various artefacts of removal and preparation which were present in this case. To conclude that shaking forces were involved in this case requires far more scientific proof that this is a bona fide mechanism of injury than exists.”

55. Dr Leestma had clearly laboured under a difficulty from lack of access to the full set of slides on which Dr Al-Sarraj and Dr Squier had relied and, at least originally, from lack of familiarity with the papers. Prior to his oral evidence before this court, however, there was a meeting at which, on examination of block 13, both experts agreed that deposits of beta app staining in the middle of the cortico-spinal tracts were of a different pattern from that which had been in other areas. They were well-defined isolated globules without a granular background. At that meeting Dr Leestma commented that Dr Al-Sarraj’s view of the significance of well-defined isolated globules “may well be right, I just don’t know”. Dr Leestma did agree that removal of the tissue post-mortem was unlikely to be a reason for the signs observed in the spinal cord. He also commented that beta app staining “was not available when he was doing this kind of work”.
56. At the hearing of the appeal Dr Al-Sarraj repeated and demonstrated particular examples of well-defined globules of beta app staining different from the structure of those to which he attributed ischaemia. He pointed out that they were specific within the cortico-spinal tract and consistent with trauma. They were not consistent with ischaemia.
57. Dr Leestma, in his oral evidence, repeated that he had used beta app testing in his career but not recently. He was, however, familiar with it. He repeated his concern as to the cause of recent bleeding. During cross-examination he revealed that he had not read either Geddes I or II and was thus unfamiliar with their references to the ability to distinguish traumatic axonal injury in the cortico-spinal tract and its significance. He admitted that he had not previously appreciated that Reichard was a blinded study. As he explained in his evidence, he had not understood the significance of the slides described as “block 13” which showed the different pattern of staining in the medulla and cortico-spinal tract. Dr Leestma asserted that it would be possible to observe a destroyed or damaged axon longitudinally and thus one should see a number of damaged axons in one cross-section.
58. This assertion led to Dr Al-Sarraj being recalled. He explained that, if an axon was torn, such damage would not be shown throughout the whole length of the axon but only at a particular location. Whether it was observed or not would depend upon where the cross-section was taken. He disagreed with Dr Leestma that one would expect the damage to extend along the length of the axon; rather the axon would be

torn at a particular location. Thus the number of axons shown as damaged in a cross-section would not be of significance.

59. Dr Leestma also placed reliance upon the absence of any damage to the axons controlling respiration. Dr Al-Sarraj took the view that those axons controlling respiration were, by reason of their location, less vulnerable. He had observed damage to the axons within the cortico-spinal tract where those axons were more vulnerable than those controlling respiration.
60. During the course of his cross-examination Dr Leestma told the court that he had diagnosed baby-shaking many years before in the middle-to-late 1980s, but that in his consulting role he had seen many cases of head injury to babies where there were external signs. He accepted that he had not systematically reviewed the literature since the mid-1990s.
61. In our judgment, Dr Leestma's experience was more historic and far more limited than that of Dr Al-Sarraj. He has not conducted autopsies or given evidence in cases involved with baby-shaking for many years. His knowledge emerges from studies of the literature which excluded the important material contained in Geddes I and II and Reichard. Until his meeting with Dr Al-Sarraj, he had never appreciated the significance of the signs of axonal damage in the cortico-spinal tract. His lack of experience and his lack of appreciation of the importance of this point leads us to the conclusion that his evidence was fundamentally flawed. His insistence on asserting that it was not possible to attribute significance to that axonal damage flies in the face of the references in both Geddes I, II and Reichard and the evidence which flows from Dr Al-Sarraj's up-to-date experience. We reject his evidence insofar as it fails to attribute significance to that which the beta app staining revealed.
62. Further, for the reasons we have given, we reject his evidence as to the significance of any re-bleed. As he himself accepts in his first report dated 10 July 2008, if the brain-stem and other structures had sustained what he described as "true axonal injury" before hospitalisation the baby would not be behaving normally and would "in many ways not be "functioning". This evidence, consistent with evidence given at trial and coupled with the effect of the eye injuries which would have almost blinded Maeve instantly, demonstrates that there is no basis for advancing the proposition that Maeve collapsed as a result of trauma on 15 February 2005 followed by a lucid interval until 2 March 2005.
63. Professor Luthert had recognised the importance of the signs of traumatic injury to the axons. In our judgment, nothing in the evidence of Dr Leestma suggested that the importance attached to those signs of injury was unfounded. Indeed, the willingness of Dr Leestma to advance propositions which he subsequently had to withdraw in the light of his greater knowledge of this case, coupled with his lack of up-to-date experience, severely damaged and undermined the effect of his evidence. We would have had considerable doubts as to whether he was properly qualified to give evidence

designed to refute the evidence given by Dr Al-Sarraj or Dr Squier at trial. We did not reach any concluded view as to that. It is sufficient to conclude that his evidence did not cast doubt upon the safety of the verdict insofar as it relied upon the signs of traumatic injury to the axons in the cortico-spinal tract.

64. The consequences of our conclusion as to Dr Leestma's evidence are that there remains at least one feature of Maeve's symptoms additional to those constituting the triad. Even if we had accepted Professor Luthert's evidence that the perimacular folds may have been attributable to the haemorrhaging and were, therefore, just an aspect of the ophthalmological symptoms of the triad, the evidence of traumatic damage to the axons affords powerful additional evidence of trauma. It forms, with the other undisputed symptoms, a safe foundation for the verdict of guilty.

Other Causes

65. Our focus on the two elements of retinal folds and axonal damage should not be thought to have been the only bases of challenge to the jury's verdict. Although the appeal was focussed on those two aspects of Maeve's symptoms, it is important to record the extent to which other possible causes of her collapse and death have been explored since the trial. On 28 February 2008 provisional grounds of appeal were advanced criticising the appellant's original defence team in their failure to call expert evidence in paediatrics, biomechanical engineering and ophthalmology. It was also suggested that some of the expert witnesses for the prosecution had given inconsistent evidence in another case. Subsequently, following the Crown's written submission in reply, the appellant abandoned all three grounds.
66. By way of further written argument dated 10 October 2008 the appellant applied to call fresh evidence from six experts; three of them, Dr Walters, a Chemical Pathologist, Dr Thibault, dealing with biomechanics, and Dr Plunkett, a General Forensic Pathologist, raised the possibility that Maeve had suffered a fall over a short distance on 15 February 2005 and thereafter had a lucid interval until a further collapse on 2 March 2005. It is necessary to recall that Dr Squier, called on behalf of the prosecution at trial, whilst agreeing with Dr Al-Sarraj as to the identification of traumatic axonal damage, nevertheless took the view, contrary to the opinion of Dr Al-Sarraj, that it could be dated back to 15 February 2005. After the trial she made a further report, although she accepted her lack of expertise in that area.
67. The difficulty with any theory of injury caused on 15 February 2005 is the evidence of Mr Elston, Mr Peter Richards and Dr Al-Sarraj that the axonal injury was not survivable and that the retinal haemorrhage and folds would have caused blindness. Thus the evidence of the appellant, as we have already pointed out, was wholly inconsistent with an earlier injury followed by a lucid interval.
68. The appellant did not pursue this line of argument and was thus left with the evidence of Professor Luthert and Dr Leestma and with a third expert whom she indicated

would be called, Professor Morris, a General Histopathologist. Professor Morris was relied upon both before and during the course of the appeal to suggest either some unknown infection or restoration of the circulation following cessation of breathing (reperfusion). The prosecution were prepared to call Professor Klein, Professor of Infectious Diseases and Immunology at the Institute of Child Health, Professor Pollard, Professor of Paediatric Infection and Immunity at University of Oxford, who gave evidence at trial, and Dr Peters, the Paediatric Intensivist. There was a warning that further experts might be relied upon by the prosecution.

69. Professor Morris gave oral evidence to us which suggested that he was prepared to consider as a realistic possibility that an unknown infection had caused hypoxic-ischaemic damage and it is that which caused both the subdural haemorrhaging and the bilateral retinal haemorrhaging. He accepted that he was not qualified to deal with the ophthalmological symptoms nor the question of axonal injury. It appeared that Professor Morris was tending to revive the unified hypothesis in Geddes III which was rejected in *R v Harris*. Mr Topolski QC, rightly and with customary frankness, abandoned reliance upon Professor Morris and made it clear that he would not contend that infection was a cause of Maeve's collapse and death. But it is necessary, in the context of the other arguments which he maintained, to record some of the written evidence and oral evidence from, particularly, Dr Peters, which places the arguments on which the appellant did rely in the context of all the expert evidence which remained relevant in the appeal.
70. Professor Morris did not persist in contending that the explanation for Maeve's symptoms was reperfusion. But he would not accept that no causal link has ever been made between hypoxic-ischaemic injury and subdural haemorrhages or retinal haemorrhages. We refer again to Dr Peters' evidence of his clinical experience, recorded [53]. Moreover, there is ample literature to support his proposition that hypoxia does not cause subdural bleeding in infants (*Byard (2007) (Paediatric and Development Pathology 10)*). The paper considered a series of eighty-two fetuses of infants and toddlers up to 3 years who died following hypoxic cardiac arrest in which no single macroscopic subdural haemorrhage was detected. To similar effect was a study of critically ill children admitted to Great Ormond Street Hospital in *Jackman (2007)* and in a more recent paper, by *Matschke in Paediatrics (the American Academy of Paediatrics December 2009)* in which, following 715 autopsies of infants who had died before the age of 1 year from one institution over a fifty year period, the data argued strongly against the unified hypothesis proffered in Geddes III and "strengthens the association between subdural bleeding and non-accidental head injury in infancy" (page 1).
71. Any suggestion of septic shock, as a cause of the bilateral multi-layer retinal bleeding or retinal folds, was dismissed in the absence of any coagulation disturbance associated with such shock.
72. In his oral evidence Dr Peters again confirmed that there was no connection between the swelling of the brain and the apparent haemorrhaging in Maeve's case. The

timing in Maeve's case excluded the possibility that the brain-swelling caused the haemorrhage since such haemorrhages were observable early after four hours whereas the swelling would peak at between twenty-four to forty-eight hours after the crisis. The only time he had seen the pattern of events apparent in Maeve's case was following trauma. We refer to this evidence again because it seems to us to demonstrate the force of the triad in this case. The appearance of the symptoms, particularly the haemorrhaging at every level in the retina, coupled with the non-space-occupying subdural haematoma and the encephalopathy showed that this was a case where the triad itself afforded a strong basis for the conclusion that shaking or shaking and impact was the cause of Maeve's sudden collapse. Neither infection nor reperfusion, on the undisputed evidence both at trial and before this court, explained the retinal haemorrhages and folds or the subdural haematoma.

Conclusion

73. Accordingly, every effort has been made to explore every available avenue in an understandable attempt to undermine the safety of the jury's verdict.
74. We describe the attempts as understandable because there remains the unsolved mystery of how so admired a childminder as this appellant should have been responsible for the use of excessive force, even momentarily, when handling this baby. But that was a problem with which the jury had to grapple. There is no basis upon which this court can say that the jury was not entitled, after being properly directed by Keith J, to conclude that the expert evidence proved, beyond a reasonable doubt, that the defendant had shaken Maeve with excessive force.
75. There has never been an issue as to the degree of force used. It has never been suggested that if the defendant shook Maeve she may not have realised she was using excessive force.
76. We must also bear in mind the admirable directions given by Keith J. They were "directions", not an unstructured journey through the expert evidence. He made clear to the jury the issues which they had to decide and the rival bases on which they could reject or accept the evidence. The route the jury took to conviction is quite plain, on reading the summing-up.
77. In particular, the judge directed the jury as to the need to bear in mind that medical science in relation to non-accidental head injury is developing, and as the defence would have it, uncertain, and that it may not be possible to identify the cause of death. He gave a positive and correct direction that the jury had to consider whether the death may be unexplained. He directed them that they had to be sure that unexplained death could be excluded.

78. Further, it is of importance to note that he stressed that the defence was under no obligation to advance any particular theory as to cause of death and that rejection of the theory of spontaneous seizure did not of itself mean that the jury was required to accept the prosecution case and convict.
79. Accordingly, this is a case where the issue of unexplained cause in an area of developing medical science was properly laid before the jury. The justification for rejection of that possibility and for acceptance of the prosecution case is plain from the summing-up.
80. We remain concerned as to how the appellant finds herself in the unenviable position of seeking to establish that the jury's verdict was unsafe, having served the severe sentence of three years. But our sympathies for everyone concerned, Maeve's parents and the appellant, cannot subvert our function. We cannot substitute, for the jury's verdict, a conclusion based upon perplexity as to how this appellant could have treated Maeve with unlawful force, even momentarily. That was an issue which the jury resolved.
81. The evidence in the Bhatnagar paper was new. The fresh evidence was, in substance, not fresh evidence at all. The witnesses were "fresh", their evidence was not. It was evidence from experts in the same disciplines as those whose reports the defence had previously obtained but decided not to call. The appellant's previous representatives had chosen not to call the expert evidence because it helped the prosecution. That was a choice within the bounds of reasonable decision. If they had called that evidence and the jury had convicted, they would, probably, not have been permitted to adduce further evidence from experts in the same disciplines. They should not be in a better position because of a previous, sensible choice not to call the experts.
82. But, given the complex nature of this case, we prefer for the reasons given in *Kai-Whitewind* to deal with this case on the basis of the substance of the evidence we heard *de bene esse*. It would have been wrong to reject that evidence without hearing it, even though the witnesses came from the same areas of expertise as those previously available.
83. Now that we have heard that evidence, we conclude, for the reasons advanced earlier, that it does not undermine the safety of the verdict. Accordingly, we dismiss the appeal.

Butler

84. On the evening of the 15th February 2007 Ellie Butler, then aged about 7 weeks, was rushed to hospital by her father. Although she bore no outward mark of recent injury, it was in due course discovered that she had suffered a serious head injury. In

particular she had an encephalopathy, multiple subdural haemorrhages and multiple retinal haemorrhages –“the Triad”.

85. At the time of admission it was noticed that the child had injuries on the forehead and hand consistent with burns. Enquiries revealed that, on 7 February, Ellie, whilst in the care of her father, had rolled off a pillow and had come into contact with a radiator. The child had been taken to the general practitioner and treated and there had been no concern at the time that there were any child protection issues. These matters were reviewed in the aftermath of the head injury and he was charged.
86. At the end of a four week trial, in which the prosecution had called fifteen medical witnesses and the defendant three, the appellant was convicted of causing grievous bodily harm contrary to s.20 of The Offences Against the Person Act 1861 and cruelty contrary to S.1 of The Children and Young Persons Act 1933 and sentenced to concurrent terms of 18 months and 1 month imprisonment. He obtained leave to appeal against conviction from the Single Judge and was on October 2009 admitted to bail by a different constitution of this court.
87. The case was tried at Croydon Crown Court before His Honour Judge Timothy Stow Q.C. and a jury. The unusual feature of this case was the fact that, happily, Ellie had made a complete recovery from her head injury and, given the absence of either external sign of this injury or any other relevant injury, it was, therefore, a case to be tried almost entirely on the basis of medical evidence and opinion.
88. Thus it was that the decision to join the other incident and then to refuse to sever became more important than it might otherwise have been. The burning incident was comparatively trivial and was never suggested as having been deliberate; it was left to the jury on the basis of recklessness. Indeed, had it stood alone, there is every reason to think that it may not have been the subject of criminal prosecution at all and would have been viewed as a piece of “new parent carelessness”. It is true that the Judge gave a careful direction to treat them separately but the reality is that this incident provided the only concrete example of any misdoing by the appellant and in a case such as this, it was asking a great deal of the jury to ignore it when they were considering the head injury. This court will, of course, be very slow to interfere with the exercise of a trial Judge’s discretion in matters such as these but the risk referred to above left us with a sense of unease as to whether that Count should have been tried with Count 2.
89. As will inevitably happen in cases such as this, the medical evidence falls into a number of categories. There was the evidence of the treating doctors: A & E, the paediatricians and the radiologists. There is no doubt that that evidence established the existence of the triad of symptoms, excluded other common medical causes for all or any of those symptoms and concluded, as on the present state of medical knowledge they would be bound to conclude, that the evidence pointed to non-accidental head injury (NAHI). The second group of medical evidence related to

those experts who were asked to express a view as to causation, although the treating doctors were allowed to do so too.

90. That group yielded effectively three subdivisions: ophthalmology, paediatric neurology and paediatric neuroradiology. The prosecution also adduced the evidence of Dr Lloyd, a consultant paediatrician, to give an overview but although his views were highly contentious at trial, it is accepted that they cannot be decisive of the appeal and accordingly nothing more need be said of them here.
91. The ophthalmic evidence was broadly agreed as between the three witnesses. They agreed that there were severe retinal haemorrhages. They agreed that it pointed primarily to trauma. They agreed that it could not be explained by other medical causes known to them although not every ENT possibility could be wholly excluded. The original view was that it was caused by shaking causing shearing injuries which caused this type of haemorrhaging. That view, however, had to be revisited when, contrary to expectation, the haemorrhaging resolved without evidence of residual damage. It was their experience that it was very rare for such severe retinal haemorrhages to recover completely.
92. That recovery cast doubt on a severe shaking injury; indeed it told against a major shaking incident. The ophthalmologists would not exclude any shaking or other trauma and certainly Dr Gregson thought that causation would be the same for both retinal and subdural haemorrhages. There was no account of which they knew which explained the symptoms. Professor Taylor recognised that this was an unusual case, for the retinal haemorrhages were more consistent with raised venous pressure than shearing. In his view the chances were even between NAHI and unknown causes.
93. It will be readily apparent that this evidence does not sit comfortably with a suggested causation of shaking though it does not exclude it. It weakens the structure of the prosecution case.
94. The neurologists in the case did not add much since the essence of the case depended on the interpretation of CT and MRI scans. They were able to demonstrate that all other known medical causes had been excluded. They too were in the position of asserting that it was a shaking or an unknown cause case subject to one possible explanation favoured by one of the neuroradiologists but firmly rejected by the other.
95. There were three specialist paediatric neuroradiologists who gave evidence but the essence of the dispute was between two of them: Dr Stoodley, called by the prosecution, and Dr Anslow, called by the defence. Both these witnesses are of considerable standing in their field of paediatric neuroradiology and both have considerable forensic experience both in the criminal and in the family jurisdictions. It was the unenviable task of this jury not only to adjudicate as between their views

but to do so without any extraneous evidence to assist them and also having to take into account the uncertainties raised in the ophthalmic evidence.

96. There is no doubt that there was an encephalopathy and that there were subdural haemorrhages. There is no doubt that these haemorrhages were both in the front and the back of the brain. There is no doubt that there was fresh blood to be seen just as there was darker blood which may have been old blood or old blood mixed with fresh blood or fresh blood mixed with cerebrospinal fluid (CSF); on scan evidence alone what it was could not be said with certainty. There had to be a reason for the fresh blood. The experts at trial either agreed or accepted that the fresh blood was traumatic in origin. It was here that the primary controversy could be identified.
97. Dr Stoodley's view was that he was seeing either fresh blood or fresh blood mixed with CSF, that there was no evidence of impact trauma and in any event that the diffuse nature of the subdural haemorrhages was against there being a single site impact. He saw clear evidence of encephalopathy and deduced from all that that the most likely cause of the subdural haemorrhages was a shaking injury. He said (and in principle this was not controversial) that shaking does not always produce retinal haemorrhages though clearly he was reticent in his views as to their causation.
98. Dr Anslow believed that he saw old blood which he ascribed to a birth-related subdural haemorrhage. He pointed to a study that demonstrated up to 46% of uneventful births in fact produced subdural haemorrhages. Whilst many cleared spontaneously in a month, it was his view that the evidence did not permit the conclusion that they all did. He acknowledged that the fresh blood required a traumatic origin but was of the view that it was impossible to assert what degree of force would be required if this, rather than a new injury, was a re-bleed from a chronic birth-related subdural haemorrhage. In acknowledging both that the research showed that birth related subdural haemorrhages were confined to the back of the head and also that there was fresh blood at the front of the brain, he explained that blood could move within the brain area.
99. That last view was vigorously contested by Dr Stoodley who asserted that in circumstances such as these, blood would not move from front to back and in that he was supported by the neurosurgeon Dr Richards. Whilst Dr Stoodley acknowledged the possibility of birth-related subdural haemorrhages (especially where, as here, there had been a ventouse delivery), he said that they usually resolved in a month and that these subdural haemorrhages were most unlikely to be birth-related.
100. 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 e.g. 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 that 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.

101. The buggy ride also raises another discrete but important issue in the case, namely that of timing. The preponderance of view was that the precipitating event (whatever it was) came just before the child went floppy. That, of course, is the only evidence that established that that event occurred whilst the child was in the care of her father. If that were not so, there was no case against him on the major count. Whilst Dr Jayamohan (a neurologist) thought that the event might have occurred before arrival at the father's home, the preponderance of view is that the father's account is consistent with the event which caused collapse happening just before the child went floppy.
102. On the basis of that evidence, we turn to consider the approach of the Judge to a submission of no case to answer. There were three essential features of the evidence on which he had to focus in order to determine whether a reasonable jury properly directed could have convicted.
103. First, in favour of the prosecution there was the evidence of Dr Stoodley, supported by the neurologists' evidence which excluded all known natural causes. It was open to the jury to reject the evidence of Dr Anslow in relation to re-bleeding, the movement of the blood and the buggy ride explanation.
104. Second, however, there was the ophthalmological evidence. It is true that the ophthalmologists did not specifically eliminate impact as a cause but all the evidence showed that if the retinal haemorrhages had been caused by severe shaking, as the prosecution suggested, the full recovery which in fact occurred would not have been expected. It was powerful evidence which told against shaking and in favour of an unknown cause.
105. Third, the jury would have to exclude an unknown cause. A jury would have had to acknowledge the imperfectly understood.
106. The Judge correctly noted that the jury would have to look at the evidence as a whole. He acknowledged that the ophthalmological evidence weakened the triad. He seemed to take the view that that weakening was offset by the evidence of subdural haemorrhage.
107. This, in our view, was not correct. Recovery demonstrated that the retinal haemorrhages could not be relied upon as evidence of shaking. On the contrary they

were evidence of an unknown cause, as Professor Taylor said. Recovery is unusual and casts doubt on the reliance which can be placed upon the “triad” at all. Of course there are cases of non-accidental head injury where some or all of the components of the triad are absent. But there must be a rational basis upon which the jury could conclude that collapse was not due to an unknown cause, and thus reach a conclusion which rejects the evidential weight to be placed on the unusual feature in this case, namely complete recovery.

108. Nowhere in his ruling did the Judge fully acknowledge the weight to be attached to the uncontradicted ophthalmological evidence. Nowhere did he identify the basis upon which the jury could reject the possibility of an unknown cause. He failed, in fact, to recognise the force of the second and third features of this evidence.
109. We repeat that the absence of one or more features of the triad does not exclude a conclusion of NAHI. The other evidence may be so compelling that it excludes an unknown cause and proves the unlawful violence alleged.
110. But not in this case. We conclude that there was no rational basis on which a jury, in the light of the ophthalmological evidence, could reject an unknown cause. Once that is acknowledged as a realistic possibility, the weight to be attached to the evidence of Dr Stoodley is itself diminished.
111. In those circumstances we allow this appeal and quash the conviction on Count 2.
112. We should add this. The summing-up contained serious misdirections. Those misdirections provide cogent support for the conclusion we have reached.
113. The Judge did not sufficiently direct the jury as to the importance of the ophthalmological evidence and its effect in undermining the triad as evidence of NAHI and as supporting an unknown cause. It was incumbent upon him to have done so. No proper direction was given to the jury that they must consider the possibility of an unknown cause, particularly in the light of the ophthalmological evidence, and should only convict if they reject it.
114. The summing-up was also defective in its structure. The Judge carefully and conscientiously recited long passages of the expert evidence to the jury. But he did not ‘direct’ the jury at all. He recited those passages in the chronological order in which the evidence was given. That was of little assistance to the jury.
115. The jury required a careful direction as to the essential issues which they had to determine and a reminder of that evidence and only that evidence which went to those issues. It was necessary to deal with that evidence issue by issue.

116. The Judge did not have the assistance of *R v Schmidt* [2009] EWHC Crim 838 in which a summing-up similar to that in this case led to the quashing of the conviction [42] and [47]. Nor did he have the assistance of the current Judicial Studies Board Crown Court Bench Book March 2010, Chapter 8. We have only drawn attention to the summing-up because it assists in emphasising the need to construct a summing-up to meet the complexities of cases such as these and to ensure the case proceeds on a logical basis. It is only fair to the Judge to state that throughout the trial and the summing-up the Judge exhibited conspicuous care and fairness.
117. We think that the conviction on Count 4 should be quashed as well. We are uneasy (for the reasons given above) that this was left to the jury with Count 2. We do not think that it would have been prosecuted had it stood alone. The judge recognised this in his ruling. We also doubt whether this is a case of recklessness. We can discern no basis in the evidence upon which it could be said that this father should have realised this baby (aged 7 weeks or so) was likely to roll without the father (who was on his own) doing something. In those circumstances we have concluded that this count too should have been withdrawn from the jury. The appeal is allowed and both convictions are quashed.
118. We do not need to address the fresh pathological evidence sought to be adduced by the appellant. That evidence is highly contentious. We did not expressly consider the application to call fresh evidence nor do we express any other view about it.

Oyediran

Introduction

119. Oladapo Oyediran, the appellant, appeals against his conviction on the 16 March 2007 for the murder of his 10-week old son Oluwafemi, “Femi” following a trial at the Central Criminal Court before HH Judge Focke QC and a jury. The trial took place between 22 January 2007 and 16 March 2007. The appellant was tried on a two count indictment. In addition to the murder count, he was charged with causing or allowing the death of a child contrary to section 5 of the Domestic Violence Crimes and Victims Act 2004. His co-defendant on the section 5 charge was the baby’s mother, Sophia Rudder. She had originally been charged with murder. Because she suffered from multiple sclerosis, the Crown decided not to proceed against her on that charge. She was acquitted by the jury of the section 5 charge, and has since died. The appellant was sentenced to life imprisonment with a minimum term of 13 years.
120. The prosecution alleged that the appellant murdered his son, aged 2 months and 10 days, on 18 October 2005, by inflicting head injuries on him, resulting in brain damage which led to his death. The head injuries had been caused by a “rotational acceleration/deceleration mechanism”, either shaking or throwing or a combination of both. The cause of death was the brain damage. The mechanism of death was

inhalation of the gastric content of the baby's stomach. The brain damage had affected his gag reflex, and he had therefore inhaled his stomach's content.

121. It was alleged that there were two incidents which resulted in the brain damage discovered at post mortem; one which had occurred about two weeks before death, and one about two to three days before death. After death, Femi was also discovered to have had an unusual fracture of his arm which it was agreed must have happened between two and four weeks before his death. The arm fracture was important evidence in support of the prosecution's case that the baby's head injuries were not accidental.
122. It was the defence case at trial that the appellant did not have any tendency to violence. He did not know how the injuries were caused but the medical evidence was not conclusive of non-accidental injury, Femi could have been dropped by his mother, because of her multiple sclerosis and this could have been the cause of the injuries which led to his death.
123. The appellant now applies pursuant to section 23 of the Criminal Appeal Act 1968 for permission to call and rely upon the evidence of two experts who were not called at the trial: (i) Dr Waney Squier, a Consultant Neuropathologist; and (ii) Dr Michael David Jones, an expert Biomechanical Engineer. It is said their evidence provides grounds for allowing the appeal because it demonstrates the first brain injury was more likely to be the result of an accident, rather than shaking, predisposing Femi to the risk of a further accidental trauma, that is, the second brain injury.
124. In his written notice of appeal, the appellant asks for permission to rely on a transcript of a meeting held between experts for the Crown and the defence in a different case (*R v Kendrick*) on 15 December 2007 to show that evidence given by Professor Risdon and Dr Richards at trial had been contradicted by them at the meeting. This ground was, rightly, not pursued.
125. At the hearing of this appeal, we heard evidence from Dr Squier and Dr Jones without determining whether their evidence should be admitted as fresh evidence. We also heard evidence from Dr Al-Sarraj a Consultant Neuropathologist who was called by the Crown. He produced a report dated 23 February 2010 in response to the fresh expert evidence from Dr Squier relied on by the appellant. Dr Al-Sarraj had provided a report for the appellant in April 2006 for use at the trial, but in the event, a decision was made not to call him. We also had before us a written response to Dr Squier's report from Dr Richards.

The Evidence at Trial

126. Femi was born on 8 August 2005. He was the son of the appellant and Sophia Rudder, who became partners at some point in 2000. They also had a daughter who

was born on 20 May 2003. At the time of Femi's death, they all lived together in a flat in Battersea.

127. Sophia Rudder had been diagnosed with MS in 1994 and was quite severely disabled by her illness. The appellant was her carer as well as her partner. Dr Oliver Foster, a Consultant Neurologist, treated her for MS for many years. He said that her condition was moderately severe. She had numerous attacks which principally affected her balance, manual dexterity, clarity of speech and eye movements. Her ability to walk was significantly restricted; she had difficulty in walking up stairs.
128. Dr Foster, who saw her on 24 February 2006, said that she would have been able to pick up either child, but might have dropped them. She was in the more severe category of multiple sclerosis sufferers. She was a vulnerable person and would not be able to stand up to someone easily. It would have been difficult for her to pick up the baby and shake it, and extremely difficult for her to have twisted his arm. She lacked the power and co-ordination. He found her to be a mild-mannered and passive person. It was unlikely she would resort to violence.
129. Dr Edwards, a Consultant Psychiatrist who interviewed Sophia Rudder in January 2006, gave evidence about her mental state and understanding. She was classified as having a learning disability. In January 2006 her mood appeared inappropriately calm and detached. She appeared to be like a bewildered child. She took the view the appellant was a good father and would never intentionally hurt their son. She denied shaking him and said she did not know how he had been injured. She said he had fallen from a sofa. She had left him on a bed and found him on the floor. Their daughter might have dropped him.
130. There was evidence that Sophia Rudder became isolated from her friends and family as a result of her relationship with the appellant, and that she was extremely vulnerable. Claire Fairman from the Battersea Field Medical Practice, who had known Sophia Rudder for 14 years, said that after her relationship with the appellant, she "no longer spoke to us and appeared to be submissive." After the birth of their daughter, the appellant brought her in for most of the appointments. She felt the appellant had a demanding attitude, and on one occasion barged into a consulting room. He could be impatient and aggressive. Geoffrey Vevers, the Vicar at her church had known her since 1998. He became concerned about the appellant's behaviour as he appeared to want to isolate her from other members of the church; he was controlling and talked over her. Diane Cole, her sister, said the appellant tried to isolate Sophia Rudder from her family.
131. When Jane Foster (a revenue officer with the Housing Association for the property of which Sophia Rudder was the tenant) spoke to her in 2004 on the telephone, the appellant intervened to say that he was her financial adviser and everything she had to say to her she could say to him. The appellant called himself Dr Dapo. Telephone messages left on the appellant's mobile and correspondence about rent arrears went

unanswered. Eventually, in May 2005, possession proceedings for non-payment of rent were begun.

132. After eviction, Sophia Rudder, by then heavily pregnant, was placed with her daughter in temporary bed and breakfast accommodation, where the appellant sometimes stayed too. There, Femi was born (on 8 August 2005); he was delivered by the appellant. Ms Thomas, the paramedic who attended, noticed that Sophia Rudder had some difficulty in holding her son.
133. On 10 August 2005 she and her new-born son returned to the temporary accommodation. On 11 August 2007 Sophia Rudder was visited by Josie Slade, a community midwife, who was concerned about her ability to look after her son on her own, in particular about her lack of upper body strength and ability to hold him. The midwife said Sophia Rudder struggled to hold her son while trying to breast feed and was unable to pick him up. As a result of these concerns, Social Services arranged for the appellant to join Sophia Rudder in the temporary accommodation, to help care for his son. There was evidence from the health visitors of the appellant's good care of Femi and his pride in having a son.
134. In July 2005 the appellant told Ms Natalie Maitre, the Homeless Prevention officer from the Wandsworth Homeless Prevention Unit, that he was a doctor and worked in neurology. He appeared forceful, and his partner seemed simply to follow his advice. He told Ms Maitre that people with neurological problems were better off with short hair and he had therefore shaved Sophia Rudder's head. At Ms Maitre's instigation, solicitors Flack & Co were instructed to act for Sophia Rudder. Ms Maitre's letter to them said the appellant had damaged relationships with all the agencies trying to help Sophia Rudder through his attitude and behaviour. However, as a result of her illness he was the only one trying to help her, and to some extent she had abdicated responsibility to him.
135. Jane Pritchard from Flack & Co said Sophia Rudder was extremely vulnerable. She appeared emotionally detached. It was like dealing with a child. She told Ms Pritchard that the appellant was a doctor. Ms Pritchard commissioned a psychiatric report which said Sophia Rudder was incapable of looking after her financial affairs. As a result of that information, and intervention, Sophia Rudder was allowed back to the flat, and went back there, with the appellant and the children, on 11 October 2005.
136. On 6 October 2005 Femi was seen by a Dr Sathananthan at the Brigstock clinic. The appellant said Femi had been "very snuffly" for the past three days and not feeding. On 18 October 2005, the appellant, accompanied by Sophia Rudder and her daughter, took Femi to the Battersea Fields Medical Practice. They arrived at about 11.10 a.m. Femi was in a car seat which the appellant placed on the reception desk. The appellant told a receptionist, Sabrina Newton, that he needed to see a doctor for his baby. Ms Newton thought the appellant was pretty calm. She noticed mucus round Femi's nose, and that he was apparently asleep. She said the appellant told her he had

fed the baby that morning, had left the baby and gone out (for nappies and formula milk amongst other things) and on his return found that milk was coming from Femi's nose and mouth.

137. Femi was not registered at the Surgery so registration forms were given to Sophia Rudder. They were filled in by the appellant. He told another receptionist, Ms Fairman, he had fed the baby at 7.30 a.m., and had gone to Boots for nappies and other products. The appellant then lifted Femi's nose with one of his fingers to show the receptionists the discharge, and said: "I only brought the baby in because I didn't want to be accused of negligence." Ms Fairman thought the appellant was calm. She thought she heard the baby snuffle. Another receptionist, Ms Muldoon, said the baby was just like a doll and fast asleep. She thought the appellant was impatient to have the baby seen.
138. Femi was seen by Dr Kate Trevelyan-Thomas, at 11.44 a.m. The appellant carried Femi in his car seat. The first thing he did was to express concern about the wait to see the doctor and the appointment system. She said his attitude was somewhat aggressive. When Dr Trevelyan-Jones saw Femi, she immediately realised he was dead. He was not moving or breathing. He was white (for a baby whose colour was black), floppy and cold. She asked a nurse to call 999. She noted a purple discolouration to his flank which she took to be post mortem changes. In her view Femi had been dead for some time. When she told the appellant Femi was dead, he said he did not believe her. He said she must be joking: "resuscitate it". She said there was no point as the baby had been dead too long. The appellant she said was unbelieving, and kept trying to wake Femi up.
139. She said Femi appeared to be a well-nourished and completely normal 2 month-old baby; with no sign of damage. Dr Trevelyan-Thomas tried to take a history from the parents. The appellant told her he had fed Femi at 7.30 a.m.; he had taken a 9 ounce bottle and was cheerful and active. The appellant had gone to Boots just before 9 a.m., had got home at about 9.30 a.m. and noticed milk coming out.
140. Dr Trevelyan-Jones asked Dr Emma Griffin, another doctor at the Surgery, to examine Femi. It was also Dr Griffin's view that Femi had been dead for some time, and she pronounced him dead at 11.53a.m. The nurse who made the 999 call told the operator the doctor had said the baby had been dead for half an hour. But in the opinion of both Dr Trevelyan-Thomas and Dr Griffin Femi had probably died before he arrived at the Surgery.
141. Femi was taken by ambulance to St George's Hospital. The appellant told Janet Drew, a member of the ambulance team, that Femi had not cried that morning, but liquid had been coming out of his nose. He sat the baby up and went to get some nappies. He decided to take Femi to the hospital on his return (though in cross-examination she accepted it may not have been in that order).

142. At the hospital the appellant repeated the account of the morning's events to Claire Hill, a Paediatric Registrar, adding that at 11 p.m. the previous evening he had fed Femi 4 ounces of pineapple juice. He then gave Femi some formula milk at 4.30 a.m. Only the appellant answered Dr Hill's questions. Both parents consented to a post mortem and she said they appeared upset but calm. The appellant said he wanted to know the cause of death.
143. P.C. Butler also spoke to the appellant at the hospital. The appellant said he fed Femi at 8.15 a.m., he drank the milk and coughed, and the appellant took it away. Femi kept burping so he gave Femi some more. He went out of the room and when he came back there was milk running out of Femi's nose. He dabbed Femi's nose and put him in a car seat in his sister's room. He left at 8.45 a.m. to go to Boots to buy some nappies. He then went to Lidl, and then to see his mother and another lady. Sophia Rudder told him Femi still had milk coming from his nose so he decided to take Femi to the clinic. She nodded from time to time as the appellant gave this history. The appellant pushed past the officer and into the resuscitation room. They struggled, then the officer took the appellant to see the baby. He appeared to be looking for injuries, and broke down crying.
144. The appellant and Sophia Rudder were arrested on the 19 October 2005 after a post mortem was conducted on Femi by Professor Risdon. They both declined to answer questions in interview but relied on prepared statements. In his statement, the appellant denied responsibility for Femi's death and questioned the injuries described in the disclosure. He asked for an independent autopsy.
145. The prosecution called evidence of an alleged "cell confession" made by the appellant to Enrico Escoffery; one of the grounds of appeal relates to this confession and we shall deal with that evidence later.
146. The prosecution called five expert medical witnesses: Professor Risdon, a Consultant Histopathologist; Dr Harding, a Consultant Paediatric Neuropathologist; Professor Luthert, a Professor of Pathology and Consultant Ophthalmologist; Professor Hall, a Consultant Paediatric Radiologist and Dr Richards, a Consultant Paediatric Neurosurgeon. The Defence called Dr Rouse, a Home Office Pathologist.
147. There was substantial agreement between the experts as to the injuries found and the mechanism of death. The main areas of challenge on behalf of the appellant were (i) whether Femi's head injuries could have resulted from an accidental short fall – and in particular, if Femi had been dropped by Sophia Rudder and/or caught and pulled back violently; (ii) whether what the experts described as the second brain injury was in fact not a separate injury, but a re-bleed (or secondary bleed) from an earlier brain injury; and (iii) whether the arm fracture revealed in a post-mortem radiological skeletal survey could have been caused by accident, for example, if Femi were

grasped in an attempt to catch him after he had been dropped. There was a fracture at the lower end of the left humerus just above the elbow joint.

148. On 19 October 2005, Professor Risdon performed the post mortem on Femi. Professor Risdon found Femi to be a well-nourished infant with no visible external injuries. He was not dehydrated. The stomach contained a little mucus and milk curd, consistent with a milk feed a few hours before death. He had a small bruise on the inner surface of the scalp over the right parietal region. There was a degree of swelling to the brain. There was a thin layer of fresh subdural haemorrhage mainly over the right side of the brain and fresh haemorrhage around both optic nerves. In his opinion, Femi had suffered a head injury characterised by a subdural haemorrhage, brain swelling and haemorrhage around the optic nerves.
149. In Professor Risdon's opinion, by far the most likely explanation was that the injuries were inflicted, not accidental, in the absence of any plausible clinical history to account for the injuries. The combination of subdural haemorrhage and brain swelling indicated a rotational acceleration/deceleration mechanism for the head injury. Such a movement would cause the delicate veins between the surface of the brain and the dura to stretch; and if the movement was violent enough, to break. Rotational acceleration/deceleration was characterised by a subdural haemorrhage, brain swelling and retinal haemorrhage (the "triad of findings"). Here there was no retinal haemorrhage. He was not surprised to find fresh bleeding round the optic nerve because it was an extension of the subdural space. The mechanism of death was aspiration of the stomach's gastric content, caused by the brain damage which affected the nerves controlling the reflex gag action. Death would have been fairly quick. Femi was cold to the touch at the Surgery, and this was consistent with Femi having been dead for at least half an hour. In his opinion, Femi had been dead for a little while, and had not died in the Surgery, though in cross-examination he said he could not exclude that possibility.
150. In cross-examination Professor Risdon confirmed that his findings demonstrated a pattern of injury that was almost always non-accidental. He said it was not impossible to produce a fatal fall from a short distance, but the characteristics of such an injury would be different from the ones he found. The fracture of the humerus and the first head injury could have occurred at the same time but he rejected as "implausible" the suggestion that those injuries could have been caused by a child falling from a parent's lap and being pulled backwards, even violently. The head injury could not have been accidental; the fracture could have been accidental theoretically, but such fractures do not occur in the way suggested. He said the older subdural haemorrhage was not the type he would associate with one caused at birth. Professor Risdon rejected the suggestion that there had been a "re-bleed" or secondary bleeding into a pre-existing site of brain injury rather than a second brain injury.
151. In re-examination he said the subdural haemorrhage was an extremely thin layer: the possibility of re-bleeding into that was obviously small. If there had been a re-bleed into a pre-existing injury he would have expected a volume of blood sufficient to

change the clinical condition of the child, and there was no evidence of that. As for short distance falls causing serious injury, they nearly always occurred in ambulant children, the children in a study by Plunkett were not individually studied, and there was no individual examination of the injuries caused. He had experience of three fatalities from short distance falls: and each involved older children who suffered a large, extra-dural, space-occupying haemorrhage, a quite different lesion from the one here. Short falls for young children were everyday events. The vast majority produced no injury at all, and certainly not damage inside the skull.

152. Dr Rouse confirmed Professor Risdon's post mortem findings. He agreed the head injury was caused on two separate occasions and that the fracture had been caused two weeks prior to birth. The head injury could have been caused at different times or the same time. It was not possible in his opinion to say whether the fracture had been caused accidentally or non-accidentally. As there was no retinal bleeding this tended to suggest the force used was less than that found in traditional "shaken baby" cases. If a child fell and were grabbed suddenly, that sort of force might produce damage to the brain. As the brain injury had not been instantly fatal and in the absence of retinal haemorrhage, the brain injury could have been caused by a short fall from shoulder height to the ground. Where a child had a two-week old brain injury any additional impact to the head, for example, a short fall, could lead to an increased propensity to bleed.
153. He agreed it was not necessary to have retinal haemorrhaging for there to have been a rotational acceleration/deceleration mechanism, and that a short fall from a bed would not replicate such a mechanism unless there were a whiplash effect.
154. Dr Harding had examined Femi's brain. He confirmed there was mild swelling of the brain and evidence of brain damage of two different ages: one of two-three days before death in the cerebellum and one of two weeks old in the cerebral cortex. In his opinion the injuries were not accidental. The damage to the cerebral cortex was extensive and very severe. There was damage to the hippocampus which was more difficult to date, but in his opinion was probably caused at the same time as the damage to the cerebral cortex, as the two areas are closely related. There was damage to the thalamus which may have been secondary to the damage to the cerebral cortex. In the dura he also found haemorrhage of two different ages: a recent subdural haemorrhage which had occurred two-three days before death; and close to it, an older haemorrhage which was at least two weeks old. In his opinion, the haemorrhages would both have been caused by a torsional twisting injury that might damage the communicating veins. He also found axonal damage - that is, damage to some of the nerves in the spinal cord - which was the same age as the second brain injury, that is, two-three days old; and similar damage in the medulla (the lowest part of the brain above the spinal cord).
155. In cross-examination he did not agree the second bleed was a re-bleed as there were two "quite distinct lesions". He said there was no good evidence for the suggestion that when there has been one subdural haematoma it is easy for a second to occur. He

was asked about short fall injuries. He said a short fall would not result in the type of injury Femi had. A short fall injury would usually result in a different type of haematoma from the one found here: he would expect to see an impact on the skull, arterial bleeding, and an extra-dural haematoma as well a subdural haemorrhage.

156. Dr Richards gave evidence about the effect of brain injuries on live infants. He said a child with a brain injury of two weeks' duration would have abnormal movement of limbs, their behaviour would be abnormal, they would usually have seizures, their cry would be high-pitched and their feeding would be very weak. They would barely be able to suck. It was inconceivable that Femi had taken a 9 ounce feed a few hours before death. It was nonetheless possible for there to be milky fluid in the stomach, as the baby could still suck, but this would be very weak. It was also inconceivable that a half-competent GP would not have recognised the symptoms when he saw the baby twelve days before its death. He did not think, therefore, the baby could have been injured at that point. A fresh haemorrhage two-three days before death would have caused crying because of the severe headache it would have caused, unless the baby was unconscious. He expected the baby would have been unconscious at some point in the two-week period after it was injured. Professor Risdon's evidence in relation to the arm fracture accorded with the evidence of Professor Hall. Dr Rouse accepted that the fracture had been caused some two weeks before death and made it more likely that the head injury was not accidental.
157. Professor Luthert had examined Femi's eyes. He confirmed the absence of retinal haemorrhages but the presence of blood on the optic nerves. He said this can be caused when intra-cranial pressure increases due to the brain swelling. The blood had been there more than forty-eight hours before death; he could not say whether it had been there two-three weeks before death or exclude the possibility it was there at the time of birth. But it was much more likely that the bleedings resulted from the second injury. In his experience, this was inflicted trauma.
158. Professor Hall gave evidence about the arm fracture. She said it was a fracture of the lateral condyle of the humerus. It was at least two weeks old, and not more than four weeks (probably nearer to four weeks). It was an unusual and quite serious fracture in this age of baby. Such a fracture can be caused by a direct blow on to the bent elbow (for example, if the baby is thrown on to the floor) or by a forceful extension of the elbow. It was more common in older children as it was caused by high velocity impacts such as falling from skateboards. It could not be caused by shaking. Shaking might cause a metaphysial fracture which was a more common child abuse injury, caused by a grip, pull or twist.
159. She said the fracture would result in immediate pain of at least ten minutes and up to half an hour, and would result in the baby screaming. The cry would be a hard cry, or a cry of pain and recognisably different from a baby's normal cry. It would be immediately apparent there was something wrong with the baby. This type of crying would re-occur every time the baby or its arm was handled in any way. This ongoing pain would last for about a week. In addition, the baby would choose not to use its

arm, and it would appear to be limp for about a week. Swelling would start immediately. The fracture would have been caused by excessive and unusual force: something more than heavy-handed or rough handling. It could not have been caused by an over-extension of the arm while dressing. If the signs of the fracture had disappeared when Femi was seen by the GP (on 6 October 2005) the fracture would have had to have occurred a week before the visit, but a GP might not pick up a small swelling on the arm.

160. The appellant gave evidence in his own defence. Sophia Rudder did not give evidence. The appellant said he would have done anything to protect Femi. He said he took him to a GP on 6 October 2005 as he had a snuffly nose and it was obvious he needed a doctor. Four days later they moved to the flat. Femi had been with the appellant's mother for four days, and the appellant picked him up from her on 15 October 2005: he did not appear to be different from how he was before.
161. He said Femi had none of the symptoms of the fracture described by the expert evidence. He did not know how the injuries had been caused and he did not cause them. He described an incident when he tripped over while holding him, but he did not hit anything and could not have been injured. He also described an incident two days before Femi's death when he left him propped up on the bed, then heard a loud bang and found him lying on the floor. His daughter was by the side of the bed. He said Femi was asleep and ok. Perhaps with hindsight he was knocked out, but he was still breathing. He said he did not think it important to tell the police about this.
162. During the early hours of 18 October 2005, between 4 a.m. and 4.30 a.m. he said he heard Femi crying. He fed him some formula milk and went back to bed. He woke at 7.30 a.m. and Femi was crying. He went to Boots to buy nappies just after 9 a.m., leaving Femi strapped in a car seat in the flat. He went to Lidl and then to see his mother. He returned home at 10.00 a.m. Sophia Rudder said: "He's been coming down with milk". He had seen milk coming out of Femi's nose before, when they were in temporary accommodation, but this time there was more. He decided to take Femi to the Surgery at 11 a.m. He denied telling Escoffrey he was responsible for Femi's death. He denied telling anyone he was a doctor. He denied threatening Ms Foster with a walking stick. She was with them at the flat for only 5 minutes. Until the night of the 17 October 2005 he saw nothing in relation to Femi that would have caused him any alarm. There were no symptoms.

Grounds of Appeal:

163. There are two grounds of appeal in respect of which permission has been given and which do not concern the medical evidence. First, it is contended that the judge wrongly admitted the evidence of Jane Foster as to her visit on 23 May 2005. The evidence was admitted pursuant to s.101(1)(c) of the Criminal Justice Act 2003 on the basis that it was important explanatory evidence as to the mental capacity of Sophia

Rudder. It demonstrated, so the prosecution contended, that she had the capacity to intervene when the appellant exhibited signs of aggression towards Ms Foster.

164. Ms Foster visited the flat on 23 May 2005 to see whether it had been abandoned or, if not, whether even at this late stage the Association could offer some help. She described “an extremely unpleasant” incident after she had seen Sophia Rudder through the open bedroom door, and had spoken to her. During the incident the appellant roughly handled his partner. He also pushed Ms Foster, verbally abused her, threatened her with a walking stick, and prevented her from leaving the flat. He eventually let her leave after Sophia Rudder told the appellant in a quiet voice to “Let her go.” After she left, Ms Foster immediately dialled 999 and reported the incident to the police. At the time Ms Foster noted that “I fear that Ms Rudder and child are at risk and need Social Services assistance immediately.”
165. Mr Valios QC, on behalf of the appellant, contended that the evidence was so prejudicial as against the appellant and of such little significance in relation to the behaviour of his co-defendant, Miss Rudder, that the judge ought either to have rejected its admissibility under s.101(1)(c) on the basis that it could not be said that the jury would find it impossible or difficult properly to understand other evidence in the case in relation to Miss Rudder’s capacity (s.102(a)) or on the basis that it would be unfair to admit it since its prejudicial effect on the jury’s view of the appellant far outweighed its utility (s.78 PACE).
166. The prosecution, at the stage it sought to adduce this evidence, appreciated that it was unlikely that the co-defendant would give evidence. Moreover, the jury was to hear a considerable body of evidence about her mental and physical capacity, all of which might have suggested that she would have been unable to appreciate the risk to her son. In those circumstances it was the only occasion on which the prosecution was in a position to call direct independent evidence as to Miss Rudder’s behaviour in the face of her concern as to the risk of violence towards Jane Foster.
167. We accept that the evidence had no relevance as to whether or not the appellant was violent towards his son. There was a body of evidence as to aggressive and forceful behaviour by the appellant, particularly when confronted by those he would have regarded as being in an official position, such as the Homeless Prevention Officer. We recall his behaviour in the surgery on 18 October 2005. But it must have been obvious to the jury that the incident concerning Miss Foster did not demonstrate any violence or aggression on the part of the appellant against his own son. The judge reminded the jury as to why it was admitted, namely, on the question as to whether the defendant Sophia Rudder was able to appreciate risk, and emphasised to the jury that the incident had no relevance at all as to whether or not the appellant had a propensity to be violent.
168. In our judgment, the decision to admit evidence demonstrating Miss Rudder’s capacity to react to the appellant’s behaviour was relevant to an important matter in

issue between her and the prosecution. Since it was the only direct evidence it was within the bounds of reasonable conclusion for the judge to hold that no injustice would be caused to this appellant by admitting the evidence.

169. In so concluding, we have not found it necessary to consider whether the evidence did fall within s.98. The evidence was only evidence of misconduct or disposition towards misconduct on the part of the appellant and not on the part of the defendant Sophia Rudder. If Sophia Rudder had been tried on her own, the evidence would plainly have been admissible without any need to invoke Chapter I of Part II of the 2003 Act. But we need not dwell on this aspect since, on the assumption (which we need not decide) that the evidence did fall within that Chapter, it was properly admitted by the judge.
170. The second ground concerns the evidence of a cell confession to Escoffrey, a serving prisoner. Escoffrey shared a cell with the appellant for just under 4 weeks (between 22 October 2005 and 17 November 2005) while the appellant was on remand at Wandsworth Prison. He had a number of convictions including for false imprisonment, sexual assault, theft and harassment.
171. According to Escoffrey, the appellant first spoke about his case about four days after they started sharing a cell. He initially denied the offence. He said he had gone to town to buy Pampers for Femi, returned, and together with his daughter, bathed Femi. He then realised Femi was ill, though he did not know why. He took Femi to see the doctor, but was made to wait (for either 45 minutes or 2 hours) during which time Femi died. He blamed the Surgery for Femi's death.
172. However, about a week and a half later, after a visit from his young daughter (brought to the prison by Social Services) Escoffrey said the appellant confessed to killing Femi; he said he was often stressed and would then shake Femi (and Escoffrey demonstrated a forwards and backwards movement) while holding him upside down by his ankles. He said he had done this on the morning of Femi's death. Escoffrey said the appellant said he thought he would get away with it as the doctors did not know what they were talking about.
173. The defence were able to advance a number of substantial grounds to demonstrate the unreliability of Escoffrey. He had a number of convictions. The details of the confession which he said he had heard were inconsistent with the prosecution case. According to Escoffrey, the child was aged 3-4 years, whereas he was much younger. The appellant had apparently spoken of a twisted bone to the police. According to Escoffrey, the appellant had said that his daughter was bathing his son and that he had been out shopping. He said that shaking had occurred after he returned from the shop. None of this bears any relationship to what the appellant in fact said about the circumstances of his son's collapse.

174. Of particular importance is the timing of the alleged confession. Escoffrey said that the confession was at about 6.00-7.00 p.m., following a visit by the appellant's daughter, accompanied by a member of social services. He said that the appellant had confessed to taking the child by the legs, holding him upside down, and shaking him. He had confessed to doing this "all the time". In cross-examination he confirmed that the confession was in the evening after the visit, although he had told the police it was in the middle of the night.
175. The evidence of the prison officer, Miss Atkins, was that Escoffrey reported the confession to her in her office at 10.00 a.m. on 11 November. This preceded the social services visit. It thus demonstrated that Escoffrey's account of the surrounding circumstances of the confession was untrue.
176. There was no transcript of the prosecution's closing submissions, but we accept that, in effect, it abandoned reliance upon Escoffrey. In those circumstances, the appellant submits that it was wrong of the judge to leave his evidence for the jury to consider. In his summing-up the judge reminded the jury of the discrepancies between what Escoffrey said and what the prosecution alleged the appellant did to his son. He reminded the jury of the discrepancies as to the circumstances in which Escoffrey said the confession had been made to him. But he did leave the matter to the jury in terms that if they were not sure that the confession was true, they should disregard it. But if, conversely, they were sure it was true, then they could rely upon it.
177. Had we been hearing the trial, we would probably have directed the jury to disregard the evidence. It was so flawed as to be of little value. But the very fact that it was so obviously unreliable can only have assisted the appellant. It was admissible and we are unable to say that the judge erred in allowing the evidence to be given, in failing to give a stronger warning to the jury, or that the evidence renders the jury's verdict unsafe.
178. We turn, then, to the further evidence which we heard from Dr Squier and Dr Jones.

Fresh Evidence

179. The appellant sought to adduce evidence from Dr Squier and from Dr Jones to support his case that his son suffered brain damage as a result of what is described as a "short fall" which caused head injury and brain damage, at least two weeks before his son's death. The fall, it was suggested, occurred when, due to her disability, Sophia Rudder was unable to hold her son who must, therefore, have fallen to the floor.
180. The appellant also relied upon Dr Squier's evidence that the possibility that the child suffered from HIV could not be excluded. We heard the evidence of both Dr Squier and Dr Jones, *de bene esse*. Dr Jones described the mechanism of a fall which might

have caused significant head injury to Femi. He accepted that there was little data relating to the distance an infant would have to fall to suffer injury to the head. It would, as we would expect, depend in part upon the surface on which the baby fell. Dr Jones said that there was general acceptance as to the minimum degree of force (50-60G) as to which Dr Jones was less persuaded and the maximum (100-150G) for fatal damage. He concluded that there would be a risk of serious head injury should the baby have fallen from a height of 82 centimetres above ground. That distance would correspond to the height of Sophia Rudder's shoulder above ground. In cross-examination Dr Jones accepted that it was difficult, although not impossible, for any biomechanical model to simulate the complex anatomy of an infant's brain. Dr Al-Sarraj, called in rebuttal by the Crown, emphasised the difficulty of applying biomechanics to humans. He emphasised the complexity of the human brain and the difficulty of drawing conclusions from biomechanical experiments. In any event, he rejected the view that a low-level fall could have caused the extent of the catastrophic injury some two weeks or more before the child's death.

181. Dr Squier accepted that it was outwith her expertise to comment on whether the baby may have suffered from a fall, although she spoke of babies, considerably older than Femi, who had fallen either from beds, standing, or from a chair.
182. It must be recalled that it was part of the appellant's case at trial that the baby's injuries may have been attributable to a fall whilst being held by his mother. This possibility was rejected by the jury. We do not think that the evidence of Dr Jones could afford any ground for allowing the appeal. Dr Jones accepted, both in his oral evidence and in his written report, that there was very little data to be derived from experiment, as we would expect. He also accepted, both in his oral evidence and in his report, the difficulty of drawing conclusions because of the complexity of a baby's brain. This court, in the *Cherry* appeal in *R v Harris & Others* [2006] 1 Cr App R5 heard conflicting evidence from biomechanical engineers, both in relation to the injury to be expected from a fall and the extent to which injury could be caused by shaking (see [81-96]). At [213(iv)] the court described the science of biomechanics as "complex, developing and (as yet) necessarily uncertain". Nothing that we heard from Dr Jones led us to take a different view as to the science from that adopted by the court in *Harris*. Nothing we heard from Dr Jones leads us to question the safety of the jury's verdict.
183. There was clear evidence on the basis of which the jury must have rejected the suggestion of a fall from the baby's disabled mother. The most important feature was the evidence relating to the fracture at the lower end of the left humerus. The evidence of Professor Hall demonstrates the unusual nature of that fracture. Moreover, it was important evidence to demonstrate the lack of reaction by this appellant to the baby's screams and cry of pain which would have occurred over the period of about a week whenever the baby or its arm was handled in any way.
184. Although Dr Foster accepted that Sophia Rudder might have been able to pick up the child and then to drop him, she would have found it difficult to pick up the baby and

shake him and it would have been extremely difficult for her to have, in some way, twisted her son's arm. She would have lacked the power and co-ordination to do so. In those circumstances, the jury was entitled to reach the conclusion that it was unrealistic to suggest that the baby fell from his mother's chest and that he was caught by the arm in order to save him. This suggestion was rejected both by Professor Risdon and Dr Harding.

185. Moreover, the failure of the appellant to react to the baby's screams of pain over a period of a week provides powerful supporting evidence of both his responsibility for the injury and of his intention. As Dr Squier herself accepted, the fact of the unusual fracture to the baby's arm amounted to powerful evidence in relation to the cause of the head injury.
186. In her report dated 8 May 2009 Dr Squier accepted that Femi must have suffered severe and extensive brain damage at least some two weeks before death and described the clinical history as unusual. She suggested that it was unlikely that the baby would have survived without resuscitation and professional assistance. For that reason she suggested that the possibility of HIV encephalitis should be explored. She described the family history as unusual, referring to the fact that the baby's father came from Nigeria where there was a high incidence of HIV and asserting that the mother was young to have died from multiple sclerosis. Accordingly, Dr Squier "wondered" whether she may have had HIV rather than MS. She suggested that Professor Jean Bell should be asked to review the brain since she "has more experience than any other pathologist in this country in paediatric HIV encephalitis".
187. The result of Professor Bell's analysis was forthcoming by the time of the appeal. It was her opinion that the pathology was not due to any form of HIV related disease or AIDS. Dr Al-Sarraj also gave evidence of the differences between the appearances of the brain and those one would expect of children infected by HIV.
188. We must emphasise, therefore, that there was no evidence whatever that the condition of Femi had anything to do with HIV. There was no evidential basis for any such suggestion. It is, therefore, a matter of regret and surprise that we must record that despite the absence of any evidence of HIV encephalitis and the positive evidence from Professor Bell, who had attracted the praise from Dr Squier which we have recorded, Dr Squier was not prepared, before us, to reject the possibility of HIV encephalitis. She repeated that HIV had not been excluded and referred to the fact that Femi's father came from a country where it was endemic. Dr Squier should not have persisted in that suggestion. She herself had recommended examination by Professor Bell. When Professor Bell rejected the suggestion, Dr Squier did not accept that rejection. Dr Squier's stance, in oral evidence before us, casts significant doubt upon the reliability of the rest of her evidence and her approach to this case. It demonstrates, to our satisfaction, that she was prepared to maintain an unsubstantiated and insupportable theory in an attempt to bolster this appeal.

189. The inadequacies of her evidence were compounded by her persistence in the suggestion that Sophia Rudder might have suffered from HIV and not from MS. In fact Sophia Rudder had been treated by an eminent neurologist, Dr Foster. It is not sensible to suggest that that doctor would have attributed Miss Rudder's condition to MS when in fact she was suffering from HIV. Dr Squier appeared unaware of Dr Foster's reputation and saw fit to suggest HIV despite the reputation of Dr Foster and in apparent ignorance of the very fact that Sophia Rudder was being treated by her. Dr Squier's approach to that aspect of the evidence supports our views as to the unreliability of her evidence. She should not have suggested that cause of Sophia Rudder's condition and death without careful consideration of her treatment by Dr Foster and without informing herself of Dr Foster's qualifications and reputation.
190. In the light of our view as to the quality of Dr Squier's evidence before us we conclude it is not capable of undermining the safety of the verdict. For those reasons, we reject the application to call fresh evidence. Considering it, in the context of the medical evidence as a whole, it is not capable of affording any grounds for allowing the appeal (see s.23(2)(b) of the Criminal Appeal Act 1968).
191. We must, nevertheless, consider the evidence at trial as a whole, in response to the appellant's more general ground of appeal, that that evidence was insufficient to justify the jury's verdict of murder. The prosecution case alleged two separate head injuries, the first between two to four weeks before the death and the second a head injury leading to the death. Further, it was alleged that Femi's left arm had been deliberately broken, either at the time the first head injury was caused or on another occasion.
192. Mr Valios QC, on behalf of the appellant, contended that the jury could not safely have concluded that there was more than one occasion when the injuries occurred. In reliance upon Dr Squier and Dr Al-Sarraj, he contended that the second subdural haemorrhage could have been caused without any further trauma but as a result of a re-bleed. Dr Al-Sarraj accepted that possibility, in evidence before this court.
193. It is true that there were unresolved issues as to the sequence of events. In particular, all the expert evidence showed that the head injury from which Femi suffered at least two weeks before his death was severe and would have had a catastrophic effect upon his condition. Femi, as Dr Harding said in evidence, had suffered from significant destruction of the brain several weeks before death. Thus by the time of either the second injury or the re-bleed he was already severely brain-damaged. The symptoms would have been severe. The child, according to Dr Richards, would have had abnormal movement of limbs, would probably suffer from seizures, his cry would have been high-pitched and feeding would be very weak. He would barely be able to suck.

194. It must be recalled that Femi was taken to Dr Sathananthan on 6 October 2005 when the appellant reported his son as being “very snuffly for the past three days” and not feeding.
195. The GP might well have not observed any slight remaining swelling due to the arm fracture, provided that that fracture had taken place at least a week before the visit on 6 October. However, it is not possible to reach any conclusion as to why or how the General Practitioner missed the consequences of the severe head injury.
196. In our view, it does not matter. Whether that injury occurred before or after the visit to the GP, the jury was entitled to reject the view that it occurred as a result of an accidental fall, whilst being held by Sophia Rudder. If that had occurred, it makes the failure of this appellant to report the catastrophic effect on his son inexplicable. Nor does it explain how his son came to suffer so unusual an arm fracture or the failure of this appellant to report it.
197. We accept that there persists a doubt as to whether the baby’s left arm was fractured at the same time as he suffered the severe first head injury or on a separate occasion. But we reject the submission on behalf of the appellant that it matters. Nor do we think of particular significance the issue whether the second brain injury was a result of a re-bleed or secondary bleeding into a pre-existing site of brain injury, or a second brain injury. Of course, if the prosecution could establish three separate occasions of deliberate injury caused to the baby, that would be powerful evidence of murderous intent.
198. However, we reject the submission that if the evidence fell short of establishing three separate occasions when deliberate injury was caused, that undermines the safety of the verdict. The combination of the severity of the injury to the brain and the fracture to the arm, even if they were caused on the same occasion, not only establishes that the perpetrator was not the disabled mother, but rather that it was this appellant. The nature and severity of the injury, particularly to the left arm, is a sufficient basis to establish a murderous intent. That evidence is further supported by the absence of any reaction, let alone any report, of the condition of his son by this appellant when faced with the cries of pain of his son and the apparent effect, which we have described, of the first injury to the baby’s head.
199. For those reasons, we reject the submissions that a verdict, other than a verdict of manslaughter, was unsafe, or that it was unsafe to attribute the injuries from which Femi ultimately died to anyone or to any cause other than this appellant’s own deliberate actions. For those reasons, his appeal is dismissed.

General

200. A just resolution of the three trials to which these appeals relate depends upon the judge ensuring, so far as possible, that they proceed on what Judge LJ described as a logically justifiable basis (*Kai-Whitewind* [90]). In Henderson that process established, for the reasons we have given, the guilt of the appellant. By contrast, in Butler there was no logically justifiable basis upon which a reasonable jury properly directed could conclude that the expert evidence adduced by the Crown established guilt. Certain lessons from these appeals in relation to case management and the structure and content of the summing-up may achieve the objective described by Judge LJ in prosecutions which depend solely on medical report evidence.
201. Justice in such cases depends upon proper advanced preparation and control of the evidence from the outset at the stage of investigation and thereafter. The police and the Crown Prosecution Service acknowledge the sensitivity of these cases and that the evidential picture may change as opinions from experts are obtained by either the prosecution or the defence. The approach of the prosecution in such cases has been published in “Guidance on the Prosecution Approach to Shaken Baby Syndrome Cases” by the Director of Public Prosecutions on 14 February 2006 and in updated legal guidance by the Crown Prosecution Service in relation to child abuse, access to which is obtainable on the internet.
202. The problem for the courts is how to manage expert evidence so that a jury may be properly directed in a way which will, so far as possible, ensure that any verdict they reach may be justified on a logical basis.
203. In *Kai-Whitewind* Judge LJ rejected the contention that where there is a conflict of opinion between reputable experts, expert evidence called by the Crown is automatically neutralised [84]. He emphasised that it was for the jury to evaluate the expert evidence even where the experts disagree as to the existence of the symptoms upon which their opinions were based [88-89]. But how is a jury to approach conflicting expert evidence? We suggest it can only do so if that evidence is properly marshalled and controlled before it is presented to the jury. Unless the evidence is properly prepared before the jury is sworn it is unlikely that proper direction can be given as to how the jury should approach that evidence. Thus the jury will be impeded in considering that evidence in a way which will enable them to reach a logically justifiable conclusion.
204. It is in those circumstances we must emphasise the importance of the pre-trial process. First, we suggest that the judge who is to hear a particular case should deal with all pre-trial hearings, save for those in which no issue of substance is to be considered. Second, it is desirable that any judge hearing cases such as these, which depend entirely on expert evidence, should have experience of the complex issues and

understanding of the medical learning. This is easy enough to achieve in the Family Division, more difficult in a criminal jurisdiction.

205. Proper and robust pre-trial management is essential. Without it, real medical issues cannot be identified. Absent such identification, a judge is unlikely to be able to prevent experts wandering into unnecessary complicated and confusing detail. Unless the real medical issues are identified in advance, avoidable detail will not be avoided.
206. The process of narrowing the real medical issues is also vital in relation to another important function of the judge in advance of the trial. He should be in a position to identify whether the expert evidence which either side wishes to adduce is admissible. This assessment is as difficult as it is important. The test adopted by this court in *Harris* was described in the judgment of King CJ in *R v Bonython* [1984] 38 SASR 45: First, whether the subject matter of the opinion falls within the class of subjects upon which the expert testimony is permissible and second, whether the witnesses acquired by study or experience have sufficient knowledge of the subject to render their opinion of value in resolving the issues before the court. *Bonython* was cited by this court in *R v Reid & Ors* [2009] EWCA Crim 2698 [111(i)] with the qualification that it is important that the court acknowledges advances to be gained from new techniques and new advances in science. *Reid* is concerned with DNA evidence but the observations of the court in relation to the admissibility of expert evidence apply with equal force to cases concerning baby shaking as it applied to the developing science of DNA. We shall return to emphasise the importance of Part 33 of the Criminal Procedures Rules 2010 in the context of these cases. We shall say no more about admissibility since the unsatisfactory state of the law has been the subject of the Law Commission Consultation paper No. 190 “The Admissibility of Expert Evidence in Criminal proceedings in England and Wales”, and is likely to lead to changes in the current approach of *laissez-faire*, which the Law Commission suggests requires reform (3.14).
207. Courts should be familiar with the Report on “Sudden Unexpected Death in Infancy: The Report of a Working Group Convened by the Royal College of Pathologists and The Royal College of Paediatrics and Child Health” chaired by Baroness Kennedy QC published in September 2004. The Kennedy report cautions against doctors using the courtroom to “fly their personal kites or push a theory from the far end of the medical spectrum”. It recommends a checklist of matters to be established by the trial judge before expert evidence is admitted, including:-
- “1. Is the proposed expert still in practice?
 2. To what extent is he an expert in the subject to which he testifies?
 3. When did he last see a case in his own clinical practice?

4. To what extent is his view widely held?"

208. We emphasise the third, which was of importance in these appeals. The fact that an expert is in clinical practice at the time he makes his report is of significance. Clinical practice affords experts the opportunity to maintain and develop their experience. Such experts acquire experience which continues and develops. Their continuing observation, their experience of both the foreseen and unforeseen, the recognised and unrecognised, form a powerful basis for their opinion. Clinicians learn from each case in which they are engaged. Each case makes them think and as their experience develops so does their understanding. Continuing experience gives them the opportunity to adjust previously held opinions, to alter their views. They are best placed to recognise that that which is unknown one day may be acknowledged the next. Such clinical experience, demonstrated, for example, by Dr Peters in the case of Henderson, may provide a far more reliable source of evidence than that provided by those who have ceased to practise their expertise in a continuing clinical setting and have retired from such practice. Such experts are, usually, engaged only in reviewing the opinions of others. They have lost the opportunity, day by day, to learn and develop from continuing experience.
209. *Reid* also contains important observations as to Part 33 of what are now the Criminal Procedure Rules 2010. Those rules need to be deployed to ensure that the overriding objective to deal with criminal cases justly is achieved (1.1). The rules are designed to ensure that the expert opinion is unbiased (33.2.1) and in particular, by virtue of 33.3(1), that an expert report provides evidence of relevant experience and accreditation (a), details of any literature relied upon (b), that any range of opinion should be summarised and reasons given before the opinion of the expert (f) and that any qualifications to that opinion should be stated (g).
210. Generally, it will be necessary that the court directs a meeting of experts so that a statement can be prepared of areas of agreement and disagreement (33.6.2(a) and (b)). Such a meeting will not achieve its purpose unless it takes place well in advance of the trial, is attended by all significant experts, including the defence experts, and a careful and detailed minute is prepared, signed by all participants. Usually it will be preferable if others, particularly legal representatives, do not attend. Absent a careful record of the true issues in the case, it is difficult to see how the trial can be properly conducted or the jury properly guided as to the rational route to a conclusion. The court may be required to exercise its important power to exclude evidence from an expert who has not complied with a direction under [33.6(2), 33.6(4)]. The court should bear in mind the need to employ single joint experts where possible (33.7).
211. In the context of Part 33 we should draw attention to the fact that defence experts are not obliged to reveal a previous report they have made in the case, still less to reveal adverse criticism made by judges in the past. But a failure to do so will not avail the defence. A judge may well be able to exercise his powers under the Criminal Procedure Rules to ensure that in advance of a trial a defence expert has made disclosure of any relevant previous reports and any adverse judicial criticism. Failure

to do so would be contrary to the overriding objective and will achieve no more than to expose the expert to cross-examination on those points at trial. It is difficult to see how those acting on behalf of the defendant could permit an expert report to be advanced without satisfying themselves that previous reports have been disclosed and any adverse judicial criticism identified and disclosed. Failure to do so by either side will only cast suspicion upon the cogency of the opinion. A defence team which advances an expert without taking those precautions is likely to damage its client's case.

212. A case management hearing may often present an opportunity for concerns as to previous criticism of an expert and an expert's previous tendency to travel beyond their expertise to be aired. Whilst such history may not be a ground for refusing the admission of the evidence, it may well trigger second thoughts as to the advisability of calling the witness.
213. As we indicated, if the case is to proceed on a logically justifiable basis, it must surely be concluded on a logically justifiable basis. A logically justifiable conclusion depends upon the structure and quality of the directions in summing up given by the judge. We have already drawn attention to the consequences in *Schmidt* of a summing-up which failed to direct the jury as to the issues [116].
214. The essential medical issues which the jury have to resolve should be clear by the time the trial starts. Those issues should have been defined and the expert evidence, identifying the sources on which the evidence is based, should also be clear before the trial starts. Thus the direction of evidence-in-chief, cross-examination and any submissions, either at the close of the prosecution case or in speeches to the jury, should be focussed. Of course the evidence in such trials, as in any criminal trial, may take on a different colour as the case progresses. But we suspect that with proper advance trial management, the unforeseen is far less likely to occur in cases which depend entirely upon expert scientific evidence.
215. By the time the judge comes to sum up the case to the jury the issues and the evidence relevant to the issues should be understood by everyone, including the jury. Whilst it is conventional to discuss the law with counsel, the judge should, generally, take the opportunity to discuss the issues of medical evidence before the time comes for counsel to address the jury. The judge will thus be in a position carefully to structure his summing-up to those issues. He will be able to identify which evidence goes to resolution of those issues. He should generally sum the case up to the jury issue by issue, dealing with the opinions and any written sources for those opinions issue by issue, unless there is good reason not to do so. Merely repeating the expert evidence in the order in which that evidence was given serves only to confuse. It is pointless, literally. It deflects the jury from their task. It does not save them, as they must be saved, from avoidable details. It blurs their focus on evidence going to the real issues. The summing-up should enable anyone concerned with an adverse verdict to understand how it has been reached.

216. In the Family Division judges will set out the features of the expert evidence on which a judgment is required and those factors which form the basis of the judgment they have reached. So too a jury should be confronted with the issues it must decide and the factors they should consider as the basis for judgment, one way or the other. Anyone reading a summing-up composed in that way should be able to understand the route followed by the jury in reaching its verdict.
217. There are two features of the content of a summing-up in cases such as these which, we suggest, are important. First, 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 Holdsworth* [2008] EWCA Crim 971 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 [57].
218. Second, the jury need directions as to how they should approach conflicting expert evidence. *Kai-Whitewind* teaches that the mere fact that expert differs from expert is no ground for withdrawing the case from the jury. But how is the jury to approach such a conflict? To suggest, in cases where the expert evidence is fundamental to the case, that the jury should approach that expert opinion in the same way as they do in every other criminal case, is inadequate. It is difficult enough for Family Division judges to express their reasons for accepting or rejecting conflicting expert evidence, despite their experience. Juries, we suggest, should not be left in cases requiring a higher standard of proof to flounder in the formation of a general impression. A conclusion cannot be left merely to impression. In the appeal of Henderson, Dr Leestma gave, if we may say so, a most beguiling impression, courteous and understated as it was. But there were, as we have concluded, sound reasons relating to his experience in comparison with Dr Al-Sarraj for rejecting what he told us. Lacking the experience of Family Division judges, a jury needs to be directed as to the pointers to reliable evidence and the basis for distinguishing that which may be relied upon and that which should be rejected.
219. In *Harris* the court pointed out the assistance given by Cresswell J [271]. That guidance is of assistance not only to judges, practitioners and experts themselves, but also to a jury. If the issue arises, a jury should be asked to judge whether the expert has, in the course of his evidence, assumed the role of an advocate, influenced by the side whose cause he seeks to advance. If it arises, the jury should be asked to judge whether the witness has gone outside his area of expertise. The jury should examine the basis of the opinion. Can the witness point to a recognised, peer-reviewed, source

for the opinion? Is the clinical experience of the witness up-to-date and equal to the experience of others whose evidence he seeks to contradict?

220. Of course, none of these features will determine the case. Not all of these features are even relevant in every case. But we seek to emphasise the importance of guiding the jury as to the proper approach to conflicting opinions. An overall impression can never be the substitute for a rational process of analysis. The jury are not required to produce reasons for their conclusion. Nevertheless, the judge should guide them by identifying those reasons which would justify either accepting or rejecting any conflicting expert opinion on which either side relies.
221. We acknowledge the danger of being over-prescriptive in relation to directions to the jury. But judges, we suggest, need to remember that their directions are part of the means by which they ensure that a case which depends on expert evidence proceeds to its conclusion on a logically justifiable basis.