Neutral Citation Number: [2012] EWHC 865 (Fam)

Case No: FD10C00445

IN THE HIGH COURT OF JUSTICE
FAMILY DIVISION

Royal Courts of Justice
Strand, London, WC2A 2LL

Date: 19 /04/2012

Before:

MRS JUSTICE THEIS DBE

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Between:

MAYOR AND BURGESSES OF THE LONDON BOROUGH OF ISLINGTON	**Applicant**
- and -	
CHANA AL-ALAS	**1st Respondent**
-and-	
ROHAN WRAY	**2nd Respondent**
-and-	
JAYDA FAITH AL-ALAS WRAY (through her Children's Guardian Dorothy Pottinger)	**5th Respondent**

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Mr Mark Love & Ms Lyndsey Sambrooks-Wright (instructed by **Local Authority Legal**) for the **Applicant**
Ms Jo Delahunty QC & Ms Kate Purkiss (instructed by Goodman Ray Solicitors) for the **1st Respondent**
Mr Ian Peddie QC and Mr Christopher McWatters (instructed by S A Carr & Co Solicitors) for the **2nd Respondent**
Mr Vinod Sharma of BKS Legal Solicitors for the **5th Respondent**

Hearing dates: 20th February – 26th March 2012

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Approved Judgment

I direct that pursuant to CPR PD 39A para 6.1 no official shorthand note shall be taken of this Judgment and that copies of this version as handed down may be treated as authentic.

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MRS JUSTICE THEIS DBE

This judgment is being handed down in private on 19th April 2012. It consists of 67 pages and has been signed and dated by the judge.

Mrs Justice Theis DBE:

Introduction

1. This matter concerns a young child, Jayda Faith Al-Alas Wray, born on 17.10.10, who is now 17 months old. She has been in the care of the Applicant Local Authority ('LA') since birth. At the time she was born her parents, Channa Al Alas and Rohan Wray, had been charged with the murder of her older brother, Jayden. Jayden was born on 7.3.09 and died on 25.7.09. The parents stood trial at the Central Criminal Court ('CCC') in November last year. The trial lasted 6 weeks. At the end of the defence case the trial judge, HHJ Kramer Q.C., acceded to the defence application that the case should not be put to the jury. The prosecution did not appeal that ruling; the jury were directed to acquit the parents on 9.12.11.

2. The hearing before me started on 20.2.12. I have heard four weeks of evidence. The vast majority of the witnesses were clinicians or medical experts. Simply put the LA's case is that Jayden died as a result of inflicted trauma caused to him whilst in the care of the parents. In addition, they allege he suffered a number of fractures that, despite having rickets, were caused by non accidental injury. They don't advance a positive case in terms of propensity against the parents; they rely on the medical evidence and the accepted fact that during the relevant time Jayden was in the de facto care of either, or both, of the parents. In effect, they rely on the medical evidence of fact and opinion to undermine the credibility of the parents.

3. At the start of this hearing the maternal grandmother, Catrina Al Alas, and maternal aunt, Rhianie Al Alas, were interveners as they had been part of the same household as Jayden during the relevant time. The case against them was based on opportunity only, there was no positive case asserted against them by the LA.

4. The interveners were discharged at the start of this hearing on the basis that no party sought to advance a positive case against them, the parents accepted that in the event that the court found Jayden suffered from any non accidental injury only they were in the pool of perpetrators.

5. It is accepted that Jayden suffered from congenital rickets. Congenital refers to timing; he was born with it. It is very likely his mother was vitamin D deficient during pregnancy; he was born with vitamin D deficiency. He was entirely breastfed by his mother, one of the sad facts of this case is, unbeknown to his mother or those advising her, that further contributed to his vitamin D deficiency.

6. This case generated a considerable amount of publicity at the time of the criminal trial. I am aware that some of the medical issues considered in this case have generated debate, both within and outside the medical arena. It is important to remember that my conclusions set out below are entirely related to this case. Despite their differences of opinion, all the medical experts agree this case is extremely complex. By their very nature cases such as this are very fact specific and great caution should be adopted in using any conclusions I reach to support any wider views outside the very specific facts of this case (*Henderson v R [1020] EWCA Crim 1269 per Moses LJ para 6 and 7*).

The Law

7. The parties have been able to submit a document agreeing the relevant law. I need do no more than summarise the relevant matters.

8. The burden of proof is on the LA and they have to satisfy the court on the balance of probabilities in accordance with principles laid down in *Re B (Care Proceedings: Standard of Proof) [2008] UKHL 35 in particular per Baroness Hale at para 70 and 72]*

9. Much of the evidence in this case has turned on the existence or not of the triad. That cannot itself, even if established, be diagnostic of inflicted abuse and the task of the court is to weigh in the balance all the evidence, including that which is inconsistent with inflicted trauma. In *R v Harris [2005] EWCA Crim 198 Lord Justice Gage said*

   "*The triad becomes central to a diagnosis of NAHI when there are no other signs or symptoms of trauma such as bruises or fractures*" [Para 65].

   " *Whilst a strong pointer to NAHI on its own we do not think it possible to find that it (the triad) must automatically and necessarily lead to a diagnosis of NAHI. All the circumstances, including the clinical picture, must be taken into account*" [Para 70]

   "*..cases of NAHI are fact specific and will be determined on their individual facts*" [Para 267].

10. It is always open to a judge to rule that the cause of an injury remains unknown. In *Re R (Care Proceedings: Causation) [2011] EWHC 1715 Hedley J*

    'In my judgment, a conclusion of unknown aetiology in respect of an infant represents neither professional nor forensic failure. It simply recognises that we still have much to learn and it also recognises that it is dangerous and wrong to infer non-accidental injury merely from the absence of any other understood mechanism. Maybe it simply represents a general acknowledgement that we are fearfully and wonderfully made.'

11. The importance of other evidence, particularly where medical opinion is not unanimous, should not be overlooked or undervalued. As Butler Sloss P said in *Re U: Re B (Serious Injury: Standard of Proof) [2004] 2 FLR 263 at para 26* the court's responsibility is to survey a '*wide canvas'* of the evidence (see: Lord Nicholls Of Birkenhead in *Re H and R (Chid Sexual Abuse: Standard of Proof) [1996] 1 FLR 80 at p23;* Ryder J in *A County Council v A Mother and Others [2005] EWHC 31 (Fam) para 44;* In *Re L (Children) [2011] EWCA Civ 1705 Thorpe LJ said in dismissing the appeal 'Clearly from the forensic standpoint, given any degree of uncertainty in the medical and scientific field, the judge's appraisal and confidence in the parent is absolutely crucial to outcome'*.)

12. The frontiers of medical science are always expanding. As Professor Luthert was quoted in *R v Harris [2005] EWCA Crim 198 para 135* '*There are areas of ignorance. It is very easy to try and fill those areas of ignorance with what we know but I think that it very important to accept that we do not necessarily have a sufficient understanding to explain every case.'*

13. Where there is disputed medical evidence guidance was laid down by Butler-Sloss P in *Re U; Re B (Serious Injury: Standard of Proof) [2004] 2 FLR 263 at para 23*

> *"In the brief summary of the submissions set out above there is a broad measure of agreement as to some of the considerations emphasised by the judgement in R v Cannings that are of direct application in care proceedings. We adopt the following:*
>
> *(i) The cause of an injury or an episode that cannot be explained scientifically remains equivocal.*
> *(ii) Recurrence is not in itself probative.*
> *(iii) Particular caution is necessary in any case where the medical experts disagree, one opinion declining to exclude a reasonable possibility of natural cause.*
> *(iv) The court must always be on guard against the over-dogmatic expert, the expert whose reputation or amour propre is at stake, or the expert who has developed a scientific prejudice.*
> *(v) The judge in care proceedings must never forget that today's medical certainty may be discarded by the next generation of experts or that scientific research will throw light into corners that are at present dark."*

14. It is important to remember that the task of the court is to decide on the evidence before it and it can depart from the view of expert evidence provided sound reasons are given for doing so (*Re B (Care: Expert Witnesses) [1996] 1 FLE 667 at 670 D-E per Ward LJ.*

15. I also have in mind the observations made by Moses LJ in *Henderson v R (ibid)* about the status of the note of the meeting convened at the Royal College of Pathologists in December 2009. Dr Bonshek, one of the Opthalmologists who attended that meeting, and gave evidence in this case, said that the inclusion of large macular folds in the conclusion relating to retinal haemorrhages was not agreed, although in the body of the document when it considers retinal haemorrhages it states '*A perimacular retinal fold has a clear association with pre-mortem head trauma.'*

Background

16. The father, Rohan Wray ('the father), was born on 12.8.89 and the mother, Chana Al Alas ('the mother'), on 8.6.92. They are now 22 and 19 respectively. The mother's sister was born on 20.1.95. The parent's relationship commenced in January 2007, when the father was 17 years and the mother 14 years. The parents saw each other most days and each of their respective families were aware of the relationship. The mother lived with her mother, Catrina Al Alas, and her sister, the father lived with his mother and his two younger step-sisters.

17. The mother started to feel sick in the summer of 2008, she attended her GP with her mother on 1.8.08, when her pregnancy was confirmed. She was then 16 years old. As Catrina Al Alas ("MGM") confirmed in her oral evidence, this was not welcome news bearing mind the age of the mother. She said they discussed the options; the

mother was adamant that she wanted to keep the baby. From then on the mother had the support of her immediate and wider family, as did the father.

18. Thereafter, the mother attended all antenatal appointments, either at the hospital, the clinic or GP surgery. The father usually accompanied and supported her. In addition to the conventional antenatal visits there was a programme designed specifically for young mothers called the Family Nurse Partnership Programme (FNP). This is a voluntary programme to provide additional support and advice for young mothers. The rationale behind this programme was that early intervention would provide long term benefits. Lorna Waite (a qualified midwife with 6 years experience) was allocated to the mother, she had her first visit on 13.8.08. The programme involves weekly visits for about six weeks and thereafter fortnightly visits. The visits last between 1 – 1 ½ hours and took place in the family home. 12 visits took place prior to Jayden's birth; the father was present for 5 of them. The MGM was also present for some too. Lorna Waite describes in her written and oral evidence the mother's co-operation in this programme, the mother was willing to engage in this service and she formed a good working relationship with her. The mother's antenatal visits at the clinic were with midwife Deborah Kroll. Her statement describes the mother's attendance at all appointments.

19. Jayden was born on 7.3.09. The father, MGM and sister were present. He was born with vitamin D deficiency and suffered from congenital rickets. This was undiagnosed at birth or in life thereafter. His birth involved an acute obstetric emergency procedure that required the mother to be place in the 'McRoberts position'. This is because during labour, Jayden could not be delivered as his shoulder became stuck. The midwife initially asked the mother's family to assist by physically holding the mother's legs up, but further assistance was required. The midwife made three attempts to free the shoulder in 3 ½ minutes. Another midwife arrived. She did not know the other midwife had also made attempts to dislodge the shoulder and she also applied the 'McRoberts' manoeuvre. Neither midwife knew Jayden had congenital rickets. The first midwife accepted when he gave evidence at the CCC that had he known it might not have been normal midwifery care; it might have been a c-section. After Jayden's birth he was examined by a third midwife who observed him to have stiffness to his shoulder, on later examination this appeared to have progressed to his right arm.

20. They were discharged the next day. At the invitation of the MGM the father effectively moved into the home full time thereafter. He returned back to his mother's home regularly for visits, particularly when he was on a back to work programme for a few weeks. There came a time when he stopped doing that due to an argument with his mother about who should be invited to her birthday party. He didn't speak to her for a period of time but by the time of her birthday, on the 11th July, they had resolved their differences, included in the papers are photos of the parents and Jayden at that party.

21. Jayden was exclusively breast fed by his mother. It was subsequently discovered, after Jayden's death, that she too was vitamin D deficient and was very likely to have been so during her pregnancy. Unbeknown to her (or anyone else) the fact that she breastfed Jayden almost certainly further contributed to his vitamin D deficiency.

22. The parents presented Jayden for all professional and clinical appointments (scheduled as well as when needed) throughout his life. In addition to the conventional post natal care the FNP programme also covered this period. Lorna Waite continued to visit, weekly after the birth for about four weeks and then fortnightly thereafter. There were 11 visits between 10.3.09 and 9.7.09. Most, if not all of her appointments were in the family home and would last for 1 – 11/2 hours. She said the parents always engaged with her and did what she advised. She also saw them at an open day on 29.6.09. She recalled speaking to the parents, seeing Jayden and giving him a cuddle. She said there was nothing of note or concern in her observations of the parents or Jayden that day.

23. None of those who came into contact with Jayden and his parents during this period raised any concerns about his care, the way each of the parents related to him and each other, or noted any injury or marks on him. The father attended the majority of the appointments with the mother.

24. The community midwife visits noted as follows:
13.3.09 *'I undressed the baby and checked him from head to foot and had no concerns, I did not see any injuries and he seemed physically well'.*
23.3.09 physical check of Jayden was carried out and records *'I was assisted in undressing him by Chana. The physical checks consisted of an all over body check and I saw no bruises or anything that caused me concern'*
30.3.09 *'conducted my checks on Jayden, I saw Chana undress and hold him and her interaction with him was appropriate. I completed a physical check on Jayden. This includes a front and back check and head to foot check. On being handled Jayden seemed fine and I saw no injuries on Jayden and appeared to be physically well on seeing his skin tone, weight and the way he was moving.'* She was satisfied that the mother no longer required their services and so discharged her from their care.

25. The parents also received visits from health visitors, they attended all appointments and there were no concerns. Sheena Gofton was one of the health visitors, she saw Jayden with his parents in clinic on 14.4.09, 5.5.09 and 30.6.09; Manni Welch (another health visitor) saw them on 2.6.09, 16.609 and 14.7.09. In her oral evidence Ms Gofton explained that the health visitors' role was in addition to the support provided by the FNP. She agreed her examinations were not limited to physical examinations of the child; she looked at the parents' relationship with each other and their interactions with Jayden. She said she spent extra time with the parents as she realised they were very young. In their statements made in August 2009 the health visitors made the following observations about each of these appointments:
14.4.09 *"it was mainly Chana who replied to my questions although I can't recall clearly her exact response. I remember thinking how nice it was that both parents had come with baby, especially as they were so young. They looked very sweet together and came across as being very together….I had no concerns with them or Jayden. They handled Jayden well and seemed to interact well with him – both looking at him and smiling….They presented as a happy family and Jayden as a happy healthy baby. I had no concerns"*
5.5.09 (Jayden's 6 – 8 week check) *"I can't remember which parent carried Jayden into my room but it was Rohan who undressed him and put him onto the exam*

trolley. It was also Rohan who carried Jayden round to the GPs room once I was finished. However Chana was the one who I was mainly addressing my questions to and also she frequently smiled at Jayden, so this caused me no concerns. I measured J's weight, head circumference and length…I do remember that all of his measurements were normal. It is the policy of the Primary Care trust that all babies aged under one year are weighed naked. I saw no visible markings or injuries to Jayden. To me he appeared as a happy, thriving, well settled baby. Both of his parents appeared to interact well with him." Ms Gofton also spoke to Chana to ensure there were no concerns regarding possible post natal depression. Ms Gofton said she had no worries on that front.

2.6.09 "*impressed by how caring he [Rohan] seemed of Jayden in that he was very attentive and gentle with him…her [Chana's] replies were all positive and she seemed very happy talking about her baby. I thought it very positive that she was breastfeeding and Jayden was in a good feeding routine…I had no concerns with him – he was clean and appeared happy. He was looking around and reacting well. He presented as a normal baby with no visible injuries or illnesses"*

16.6.09 *"On 16th June I didn't observe anything wrong with him. Again he presented as a clean and well cared for baby. He was looking around. He appeared settled. I can't recall on this occasion which parent handled him but I can say that I believe both parents to smile at him and interact with him well. The father, Rohan, asked appropriate questions about Jayden's feeding as he was worried he was now sleeping through the night whereas before he had been waking up for a feed. I reassured him that this was perfectly normal for a baby of Jayden's age. The mother Chana was not worried about anything and I saw no reason for her to be so. She was again quiet, unless spoken to, but seemed happy and responded appropriately to anything asked of her"*

30.6.09 (this was a drop in clinic which parents' can attend) *"Rohan predominantly handled the baby but Chana was attentive of Jayden and often smiled at him….I remember praising Chana for how well she was doing breast feeding and she was really happy with this praise – her whole face lit up. Again Jayden presented as a happy and settled baby. Both parents interacted well with him….Nothing from their demeanour caused me any worrie*s"

14.7.09 *"Jayden was again accompanied by both parents and again nothing in their demeanour worried me. Both seemed happy and well adjusted…Jayden presented to me as a happy, healthy child and his parents appeared well adjusted and attentive of him"* On that occasion a student nurse was present and Ms Welch said she oversaw and repeated everything that the student nurse did. It was a relatively short visit as it was only a weight check but she noted nothing of concern.

26. Ms Welch in her statement added *"Jayden being well established in his feeding routine, as he appeared and his mother, Chana doing so well with the breastfeeding, indicated to me that she was happy with her baby and caring towards him. Often if a mother is emotionally bothered this can affect her milk and connection to the child through the feeding, which was not the case with Chana"* Then at the end of her second statement *"In my professional judgment I had no concerns regarding Rohan or Chana or Jayden…[Chana] appeared to me to be interested in her son in that she watched him and smiled at him. She responded to any questions I had verbally and smiled broadly at me when I praised her about doing so well with breastfeeding….I would say Chana and Rohan were both*

emotionally positive in themselves and towards Jayden. They were good attendees to the clinic and I had no concerns"

27. Following Jayden's birth there were a number of appointments with the GP. Jayden attended all required appointments for immunisations. On 21.4.09 the mother attended for her post natal check, which raised no concerns. On 5.5.09 Jayden was seen for his 6 – 8 week check by Dr Mills in conjunction with the appointment with the health visitor, Ms Gofton, referred to above. Dr Mills gave details in her oral evidence of what that involved. It involved a detailed examination of his hands and fingers, hips and genitalia (without his nappy). She would watch carefully how the baby was moving his limbs, whether he turns his head and his reactions. She would turn him over to check his spine and would check his head. She said she would have been able to see if there were any marks or bruising. She said there was nothing in the physical examination, in the way the parents reacted to each other, or to Jayden that caused her any concern.

28. On 20.5.09 Jayden was taken to the GP due to the parents concern about bleeding from his gums. In his statement Dr Adesanoye described examining Jayden with his vest on and not seeing any marks or bruises. He diagnosed a possible cold and prescribed some nasal drops. The father was cross examined about an entry in the GP records about this visit that recorded *'crying infant'* and *'crying all day'* as being inconsistent with the account the parents gave of Jayden being an easy baby. The father said that was not the primary reason for seeing the doctor and the mother was not asked about this entry in these proceedings.

29. On 22.5.09 Jayden was taken to the GP, with a history of posseting blood specs. The GP referred Jayden to A&E at University College London Hospital (UCLH). He was also examined by Dr Ian Cole, paediatric senior house officer, who noted Jayden looked well, was affable and all his observations were normal. In his statement he states *'On examination…from a neurological viewpoint Jayden was alert interested in his surroundings and moving all his limbs independently…Jayden's parents were appropriately concerned and wanted to know what the cause was'.* He admitted Jayden to the paediatric ward and he was fully examined by Dr Yasmin Baki, paediatric registrar, at about 8.30pm that evening. After examining him (*'I examined Jayden, I listened to his chest and heart, which was normal, and I felt his belly, which was soft and normal. I checked his nappy and there was no blood in the stool and urine. I checked his mouth and there were no tears in his mouth. I reassured his Mum and explained if there were any further concerns to return to the hospital')* her statement confirmed there were no concerns regarding Jayden and he was discharged.

30. On 27.6.09 Jayden was taken to see his GP as he had a scab under his nose. The GP notes that he was *'alert, good social smiles'* an ointment was prescribed.

31. On 1.7.09 Jayden received his third set of immunisations at the GP surgery.

32. On 14.7.09 when he was seen at the clinic for a routine check his parents mentioned that they were worried Jayden may have flu because the mother had been unwell with flu. Calpol was advised.

33. On 17.7.09 Jayden was taken to the GPs. He was seen by Dr Rahman with a history of cold and cough. In her statement she recorded *'on examination Jayden looked unwell with an increased respiratory rate..fast pulse..and mild fever..he exhibited a puffy appearance round his eyes and appeared to be drowsy..he was well hydrated..his chest was clear'* She referred Jayden to UCLH for review where he was seen by Dr Williams (paediatric registrar) after being seen by the nurse who took a history and weighed and measured him. Dr Williams provided a statement and gave oral evidence. She said the parents told her that over the last 2 days Jayden had become unwell; he had a runny nose, cough and had been sleepier than usual, although he was waking for feeds. She examined Jayden (who was wearing only a nappy when examined). She described him in her statement as *'alert, smiling, playing and did not appear unwell'*. She did not notice any unusual skin marks, including in the groin region when she undid the nappy to feel his femoral pulses and examine his genitals. Temperature, pulse and respiratory rate were all normal. There were no signs of respiratory distress and his chest was clear. He had dried nasal secretions in his nostril and an inflamed throat. She diagnosed him as having an upper respiratory tract infection and discussed with the parents to either return to A&E or contact the GP if he developed poor feeding. She reported both parents as interacting appropriately with Jayden.

34. According to Professor Malcolm Jayden had suffered all significant fractures by this date, none of which were detected by Dr Williams and there was nothing about the way the parents related to each other, or to Jayden, that concerned her.

35. In the 19 weeks after his birth and prior to his admission on 22.7.09 Jayden had been seen by about 20 medical and health professionals on about 30 occasions when no concerns were noted.

36. According to the parents nothing remarkable happened over the next few days. They continued to give Jayden calpol, on some days he appeared to be improving a little. There were no difficulties with his feeding and they did not feel the need to return back to the hospital. The MGM was around in the home and did not have any concerns. The three bedroom flat they lived in has thin walls and it is easy to hear what is going on in other rooms.

37. On the evening of 21st July, according to the parents, Jayden was fed about 9pm and the MGM put him in his cot as she was going out for the evening and staying out for night (the first time she had done so), she said she had no concerns about him other than he was under the weather. The parents said he was fed again at about midnight, then winded and put in his cot. It was a particularly hot night and the father slept on the floor. The mother said she woke at 5 am and Jayden was in an unusual position in the cot with his head against the bars and his arms through the bars. The father thought he was on his tummy whereas the mother said he was on his back. The father recollects adjusting him in the cot and he slept for a bit longer, the mother didn't recollect that. When he was taken out of his cot for a feed she said his mouth was shut and it seemed his tongue was stuck to the roof of his mouth. She could not get him to feed. The father held him for a bit and she then played with him in his baby bouncer. She said she tried the keys, they were his favourite toy and he reached out for them to put them in his mouth and followed them with his eyes. She said he looked wherever she was going. She continued to try and feed him but was

not successful. At some point she dressed him and did not notice anything unusual in his limbs.

38. They decided to contact the hospital using the number they had been given on the 17.7.09. The father said he spoke to someone there. He described the symptoms and was told that it didn't sound like anything serious and to take him to the GP. The father rang the surgery and made an appointment. The mother said she was still checking Jayden and he was looking where her hands were going and she was talking to him. They walked to the surgery with Jayden in his buggy, the mother noticed Jayden's arms were shivering, the father thought he was shaking. He said he picked him up as he thought he may have been cold.

39. The parents arrived at the surgery at about 9.10 am for a 9.20 appointment. They saw the GP, Dr Jakhra, at 9.28 and left the surgery at about 10.05. According to the records the history was given by the father that *'since this morning, parents have been worried, as he had not been crying/making any noise, not opening mouth, tongue stuck to mouth and having fit like symptoms – jerking a little but no loss of consciousness. No vomiting, diarrhoea, fever. Not on any meds'* Dr Jakhra provided a statement and gave oral evidence. He was a student doctor in his second year. He said he took a history from the parents whilst Jayden was in front of him being held by his parents, he saw them undress him, he then examined Jayden and then saw the parents dress him again. On his examination he noticed Jayden was moving all four limbs spontaneously, they did not look abnormal in the way they moved; the movements weren't jerky. The limbs were not stiff when he moved them. He described Jayden as 'Alert'. He said he was concerned enough to seek the advice of a senior GP who advised referral to the UCLH Paediatric walk in clinic. The GP wrote a short referral letter, printed off the notes, checked the parents knew where to go and advised them to go there. They went by public transport. The mother said her main concern was that Jayden had not been fed. Dr Jakhra said that there were no indications on what he saw that Jayden was suffering from a life threatening condition.

40. I have seen the CCTV footage that follows some of the journey the parents made to the hospital. In particular there is footage of him on the bus sitting on his mother's knee, where he appears to be awake and does not appear to be stiff and posturing. There is nothing unusual about the way they behaved (on what could be seen of the journey) and was consistent with it not being an emergency.

41. The parents arrive at UCLH at 10.49. The father is carrying Jayden and the mother is pushing the buggy. They went into the general reception area, where they had gone on 17.709, and were directed to the rapid referral unit.

42. They were first seen by a Nurse Edwards. She said in her statement and oral evidence that she was immediately concerned that Jayden was convulsing and was likely to need urgent medication so he needed to be weighed. Whilst she was weighing him he noticed that his right leg was shaking. She said she was so concerned she went to see the paediatrician Dr Sutcliffe, even though he was with another patient. She described what she had seen, he saw Jayden and the parents but did not note any abnormal movements. He asked the parents to wait outside with Jayden and complete the questionnaire. According to the parents he said Jayden

looked 'fit as a fiddle'. Dr Sutcliffe did not recollect saying that. However, something had re-assured the mother as she phoned her mother and said Jayden seemed to be fine. This was confirmed by her mother in her evidence. Nurse Edwards said she was concerned about the baby's condition. She asked a play therapist Liz Wilkinson to sit with them, she noted what appeared to be slight intermittent convulsions on Jayden's face and one arm appeared immobile. Nurse Edwards again requested Dr Sutcliffe to see Jayden straight away. She went to get them and found them in the weighing room, changing Jayden's nappy. She took them into to Dr Sutcliffe.

43. Dr Sutcliffe's note of the consultation (written in retrospect much later that day) states he was asked to see Jayden because of Nurse Edward's concern about '*abnormal movements ?fits'* and on examination he had abnormal movements. In his statement dated 6.8.09 he doesn't mention the first time Nurse Edwards brought Jayden in to see him. In his oral evidence, when reminded, he could recall this sequence but did not observe any concerns when he first saw Jayden and that's why he asked them to wait outside. He said had Jayden been jerking there and then on his first examination he would have prioritised him but he wasn't and the evidence of seizures was intermittent. He did not see the need to interrupt the consultation he was part way through.

44. When he saw Jayden a second time he thought he was clearly neurologically abnormal; he was moving his arms up and down slowly. Rather than administer any drugs, he made an immediate referral to the resuscitation unit by A&E. He was questioned why he did not administer any medication himself or call the unit to him. He said the unit was close by and he considered it was better that Jayden was taken to them. A SHO accompanied the parents. By the time he wrote his clinical note it was known that Jayden's condition had deteriorated significantly. He said when he gave evidence that this had been a traumatic experience for all the staff. The parents submit that with hindsight and in the knowledge of the criminal investigation he has sought to portray Jayden's presentation as being more serious than it was. This is supported by the fact that he did not volunteer in his statement the first time he saw Jayden or that Nurse Edwards had gone back in to ask him to see Jayden. He said in the witness box that what he saw was '*decerebrate'* and, it is submitted the court should treat that description with caution as it is not in any records made around that time. Nurse Edward's description was that his condition was fluctuating.

45. There is a CCTV recording of some of the journey to A&E. They appear to proceed at a steady pace. It is possible to see Jayden move his legs.

46. On arrival at A&E they were met by Nurse Leech, she said Jayden seemed to be fitting, jerking and making an abnormal sound. She said all four limbs were twitching and he was making a strange gurgling sound. She said she had seen similar presentations before and she concluded Jayden was fitting. She undressed him, was able to move his limbs in doing so, and put him on a trolley. The records start at 11.25 and continue in 10/15 minute recordings until he is transferred to GOSH later that afternoon. Dr Salt, Consultant Paediatrician, arrived soon afterwards to make an assessment, she had been alerted by Dr Sutcliffe that he was referring Jayden as he was 'fitting'. Dr Salt was assessing him and Nurse Leech was making the recordings; they operate as a team.

47. The contemporaneous records begin with Jayden being assessed as being 'A' under the AVPU scoring, with 'A' being alert and 'U' being unconscious. Dr Salt said he was responding to pain, he was not unresponsive and his pupils were equal and reactive to light. This is not a stand alone assessment as it is closely followed by regular recordings following the Glasgow Coma Scale ('GCS'). This is a scale that gives scores under three categories; eyes, verbal response and motor response. It is used to assess level of consciousness in adults, under fives and under 12 months with different sub-categories. By definition this assessment must involve an element of subjectivity. Dr Peters said this was an out of date form, he had not seen one like it before and the categorisations had been re-calibrated for young children. Dr Salt, in her oral evidence, thought she may have scored Jayden slightly differently. The score recorded at 11.35 is 13/15 (on one view it should be 13/14 as the motor response category on this form only has 4 sub-categories). It was not suggested that this was a form that was unfamiliar to those who worked in UCLH and it's reasonable to infer they had been using it for some time. According to this record Jayden is recorded with a score of 13/15 at 11.35 am which then drops to 6/15 at 12.20 pm. Nurse Leech said she understood the difference between tonic/clonic seizures and decerebrate posturing. She explained the differences in Jayden's GGS scores. Once you get to a score of 8 you are unable to protect your own airway hence the need to intubate. The decision to intubate was made at 12.30pm; he was not intubated until 1.55pm because of the need to ensure proper preparation and to alert the anaesthetic team. It was clearly a busy time, swine flu had just broken out and Dr Salt said there was one other child under her care who was in an equally serious condition. Her note was written in retrospect later in the day. Dr Salt said Jayden appeared to be fitting and had tonic/clonic seizures, she agreed his jaw being clenched was a sign of the tonic part of the tonic clonic convulsion.

48. Dr Salt implemented the hospital protocol that deals with children who have prolonged fits; intravenous access is utilised to administer anti-fitting medication. He was administered lorazepam twice (11.35 and 11.40) as he had not stopped fitting. In her statement (dated 25.7.09) she said after the second dose the jerking movements subsided, his tone remained increased he had *'decerebrate posture'* which she describes as *'abnormal posture of his upper arms and clonus of his lower limbs that was sustained'*. She said she loaded Jayden with another anti-convulsant drug, Phenytoin, at 12 noon and asked the anaesthetist to attend to intubate Jayden. She said the procedure is the registrar attends first, makes an assessment and then contacts the consultant, Dr Pritchard. The necessary preparations are then done and then intubation takes place. Although the request was around 12.30 Jayden was not intubated until just after 2pm. An x-ray was taken before and after intubation. One of the difficulties in trying to work out the sequence of events is that apart from a note written in retrospect and some references in the running clinical note there is no statement or further detail from the anaesthetist. She said she discussed the intubation with the anaesthetist who attended and discussed Jayden's case with the retrieval team, making a request for Jayden to be moved to Great Ormond Street Hospital ('GOSH'). Jayden had been given antibiotics to cover any possible encephalitis. Dr Salt then left to deal with another child who was in a serious condition. She confirmed the unit was very busy that day. Dr Salt agreed that once Jayden was intubated and sedated it would not have been possible to tell whether or not he continued to have seizures or not. She said, when she was recalled to give evidence, that phenytoin worked over 24 hours and thiopentone, a muscle relaxant,

had anti-convulscant properties. The parents had been by the bed at the early stages but were taken to the relatives' room when Jayden was intubated.

49. The plan, according to Dr Salt's note and which she accepted was for him to go for a CT scan and he was expected to return to A&E in about an hour. Dr Salt accepted when she was recalled that once Jayden was intubated the anaesthetists are in charge of that aspect. Apart from when she was contacted by the radiologist, Dr Shaw (about the wrongly placed intubation tube and possible metabolic disorder), her involvement thereafter was limited to the practical arrangements about contacting the retrieval team, making a fall back plan if Jayden needed to remain at UCLH and getting the parents consent to the MRI. She was unaware, for example, about the changes in the CO2 levels or kept up to date about that. This is supported by the fact that the clinical records record the blood gas levels at 11.41 and 12.43, thereafter there is no clinical record of them (other than the computer print out of the recordings) or records of any actions taken on them.

50. The records provided by Dr Shaw provide the chronology for the imaging. Jayden's CT scan was booked at 13.38 and carried out at 14.39. The medical notes confirm the first attempt to intubate Jayden was at 13.59, it failed and a second attempt was made which was successful at 14.03. After intubation an x-ray was taken between 14.08 and 14.12 to confirm the position of the tube, Dr Shaw reviewed the chest x-ray at 14.34. She identified the tube had been put in too far and the left lung was starting to collapse. She said she called Dr Salt immediately about this. By then it is likely that Jayden was enroute to the scan, or was already there. There is a note in the clinical records that the tube was pulled back. It is unclear how long after the alert from Dr Shaw this was done. It appears Jayden was in this position for at least 25 minutes. Dr Salt sought to suggest that this could have been done earlier by the anaesthetists, but no time is given by the clinical recording and if it was done in the radiology department there would have been no need for Dr Shaw to contact Dr Salt, as she said she did after viewing the x-ray. It was during this time that the capillary blood gases show that Jayden's CO2 level rose significantly to 11.3 (nearly twice the normal level). The next effective reading was nearly 2 hours later as the test done at 15.13 was insufficient to measure.

51. Dr Shaw said she made a second call to Dr Salt following her review of the x-rays as she was concerned about a possible metabolic disorder due to the appearance on the images (in particular at the top of the left humerus), her note records *'..There is some irregularity of the metaphyses of both humeri. The significance of this is uncertain. Is there any clinical support for Rickets?'* Dr Salt responded that the calcium levels were normal.

52. Jayden's CT scan took place at 14.39, he was booked in for an MRI at 15.00. The time written on the MRI images range from 16.38 to 16.55. Dr Salt said she didn't know the reason for the delay, sometimes they need to wait for a machine to become available. Dr Salt said she had discussed Jayden with the neonatal team in the event that he remained at UCLH. The records record that team arriving at 17.00, Dr Salt discussed the MRI with Dr Salwood and Dr Kenal and Dr Salwood advised a CT of the spine. At 17.15 Dr Salt ordered a CT of the spine which was performed at 17.36.

53. On viewing the MRI scan at UCLH, Dr Dawn Saunders described widespread swelling and infarction of the cerebral hemispheres, with some sparing of the left occipital pole. The posterior fossa, brainstem and cerebellum were normal. There was thin film blood over both hemispheres with more over the left hemisphere, extending under the temporal lobe. There was also fluid over the posterior fossa surrounding the cerebellum and there was layering of the blood (mm only). Layering can take place before the blood has clotted and is likely to occur within 48 hours but more likely within 24 hours. There was soft tissue swelling to the back of the head on the right, which correlated with the site of fracture on the CT scan: Dr Saunders linked the two. Beneath the soft tissue space was a small collection of blood, which looked like it lay in the extradural space.

54. At 17.55 Jayden was commenced on morphine, atracrum and midazolam. This was the first anti-seizure medication that had been administered since midday.

55. Jayden was brought back to A&E at 18.20. The retrieval team (Dr Runnacles and Nurse Mogridge) were there. By this time Jayden was in a critical condition. In her statement Dr Runnacles said she had spoken to Dr Salt when they were enroute to UCLH, she said she advised 3ml of 3% saline to treat possible raised ICP in view of the *'high blood pressure'*. Dr Salt and Dr Pritchard did a verbal handover when they arrived. She examined him at 18.40 she *immediately noticed that his left pupil was dilated and unreactive (size 7) and his right pupil poorly reactive and smaller (size 3). These had been recorded as being equal and reactive 30 minutes previously on the nursing observation chart. He was hypertonic, fittery with decerebrate posturng, this together with his low heart rate and raised blood pressure is a clinical indication of raised intra cranial pressure*'. She immediately gave him a bolus of 3% saline solution and was advised by the consultant attached to her team to continue giving him boluses. He was given 9 3% saline boluses over the next hour to seek to reduce the ICP; 7 were between 6.45 and 7.15. She noted at 18.45 Jayden showed some abnormal tonic movements in upper and lower limbs, she gave him lorazepam and a loading dose of phenobarbitone to treat ongoing seizure activity. This settled the movements. She said she made the decision to move him to GOSH, stabilised his CO2 level (to between 4 – 5, by increasing the rate on the ventilator). Both she and Dr Salt explained to the parents and the MGM (who had arrived by that time) the seriousness of the situation and their concern that Jayden may not survive the journey to GOSH. The CCTV taken in the hospital graphically shows the enormous distress caused to the parents by this news, in particular the mother who is seen to collapse to the floor. The retrieval team were able to accommodate taking the parents in the ambulance and took Jayden to GOSH. By the time they arrived at GOSH his left pupil had reduced from size 7 to size 3 and his right pupil remained at 2, both poorly reactive. They arrived at GOSH at 19.45 where the retrieval team handed over to Dr Walsh and Nurse Cottrell.

56. Jayden was reviewed on the ward at 22.10 by Dr Peters, Consultant Paediatric Intensivist. He noted in the records *'in the absence of any explanation this has all the features of inflicted head trauma'*. Arrangements were made for the parents to stay at GOSH and MGM went home.

57. The parents were arrested at the hospital at 1.30am on 23.7.09, taken to the police station and kept overnight; they did not see Jayden again.

58. At 5.30 am on 23.7.09 Jayden's left pupil blew. At 10 am on 23.7.09 the police informed GOSH that no information about Jayden or the whereabouts of the parents were to be given to the wider family. At midday an EEG and SNEP were conducted and showed total brain inactivity.

59. At 2pm on 23.7.09 Jayden's right pupil blew. At the same time a strategy meeting was held at GOSH attended by representatives from the police, LA and the hospital. The police requested no visits by the parents to Jayden's bedside and the LA vetoed visits from other family members.

60. Jayden had a skeletal survey at 15.00 23.7.09**,** this was reviewed by Dr Hiorns, consultant paediatric radiologist, together with another radiologist. She identified the fractures and was unaware Jayden had rickets until she was informed when she gave evidence at the CCC. She did not identify any signs from the images. She was recalled at this hearing after Dr Barnes gave evidence. He had pointed out on the images during his evidence the radiological signs which he said identified rickets. Dr Hiorns disagreed and said the changes that were pointed out to her were, in her view, in the normal range of changes. She was unaware that Dr Shaw, the paediatric radiologist at UCLH, had raised a query regarding rickets.

61. At about 4pm on 23.7.09 Jayden was reviewed by Dr Nischal, a consultant ophthalmologist. He observed widespread bilateral retinal haemorrhages extending beyond the equator. These were, in his experience, at the severe end of the spectrum and only seen by him in association with trauma.

62. The parents were interviewed on the afternoon of 23.7.09. They were released on police bail with a condition not to attend GOSH.

63. At 3pm on 24.7.09 a meeting was held at GOSH to obtain parental consent to end of life treatment. The parents were not allowed to see Jayden. They asked for him to be christened. Neither they or the wider family, were allowed to attend the ceremony.

64. At 5pm on 24.7.09 Jayden was extubated. He died at 03.35am on 25.7.09.

65. There was unfortunately a delay in carrying out the post mortem due to a debate between the Coroner and the Metropolitan Police as to who should take the lead in the post mortem. An arrangement was arrived at where the Coroner took the lead, instructing Dr Scheimberg and Dr Rouse to conduct the post mortem with Dr Cary present as an observer on behalf of the police. The post mortem was carried out on 9.8.09. I deal with what took place when I consider the evidence of the pathologists below.

Dr Peters

66. At GOSH from admission until he handed over to Dr Agrawal on the afternoon of 24.7.09 Jayden was under the care of Dr Mark Peters. He is a Senior Lecturer in Paediatric and Neonatal Intensive Care, which is a research position at University College London he has held for the last three years. He has been employed by GOSH since 1993, has been a Consultant in Paediatric and Neonatal Intensive Care since April 2000 and that still forms the vast majority of his work. He is also a

Consultant for CATS (Children Acute Transport Team) since its inception in 2001. In his role he has been involved in the care of between 1,200 and 1,500 critically ill children each year, including on average about 30 serious head injuries.

67. He said the referral in relation to Jayden had been made by Dr Ram, from the CATS team. He understood that a CT scan had been performed at UCLH and this subsequently excluded a requirement for a neurosurgical operation, but did reveal subdural blood and a skull fracture. He said in his section 9 statement dated 6.8.09 *'It was clear to me and the CATS Team, that trauma was the most likely precipitating cause given the CT scan findings.'* He said Jayden was received by Dr Walsh. Dr Peters spoke to Mr Harkness (who was the on call Neurosurgical Consultant) they discussed a management plan and agreed to continue maximum medical therapy, but recognised Jayden was likely to die from these injuries. Dr Peters saw Jayden at 22.10, examined him and reviewed his CT scan. His opinion was that *'this was an unsurvivable severe brain injury. No other explanation other than trauma appeared credible to me then or now.'* He said he saw the parents, was struck by their flat effect and no visible signs of emotion, although notes that the mother appeared distressed. He said he explained to them his view was this was as a result of head injury.

68. The following day he said there was no sign of improvement; EEG tests confirmed the extreme severity of the brain injury. Jayden was reviewed by a number of specialists, in particular Dr Liesner (Consultant Haematologist) who advised a series of blood tests for abnormal bleeding tendencies; Dr Grunweld, a Metabolic Consultant, to consider the credibility of a metabolic disease causing the whole picture; Dr Ken Nischal (Consultant Opthalmologist) who confirmed the presence of bilaternal haemorrhage; Dr Rob Robinson (Consultant Neurologist) and Mr William Harkness (Neurosurgical Consultant).

69. On 24th July Dr Peters handed over the care to Dr Agrawal, the on call Paediatric Consultant.

70. In his section 9 statement in August 2009 Dr Peters said *'In my view the only credible cause of the fatal combination of sudden onset of severe and irreversible encephalopathy and subdural bleeding, skull fracture and bilateral widespread retinal haemorrhages in this case is a severe head injury. I believe the encephalopathy would be immediately apparent after an injury of this severity. There is nothing in the history given to me that suggests or provides an explanation. The fractures seen on the skeletal survey of varying ages and locations similarly have no explanation of which I am aware other than repeated trauma.'*

71. In a section 9 statement dated 2.11.11 (just prior to giving evidence at the CCC) he stated *'I have been asked to attempt to summarise the tests done at University College Hospital and Great Ormond Street Hospital into the causes of Jayden Wray's sudden collapse and death in simple terms.'* His statement then outlines the tests undertaken at GOSH. He confirmed that having seen all the expert reports his opinion remained the same, that trauma was the only credible cause of the full combination of Jayden's signs and symptoms. He briefly considered the differential explanations in those reports and concluded *'Trauma, in contrast, explains every finding. In my opinion it remains the only credible cause.'*

72. When he gave oral evidence at the CCC it became clear he had not seen the clinical records from UCHL, although he had seen the various medical reports that summarised the history from these records. He said he was struck by the results of the EEG tests. He said that was in his experience unusually severe so soon after an admission. In relation to the various tests undertaken he said the calcium level in the blood was slightly low and with that, a related enzyme, something called alkaline phosphatase, was slightly high. He said this was a common finding in children who come to intensive care. He said his opinion following the tests is that there were no signs of a significant infection in Jayden. In his oral evidence in the CCC he said *'So my observation of these tests in Jayden's case is that they were not significantly abnormal and if he was suffering an immediately life-threatening infection I would expect them [white blood cell count and CRP] to be really very profoundly abnormal indeed, and they were not.'*

73. He said the presence of the skull fracture was not crucial to his conclusions; his opinion relied on acute severe encephalopathy, intracranial bleeding, brain swelling, retinal haemorrhages in both eyes and in more than one layer. He said he had only seen this combination in the context of a trauma. He said he makes no judgments at all about the adequacy of parents or otherwise *'I make my judgment on the clinical, radiological and laboratory findings'.*

74. He confirmed he stood by the statement in his section 9 statement that he believed encephalopathy would be immediately apparent after an injury of this severity, he continued '*…My point is that having suffered a head injury of sufficient severity to cause these other findings, my view is that unconsciousness would have been apparent immediately or within very few second or [one or two] minutes.'* He said everybody struggles with how you define unconsciousness in an infant; it is a challenge to diagnose. He said what you would expect is either not crying or a very abnormal cry and movements that were either absent or different to normal, and eye opening or movements that were either absent or different to normal. He continued *'I believe that a baby who had suffered a head injury resulting in these findings would immediately be recognised by most people as very different to normal, and I think most people would recognise something was terribly wrong'.*

75. In his oral evidence at the CCC he was taken through the early history on the 22nd July, which he was not aware of, for example the detailed observations of the GP, the fact that the parents had been sent by public transport from the GPs to UCHL. He agreed a GCS of 13/15 was not what he would have expected but questioned the reliability of this method of assessment in such a young child. In his evidence to this court he said the UCLH notes were not in an up to date format for young children. He saw the UCHL notes for the first time in the witness box at the CCC. It remained unclear to me, when Dr Peters gave evidence in this hearing, precisely what the arrangements are for the transfer of clinical notes on a transfer to GOSH. Dr Peters described them as *'chaotic'* and there was no system in place on admission to request them if they had not all been sent.

76. He looked at the blood gas readings whilst in the witness box at the CCC and said *'….if you do not control breathing close to normal, carbon dioxide builds up in your blood, and that controls how much blood goes to the head, and simply put, if you have too much carbon dioxide in your blood because you are not breathing enough,*

too much blood goes to your head and the pressure goes up, and so after unconsciousness of whatever cause, but it is best understood after head injury, one of our jobs is to make sure the carbon dioxide level is just acceptable, just at the top end of normal.' When he is taken to the CO2 level at 14.24 on 22.7.09 which had increased to 11.3 he said *'That is not helpful. That would definitely have increased the pressure in the brain during that episode.'*

77. He referred to a handwritten note at the top of one of the clinical pages that says ETCO2, he said that stands for end tidal CO2 and continues *'..what I cannot see is where the measurement of that – so this is the way that we measure continuously carbon dioxide after we have intubated a patient, and good practice would mean they had done that.. I cannot see where that value corresponds..'* He continued *'If the blood gas is the only information we have on the carbon dioxide level and it is in double figures around 2.30pm..then that is much too high and will not have helped the brain Just to put that in context, though, nearly every road traffic accident who has a significant head injury will have raised carbon dioxide levels, and it is one of the things that contributes to the severity of the injury, and good care involves controlling that.'*

78. It was put to him in cross examination at the CCC that his original opinion included the skull fracture and he now accepted that due to the dating the skull fracture was too old. It was suggested to him that that meant the triad was left but with no evidence of external trauma, he replied *'Is that correct? I am not up to date on the views of all the fractures'*. He maintained his opinion that the likely cause of the brain injury was trauma. He agreed from the clinical notes the GCS and the AVPU were not consistent with trauma bringing on these things and causing immediate encephalopathy, but again cautioned about reliance on these methods. However, he agreed that in his experience in a collapse caused by a shaking mechanism there would be a significant and substantial change in that child's presentation. That was the typical pattern that came to him, although he said in the literature and other clinician's experience there were less severe cases that don't result in an immediate collapse and disturbance to breathing. But that, he accepted, would not be consistent with the severity of the scan results.

79. He said there can sometimes be difficulties in placing too much reliance on clinical observations due to their subjective element. He relies on the more objective data (i) CT scans (ii) the natural history in comparison to his experience of cases of encephalopathy previously and (iii) lactate levels on admission.

80. In his evidence in this hearing, Dr Peters was taken through the blood gas readings again. He said the increase in CO2 noted at 14.24 was an *'adaptive response'* he said it allows you to deliver oxygen more effectively (something called the "Bohr effect"), it changes the way the oxygen carrying molecule works and allows you to offload that into the tissues more easily. He said when those sort of levels are seen *'we are not concerned unduly, but in a situation where someone already has signs of severe brain injury we want to correct that CO2 down towards normal'* In relation to the difficulties with the endobronchial intubation, it is of concern if there is a low oxygen level, he said it was recorded as 100% during his time at UCLH so there was no sign of hypoxia; '*One of the problems I have with the suggestion of a hypoxic-ischemic injury later is that he wasn't hypoxic.'* He said a degree of CO2

retention is not ideal but they would see this every day on the ICU. This appeared to be a more relaxed response to this evidence than he gave at the CCC. He said the lactate levels were supportive of this, on admission the lactate level is very high, 7.5. The normal level would be less that 2. This is evidence of an oxygen debt; Jayden has consumed more oxygen than he has delivered when he comes into hospital. Professor Nussey said this is consistent with repeated seizures; Dr Peters agreed *'seizures alone for many hours could do it'*.

81. Dr Peters said the subsequent reduction in lactate level after admission to UCHL means there is evidence of improving perfusion, or there is no evidence of hypoxia or, in his opinion, ischaemia subsequent to admission to UCLH. When asked whether these readings caused Jayden any complications he replied *'No, quite the opposite, actually. I think that what you're referring to is that there is blood gas where he has a low carbon dioxide tension…I think the suggestion that that is harmful is based on an incomplete picture.'* He referred to the pH level of 7.17 which shows the blood is acid, he said the effect of CO2 falling is the opposite to that; it would raise your pH. He said if you look down the page to see what has caused that acidosis and he said this is evidenced by the low concentration of bicarbonate. He said *'The way anybody who looks at blood gases day in day out would interpret this, this is metabolic acidosis. The change in bicarbonate concentration has caused the pH to fall and the CO2 being low is an adaptive response to that. If you did not have the CO2 low in this situation you would cause more harm, because the pH would fall further and severe acidosis is dangerous.'* He said the core issue is the influence of the pH and CO2 on how much blood is flowing to the brain. He said what determines how much your blood flows to the brain is the pH of the cerebrospinal fluid, which in the same way as the blood is determined by a combination of bicarbonate and CO2 level. Having a low CO2 level here means that the brain pH would be closer to normal than if the CO2 was normal or high. He compared it with diabetic patients, when they first come into hospital they have a very severe metabolic acidosis, they have a very low pH. What they do to protect themselves is breathe faster and faster to bring their CO2 down, he said that's analogous to this situation. The low CO2 in that situation helps maintain the blood flow to the brain. The bicarbonate level on admission was 19.4, by 17.17 it is 9.5. In answer to the question as to what process would cause the bicarbonate level to halve he said *'It's not clear from the notes what has done that, possibilities include him becoming…vasoconstricted, him closing down many of his blood vessels so that there are areas of skin that are not receiving such generous blood flow. I can't see signs of that in the notes. Another possibility is he is passing large volumes of urine…[or]a lot of chloride administration in the sodium chloride…I wasn't able to work out what had caused that with any certainty.'*

82. However, vasoconstriction is challenged by other factors, including the low lactate level. He said he can't be confident about any of these explanations because of the detail of the clinical notes. He said it could be explained that you have taken the lactate from an area that is well perfused and the non-perfused areas are not at all perfused so they are not contributing to the circulation. He accepted that high carbon dioxide can definitely worsen a prior brain injury; its impact depends on how fast you get there and the prior state of your brain. The worse Jayden's brain was prior, the more harm he stood to suffer as a result of any rise in CO2. He said you

can't have both he was either encephalopathic and at risk or he was not encephalopathic and not at risk.

83. In his clinical experience he said this is a familiar clinical picture, it would be very common to see a child with a profound and prolonged episode of seizures who then, either as a combination of the seizures or of the drugs given to stop the seizures, suffers a respiratory acidosis to this level or beyond. He does not see this sort of widespread ischemic encephalopathy in that scenario. However, he accepted that it is possible with the oxygen observations to have brain ischemia, that it is possible to have regional ischemia in that one area of the body is not getting adequate blood flow. He said the fact that there is no ischemia any where else makes it less likely.

84. He recognised the management of the CO2 in UCLH was suboptimal, but says the levels are well within that which is seen in most seizure cases and the vast majority of trauma cases, he did not think it had a major effect. It is the sort of range he sees in clinical practice and doesn't, in his view, explain the fact that Jayden's scans were so abnormal so early. He said he was happy to accept that there could have been better monitoring at UCLH but there is more than one explanation than treatment was not adequate. The other explanation being that the condition was progressive and that is the natural history after whatever cause of encephalopathy with time. The natural history of a brain that is profoundly injured is it continues to swell, and when it swells enough you see the pupils change. He said in his view electrical seizures without a systemic component don't cause the brain to die rapidly. He said that is supported by population based research from Professor Rod Scott. He accepted that it was not recorded in that study whether any had vitamin D deficiency, but said the mechanism of early death was not hypoxia ischemic encephalopathy in any of that series. He said all he can offer is his clinical experience and his understanding of the literature. He said vitamin D insufficiency is not particularly rare and there is an overlap between the epidemiology of vitamin D deficiency and insufficiency and critical illness in children. If it was unique to Jayden it needs an explanation why it was unique to him, because he said he would expect to see this pattern more often, given that vitamin D deficiency is not so rare.

85. He said the main problem was trying to reconcile all these bits; no explanation seems to fit them all. It is his view from the observations in hospital there is no hypoxia, there's no apparent ischaemia, the lactate level is 4; he considered there was no evidence of hypoxic ischemic injury in hospital. There is a strong indication of hypoxic ischemic injury prior to hospital in the high lactate and the maturity of the findings on the imaging. In his oral evidence to this court he agreed either explanation (trauma or seizures) is challenged by Jayden's condition on arriving at UCLH, whether it's prior seizures or whether it's trauma. The reason he favours trauma is the presence of the subdural haemorrhages which he later changed to '*..and the main component of that is the subdural bleeding for me'*. He said the only situation in which he has seen seizures cause an acute hypoxic death in this kind of time frame is when somebody had a series of fits in the bath and drowned as a consequence, and his heart stopped before he came into hospital. He agreed that once Jayden had been paralysed you wouldn't know that the seizures had stopped unless you had an EEG, but he said you could get clues if you're tuned into looking at monitors, seizure activity often looks like an unexpected change in heart rate or blood pressure. He said his experience is that electrical seizures without clinical

movement associated with it are a completely different clinical problem than are the full bloodied shaking around situation. The main danger is you lose control of your airway or your circulation. He said his experience in treating children with epilepsy is that very rarely a child that has been fitting for weeks when it's only electrical fits and the manifestations in the rest of the body are controlled they do die, but it takes many days or weeks for them to get into that condition.

86. His evidence is clearly important on two fronts; as the treating physician and his long experience in dealing with acute paediatric situations. Against that it is said he reached a clear conclusion very early (on one view prior to Jayden's admission to GOSH) which has made it increasingly difficult for him to re-consider in the light of the evidence of the clinical picture. This is complex area of medicine where, it is submitted, the court should be wary of practitioners who adopt a too dogmatic approach. It is submitted the change in emphasis in his evidence from the CCC and this hearing regarding the impact of the CO2 levels, the level of consciousness observed and subdurals show a tendency to elevate matters that had perhaps not had the same significance before in order to justify his early conclusions.

87. He did accept the combination of low calcium levels and infection causing seizures at some early point and continuing for many hours is a credible cause of Jayden's encephalopathy but, in his view, it doesn't explain the SDH and retinal haemorrhages. He said *'....I am saying there are two candidate explanations [seizures or trauma]. Both suffer the same weakness that in order to – in order to explain the subsequent natural history, they have to have been significantly worse than we have in our history pre-hospital.'*

Agreed findings at the post mortem

88. The post mortem was conducted on 3.8.09. The parties submitted a document at the start of this hearing that set out the agreed findings at the post mortem:

At the post mortem on August 3rd 2009 the following findings were made:
External Examination
(i) Various marks consistent with therapy
(ii)No old or recent marks of injury

Internal Examination
Sub-scalp bruising on the right parietal/occipital region over an area of 5cm x 5cm. Associated sub-periosteal bleeding from the midline to the right over the occipital bone over 3cm x 1cm area. A thin film of subdural haemorrhage over both hemispheres [more on the left] adherent to the dura. Some thin film subdural haemorrhage involving the skull on the left side. A suspected underlying fracture on the right occipital bone.

Radiological Findings confirmed by later bone histopathology-
1. Left hand: *1st left metatarsal: Incomplete mid-shaft fracture. 2nd left metatarsal: Incomplete mid-shaft fracture and crack fracture towards the end of the bone. 3rd left metatarsal: Complete mid-shaft fracture.4th left metacarpal: Double complete shaft fractures.*
2. 2 left ribs, costchondral area: Abnormal growth plates attributable to established rickets.
3. Left proximal humerus: Fracture of the proximal humerus between the growth plate and the bone (a metaphyseal fracture).

4. Left proximal femur: Fracture of the proximal femur at the interface between the growth plate and the bone (a metaphyseal fracture).
5. Right distal humerus in two places (old fracture).
6. Proximal humerus: Fracture of the proximal humerus at the interface between the growth plate and bone (a metaphyseal fracture).
7. Right mid-tibia: Old healed greenstick fracture and a complete fracture of the shaft.
8. Right parietal bone: A displaced fracture of the skull bone.
9. Right occipital bone: A traumatised fissure.
10. Left radius: Incomplete crack fracture of the shaft with rickets affecting the growth plate.
11. Left ulna: Rickets in the growth plate.
12 .Right radius: Rickets affecting the growth plate.

The Brain
Subdural haemorrhage, global ischemic injury, ischemic axonal injury and axonal injury to the pyramids and spinal nerve root axonal injury.

The Eye
Fresh retinal haemorrhages in both eyes with bilateral papilloedema (optic nerve head swelling). There was also subdural bleeding associated with the optic nerve.

The LA's case

89. The LA's case is that these findings are the result of non accidental injury caused by one or other of the parents. In relation to the fractures they rely on Professor Malcolm's conclusion and allege that items 3 – 9 (excluding the greenstick fracture of the right tibia) on the agreed list of fractures/radiological findings were as a result of inflicted injury. The remainder they accept were caused by rickets and rough handling. In relation to the findings regarding the brain injury and retinal haemorrhages they rely on Dr Cary's view that they are compatible with shaking and/or impact events. Their case turns on the medical evidence. They rely on the medical evidence of fact and opinion to undermine the credibility of the parents.

Expert evidence

Pathologists

Dr Scheimberg

90. Dr Scheimberg is a Consultant Paediatric and Perinatal Pathologist at the Royal London Hospital. She has been a consultant since 1997, prior to that she was a registrar at GOSH and UCHL. The job of a paediatric and pre-natal pathologist has two components: one is looking at the surgical pathology, which is looking at the biopsies that are taken from living children, in order to diagnose diseases to help in their treatment. As a paediatric and pre-natal pathologist she conducts post-mortem examinations of foetuses and babies. The foetuses she deals with can be as small as 12 weeks gestation through to babies of all ages, children and teenagers. She conducts two types of autopsies: one requested by hospitals with the parents' consent and post-mortems requested by the coroner, which do not require parental consent. In the last three years she has conducted around 600 such procedures each year. Her unit has done nearly 2,000 post-mortems over the last three years. In the last year she has conducted 40 coroner post-mortems and in the previous year 50.

The majority of the post-mortems are babies, rather than children. In relation to the coroner post-mortems the majority have been babies between two and seven months old.

91. Regarding her experience in dealing with cases involving Vitamin D deficiency and rickets she said she thought it is returning. Since dealing with this case in 2009 she said they started measuring vitamin D. She said 37 cases had vitamin D deficiency or insufficiency. Some of the children had radiological changes and some did not. The majority had abnormalities in the growth plate; some were florid but none as florid as was seen in Jayden.

92. She thinks the reasons why these cases have not been picked up before is that according to the literature they say you start seeing radiological changes after six months, rarely before that. She thought that was probably correct when considering acquired rickets (which was what Professor Malcolm described seeing in Glasgow in the 1970s). She thinks the difference now is that we are now seeing mothers who are vitamin D deficient, which increases the risk of congenital rickets. The difficulties are then compounded if the child is breastfed. That is often not picked up radiologically in young children as radiology is a gross tool; you see shadows not the reality.

93. She said she was instructed by the coroner to conduct the post mortem of Jayden with Dr Rouse, a Consultant Forensic Pathologist. The role of the forensic pathologist is to look at the aspects that are more related to trauma, her contribution comes from her expertise in dealing with children. Dr Cary, Consultant Forensic Pathologist was present as an observer at the post mortem, at the request of the police.

94. She said she was alerted to the possible existence of rickets from her reading of the clinical notes. Jayden had low calcium and high alkalised phosphates and she noted there had been a suggestion to measure vitamin D. She viewed the x-rays that had been taken at GOSH and noticed the edges of the wrists and humerus were splayed and there was fraying of the metaphysis. When she conducted the post mortem she said the first thing that is cut is the chest, she said she could see immediately very advanced rickets, the 'rosary'. She said the beading was inside. She said his rickets were, in her view, extremely severe for a child of his age. It indicated the length of the vitamin D deficiency. She said the only thing that would have been more severe would have been fractures in utero. She said this was the most severe case she has come across in her experience. She said the first cases in which they had a diagnosis of rickets in her unit was in 2002. The only other case in which she had seen abnormalities in the ribs in another post-mortem was in a child who had normal x-rays, but on examination had multiple rib fractures. That child was 14 months old. She had also seen a 2 month old child who had normal x-rays, when tested was vitamin D deficient and had 11 rib fractures (8 posterior) from resuscitation. She thought that child had congenital rickets and through the coroner they recommended the mother to start taking vitamin D. Her experience was similar to Professor Malcolm who had only seen one other case of this degree of severity in the last 30 years.

95. During the course of the post-mortem she snapped one of the ribs. She described snapping one of the ribs with her fingers by a flicking/twisting motion. This is confirmed by Dr Rouse. She said she has been snapping baby's bones at post mortems for 15 – 20 years; she does it as a matter of course. She said she found it *'brittle'* it was *'too easy to crack'*. She said she finds it hard to find the right word, but to her brittle means is that it's too easy to break compared to a normal bone. She said it did not feel like the rib of a 4 ½ month old child, it felt more like a newborn baby.

96. She also thought she damaged the occipital bone as she was removing it during the post-mortem. She told Dr Rouse and it was confirmed by a comparison of the pre-mortem and post-mortem x-rays. There was an issue raised by Dr Cary about this in the criminal trial as he said he had not seen it or heard mention about it at the time. This was not pursued in this hearing. She was asked about the strength of the skull bone, she said when you look at the x-rays the bone is not uniform, there are areas with more or less calcification. She said in her experience the bits with no calcium are more bendable whereas when there is little calcium it cracks very easily.

97. She described the process she undertakes to see if the bridging veins are intact. This is undertaken to try and see the source of the subdural bleeding. In this case she said it was a thin bleed. She said the bridging veins she saw were intact.

98. Her report after the post mortem did not give a cause of death. She said there were a range of differential diagnosis, which included trauma, seizures, hypocalcaemia. At that stage it was thought that the skull fracture was contemporaneous with the collapse. The initial report lists what has been done and then awaits further reports and tests. Various samples were sent off to specialist, in particular Dr Smith, Professor Malcolm and Dr Luthert. A blood test was taken from Jayden, the results confirmed the vitamin D deficiency and subsequent test taken from the mother confirmed she was too. This case has proceeded on the basis that she probably was vitamin D deficient during her pregnancy.

99. Once the various reports were back Dr Scheimberg completed her second report for the coroner on 8.1.10. In it she gave the cause of death as hypoxic ischemic encephalopathy and multiple fractures in a background of congenital rickets. She said for the coroner's court the standard of proof was the balance of probabilities; she said she could not tell one way or the other regarding the competing explanations.

100. It became apparent that she was not being called by the Crown in the CCC and she was instructed by the defence to prepare a report. She considered all the updated reports and the notes from UCHL (which she said were more voluminous than those she had seen as part of the GOSH notes she received prior to the post mortem). Her detailed report is dated 3.10.11. She said that although she thought it was right that trauma was on the differential diagnosis, however there were, in her view, more things pointing away from trauma, as when you looked at all the factors it *'just did not fit with trauma'*. The factors that pointed her away from trauma included (i) the low calcium level being a trigger factor for seizures; (ii) Jayden's seizures which could have been caused by an infection, the low calcium making him more susceptible to seizures; (iii) the raised white blood cell count and C-reactive protein;

(iv) the extent of the swelling of the brain; (v) the fragility of the bones and the fact that the fresh bleeding occurred when the child was in hospital; (vi) the lack of any external injuries consistent with trauma and/or shaking; (vii) other causes of subdurals than trauma; (viii) the lack of evidence from the neuropathologists to indicate trauma; (ix) the retinal haemorrhages could have been caused by the increased intra-cranial pressure which had peaks; (x) the presentation of the child was atypical, not the typical clinical presentation of catastrophic collapse as so often seen in children who go on to die from traumatic head injury; (xi) none of the fractures were contemporaneous with the collapse. As she said in her evidence *'There were too many things that did not fit in the trauma diagnosis. And I tried to mix them in all possible ways and there was always something that didn't fit.'*

101. She was pressed about the existence of the 5x5cm sub scalp bruise seen at the post mortem she said *'what I think may have happened is that when he had his skull fractures by some minor accident that nobody recalls, he could have had a bruising that was present there…it's very easy to get a bruise to re-bleed…what it doesn't do is point to is a severe trauma..how can you have fracture with minor trauma and then have just re-bleeding in a bruise with severe trauma without even causing a fracture? It just doesn't make sense.'* A little later *'The only thing I know is that it's [the recent bleeding] is not linked to the collapse, that it is likely to have been around 24 hours [before death], and I don't have a clue how it occurred. What I cannot understand is how a severe trauma would just cause a bruise when a minor trauma causes a fracture'*.

102. In relation to bone strength she thought she had asked Professor Malcolm to undertake bone strength tests. She said she asked him on the phone and followed it up with an email. The email is not in the clearest terms it's dated 15th September 2009 and states "*I've just checked the baby's mother's results in our computer. She also has a vitamin D deficiency, 23 nanomols, which means the baby was vitamin D deficient from birth. I think the non-decla bone would be very important in this case.'* She said what she meant was to do a test on the non-decalcified bone. She said once you decalcify the bone it becomes soft tissue that you can cut. She wanted him to leave some non-decalcified to see if the strength could be tested.

103. She agreed with Professor Malcolm's view that the fractures that were observed post mortem were all due to events after Jayden's birth. However, she disagreed with his view that only some of the fractures could be due to rickets and rough handling. She said *'my view is that the bones of this child were thin, brittle, and weak enough for any minor trauma to possibly cause the fracture, particularly when nobody noticed, 7 days before, 14 days before, maybe 10 days before…this child had sustained a skull fracture, despite the fact that he had been seen by doctors. It has to be something so minor that nobody realised that the child had fractures. Anyway it's a typical presentation…we're seeing a change in picture to which we have to adapt, because it's no more the rickets of the seventies of children with nutritional rickets that you could see after a certain age….the problem starts in utero.'* Jayden did not have any observable marks that would accord with the fractures being caused by a severe trauma. In relation to the bruise in the sub scalp she said it was a gelatinous swelling in which there was fresh blood that came at the time when he was in hospital. She considered the bruise on the scalp was caused by a minor trauma.

104. She was pressed about the existence of the subdural haemorrhage, she said in her view there can be other causes for a thin film subdural haemorrhage. In relation to the debate about whether subdural haemorrhages are caused by torn bridging veins and whether there was any point in looking for them at a post-mortem, she said in her view it was worth still looking at this as it may assist in trying to understand the source of the haemorrhage.

Dr Rouse

105. Dr Rouse is the forensic pathologist who conducted the post mortem with Dr Scheimberg. He too is very experienced. He gave evidence at the CCC but was not required to give evidence here. He did not disagree with the findings made by Dr Scheimberg.

Dr Cary

106. Dr Cary is a Consultant Forensic Pathologist. He has been a Home Office Registered Consultant Forensic Pathologist for 19 years and currently practises as a full time Consultant Forensic Pathologist within a practice of 10 partners.

107. He was an observer at the post mortem, at the request of the police. He provided a witness statement a week after the post mortem where he stated the provisional cause of death was head injury. It continued *'The finding of skull fracturing and thin film sub dural haemorrhage together with the findings in life of retinal haemorrhages and encephalopathy are in keeping with head injury, most likely as a result of impact. As well as skull fracturing multiple other bony abnormalities including fractures were detected on X-radiographs. The various sites of abnormality were sampled. A final opinion in relation to the nature, timing and likely origin of these will await detailed examination by Professor Malcolm.'* He made clear this was a provisional report. His final report is dated 3.3.10. It contained, in my view, somewhat over emotional language relating to the instruction of Dr Smith rather than Dr Al-Sarraj (the latter he had requested), whilst not (apparently) questioning Dr Smith's expertise. In relation to Professor Luthert he seemed to imply that he had made no attempt to reconcile the ophthalmic findings in life made by Dr Nishcal when Professor Luthert's statement did not dispute them. In relation to the fractures he expressed the view that the right parietal fracture would have required *'substantial blunt impact on the right side of the head'* His conclusion was that the case shows the classic triad of thin film subdural haemorrhage, extensive retinal haemorrhages (based on clinical findings) and the sudden development of encephalopathy characterised principally as hypoxic-ischemic brain injury. He said *'As there does not appear to be any competing alternative explanation for any of the components of the triad, in my opinion there is prima facie evidence that this is an example of paediatric head injury of the shaking/impact type.'*

108. He confirmed in his oral evidence that his view remains that the cause of death is head injury. He disagrees with the view held by Drs Cohen and Scheimberg that there is a possibility of an association between hypoxic ischemic brain injury and low volume subdural haemorrhage; in his view such an association is fundamentally flawed. He was concerned that it was being used to support a hypothesis. He said it

was unclear what their case material consisted of, he considered there was a selection bias as in his view it was chosen for the presence of what was called intradural (bleeding into the dura) and (on some occasions) subdural haemorrhage. All the babies selected had been through some sort of birthing process so they would all have been subjected to trauma of birth to a greater or lesser extent. He said hypoxic brain injury is very common in dead neonates and foetuses. In his view it is no surprise that whatever the precise selection criteria were, a number of these cases had subdural haemorrhage because they had all been exposed to birth trauma. The association is no more than that. He acknowledged some were by caesarean but said in his view it would make no difference as there was likely to be trauma if it was an emergency caesarean. His view was that the blood found in subdural haemorrhaging comes from rupture of bridging veins that run from the surface of the brain to the lining of the dura of the skull. He does not accept Dr Scheimberg's view that she saw the bridging veins intact as he said you can't see them all and they can be masked by the blood.

109. In relation to the sub scalp bruising he said that suggests there had been relatively recent trauma to that region.

110. He referred in his oral evidence at this hearing to the fact that the stomach was found to have 50ml of milk. He said that supported the view that '*something catastrophic'* must have happened not long after the last feed. He said this had appeared to become relevant to him following the suggestion from the criminal proceedings that there was not much major illness early on because Jayden was not in a coma. He then said *'It is fair to point out stomach emptying is an incredibly imprecise business. It is, though, nevertheless surprising there was still milk there many hours later.'* He went on *'I think the presence of milk, still present at autopsy, suggests that Jayden became critically ill at an early stage in close proximity to the midnight feed….that is not saying it is necessarily a head injury..'.* In relation to the fresh bleeding he said that told him that both the fissure and the fracture have been exposed to trauma at around the time of presentation, or at least the findings would be consistent with that.

111. He said much has been said about the triad. He drew a distinction between a baby presenting at hospital with those three things present and a baby who has had a full and detailed pathological investigation of each of those components where the underlying conclusion for each of those components is that trauma is either the only explanation or is by far the most likely explanation, he called that *'a worked up case of the triad'*. He said the differential diagnosis which had been put forward, in his view, did not stand up. He said if there was anything that broke the link between the three components of the triad that would mean suggesting trauma was the only cause was *'unsustainable'*. He accepted he was dependent on the specialists, in particular the neuropathologists and eye pathologists; '*If, for instance, the eye pathologist or pathologist said "I am not so sure about trauma now", that would mean I could not be so sure about trauma. I cannot take an independent view of the specialist areas of pathology. I rely on those others.'*

112. In relation to the history he accepted he had not seen the full UCLH clinical notes. He agreed breaking ribs was a standard procedure in a post mortem and that damage can be done to skulls.

113. He agreed that he had placed a lot of weight on Dr Smith's view. If Dr Smith had expounded caution in his evidence to this court when he said that the two matters that he had raised as indicating trauma were no higher than a possibility and not diagnostic of trauma he agreed it would diminish the likelihood of head injury. He then said *'I saw undoubted fresh trauma contemporaneous to presentation, so there has been an impact injury and no one was suggesting, I think, there was any easy likelihood of bruising here..so that does provide evidence of impact related trauma at the time of presentation. Similarly, the eye evidence stands in its own right as evidence of head injury and one may be left with the situation, as you can be left in these sort of triad cases, of one or two of the three being possible, or strongly supported but not definitive, but when taken together, the three lead you to inevitable conclusion. What I am saying is if the eye findings were equally only possibilities, I would agree with you, the whole thing would collapse.'* He agreed that if Dr Smith said that he can't give any conclusive evidence as to cause or suggest on the balance of probabilities this was inflicted injury he agreed a fundamental flank of the triad has been weakened.

Neuropathologists

Dr Colin Smith

114. Dr Colin Smith is a Reader in Pathology at the University of Edinburgh, he has a particular interest in neuropathology and is Honoury Consultant Neuropathologist for the NHS in Lothian. He has been a consultant neuropathologist for 11 years.

115. The brain was referred to him by Dr Scheimberg under the authority of the Coroner.

116. His first report was dated 7.10.09, it was replicated in identical terms in a section 9 statement on 16.3.10, he prepared addendum statements on 14.2.11 and 29.9.11 and took part in an experts meeting on 27.10.11. He gave evidence at the CCC and gave evidence in this hearing.

117. He was sent samples from Jayden's brain for examination. His examination was divided between a macroscopic and a microscopic examination. He comments in his report that that the examination he conducted demonstrated a range of pathologies. They can be summarised from his first report as follows:

(1) Acute subdural bleeding associated with focal intradural bleeding. This is seen in the cerebrum and also microscopically within the spinal cord. Within the spinal cord the bleeding extends along the spinal nerve route sheaths. The main pathology within the cerebrum is that of global ischemic brain injury. The ischaemia extends through the neocortex, hippocampi and deep grey nuclei and is associated with glial cytoplasmic swelling.
(2) Neutrophils (type of white blood cell) are seen overlaying the convexity. Dr Smith had considered acute purulent meningitis, but considered this neutrophilic infiltrate to be secondary to global ischemic brain injury. He was unable to demonstrate bacteria or fungi using special stains.
(3) The ischemic brain injury extends into cerebellum and brain stem and there is evidence of widespread ischemic white matter injury as highlighted by the

beta APP staining. He considers this to be secondary to brain swelling caused by global ischaemia.

(4) Within the cerebellum and midbrain there are one of two foci, which suggest a more established focal ischemic injury in that there is more prominent neuronal loss and reactive gliosis associated with macrophage infiltration. This raises the possibility of a separate less prominent ischemic injury prior to the main collapse of the child.

(5) Prominent axonal spheriods seen within the medulla suggesting a hyperflexion/hypertension type injury, although they have to be interpreted with great caution due to the widespread ischemic axonal injury.

(6) Also of concern is the prominent beta APP immunostaining of spinal nerve roots at the lumbosacral region associated with haemorrhage extending along the dural sheath again these need to be interpreted with caution.

He concludes that much of the neuropathology is non-specific and global cerebral ischaemia can be caused by a wide range of insults ultimately resulting in some degree of cardiorespiratory distress. He reported that there were two aspects that concerned him to a possible traumatic origin in this case (1) the presence of axonal spheroids within the pyramids, and (2) significant axonal damage to the lumbosacral spinal nerve roots with associate dural sheath haemorrhage. Finally, in his written report, he notes caution that the findings he made need to be interpreted very carefully in view of the widespread ischaemic pathology.

118. When asked for further clarification from junior counsel for the prosecution Dr Smith reported that the following features were suggestive or indicative of trauma (1) skull fracture (2) acute subdural bleeding (3) spinal nerve root axonal injury (4) axonal injury within the pyramids of the pons. The following features suggest an older injury (1) microglial nodules, and (2) axonal spheroids which did not stain with beta APP.

119. The experts meeting that took place on 27.10.11 was between Dr Smith, Dr Squier (Neuropathologist), Dr Ramsey (Neuropathologist) and Dr Scheimberg (Paediatric Pathologist). The note of the meeting records the following:

(1) All three neuropathologists agreed the following lesions were present: Subdural haemorrhage (SDH), severe brain ischaemia, brainstem injury (axonal spheroids), spinal nerve route damage and meningeal inflammation.

(2) With respect to the meningeal inflammation the differential diagnosis is between inflammation reactive to the hypoxic ischemic injury and partially treated meningitis. Whilst all agreed the former was more likely, they also agreed the latter can't be ruled out.

(3) With respect to the evidence of the head trauma contemporaneous with the collapse all participants agreed that there was no evidence of fractures or gripping marks. Dr Smith is noted as saying the presence of SDH was indicative of contemporaneous trauma but he agreed there was no other evidence to support trauma at the time of collapse.

(4) With respect to the old axonal injury in the brainstem all agreed that this was not contemporaneous with the collapse. Dr Smith noted that no studies have demonstrated how long axonal spheroids take to develop or how long they retain their APP immunolabelling in children, particularly when they are

secondary to global ischaemia. Dr Ramsay was concerned about the aetiology as the pattern, in his view, was not typical of traumatic axonal injury.

(5) There was discussion of the relevance of axonal injury in nerve roots. Dr Smith notes that this aspect raised concern in relation to the traumatic episode, although recognised the need for caution particularly in the light of recent publications. Dr Ramsay considered such findings as generally unreliable indicators of spinal injury in the absence of other findings of spinal trauma. He also thought there were other abnormal findings in the spinal cord leading to the possibility of a more pervasive metabolic or other CNS process.

120. In his oral evidence in this hearing Dr Smith was asked about the bleeding he saw and said that due to the appearance of the clotting he estimated it was within 72 hours of death. When asked where the blood came from he said it had to come from the vascular system. He said there were three sources: (1) the commonest was from damage to the surface of the brain, bruising causing the vessels to bleed. Not so common in children; (2) damaged bridging veins which allow blood into the subdura; (3) hypoxic ischemic injury to the brain, but the evidence base for this is limited. The rationale is that the hypoxic ischemic injury causes intra dural bleeding to extend to the sub dural space. His concern is that these observations have not been proven yet, no-one else has seen these observations and he considered it a *'great leap'* to say hypoxic ischaemia can lead to subdural bleeding.

121. Mr Love asked him about the differential diagnosis of other experts. He said he did not consider infection was likely although he did observe inflammation in one of the sections, but considered that to be more likely related to hypoxic ischemic injury rather than infection (such as meningitis). In relation to calcium levels he said he did not see how this could cause any significant changes to the brain. In relation to seizures/fitting whilst he accepted that could be damaging to the brain he had not seen seizure related brain injury as diffuse as this. Turning to the two aspects that concerned him and made him consider trauma being a possible cause he said that he had only seen axonal spheroids within the pyramids in one other setting other than children who had been victims of abusive head trauma, where adults land on their head on the ground and the head flexes backwards or forwards. In relation to the lumbosacral nerve root changes this is at the bottom of the back and such damage is not normally associated with ischemic hypoxic injury. In relation to the head injury he said on timing alone it could be related to birth injury but the pathological distribution was not consistent with that time frame. The existence of Vitamin D deficiency/rickets may account for the fractures but the bleeding and the damage to the spinal cord or brain stem is, in his view, likely to be directly related to trauma.

122. In cross examination he said the delay in the post mortem did not adversely effect his examination. He agreed in reaching his conclusions it involved a subjective element as it brings in his professional experience. He accepted it was important when reaching a conclusion to look at the whole picture, both in life and death. His view was that trauma played a role but was not and could not suggest whether accidental or non accidental, as he said pathology can't go backwards. Although not within his expertise he agreed with the description given that if it was trauma the child would collapse at the time of injury and would be seen as requiring medical attention. He agreed with Dr Ramsey that in such a situation you do not see a child apparently decline, recover and then collapse. When asked about the thin film SDH

he said it was a common amount save in birth related cases, when there would be more. He was asked about Dr Scheimberg's description of lifting the dura to see the bridging veins to see if they are intact. He agreed that is what she reports but commented she would only be able to comment on the veins she could see. In relation to infection he would defer to the paediatricians, he considered it unlikely. In relation to the axonal swellings he agreed they raised the possibility of trauma but agreed the whole clinical picture has become more complex and it was an important component. He agreed, in re-examination by Mr Love that this is a very difficult case because elements of clinical history do not equal the pathology; on pure pathology trauma appeared to play a part but that does not match the clinical picture.

Dr David Ramsay

123. Dr Ramsay provided a report and gave evidence; he was called on behalf of the father. He qualified in this country and is based in Canada. He is the equivalent of a Consultant Neuropathologist at the London Health Services Centre and Professor of Pathology at the University of Western Ontario. He qualified as a Neuropathologist in 1990 and since then has conducted about 4,000 autopsies, he estimates about 10 – 15% were in relation to infants. He said over the last five years he has conducted between 22 – 24 coroners' autopsies each year in children under the age of 5. He is responsible for all the neuropathological and optical investigations for suspicious infant deaths in London, Ontario and many similar ones in Toronto. He has been a medico-legal expert since 1992 and over 80% of his reports are prepared for the prosecution.

124. He saw the same glass microscopic slides as Dr Smith. He said he largely agrees with both the macroscopic and microscopic findings of Dr Smith. However, there are a number of areas where they differ on findings and emphasis and the conclusions that can be drawn from them. The main areas can be summarised as follows:

(1) In relation to the leptomeningeal inflammation Dr Ramsay considered, in his experience, that it was unusual to find this degree of inflammation purely in association with hypoxic ischemic brain injury. He said the appearance indicates that something is stimulating or irritating that part of the body and specifically with the pattern like this, an infection by bacteria. Dr Smith considers this is secondary to the brain injury which Dr Ramsay does not disagree with but his experience puts an infection higher than Dr Smith but did acknowledge *'At the end of the day, the possibility of infection, based on the clinical evidence, would seem to be unlikely'*.

(2) Definite hypoxic-ischemic neuronal change is not visible in the spinal cord. Dr Ramsay said the findings of the hypoxic-ischemic neorons is fairly non-specific and there are a number of explanations for why they should become red. He concludes it is caused by the *'bombardment of the nerve cells by the increased electrical activity in epilepsy can itself damage the neurons in this way'*

(3) In relation to axonal injury he said the ability to identify abnormal axons has been greatly improved by the use of a specific technique referred to as beta-amyloid precursor protein immunohistochemistry, (usefully shortened to

BAPP) which identifies where the flow of protein up and down the axons has been compromised or slowed down by some process. That process can be trauma, hypoxic ischemic injury and/or brain swelling. Dr Ramsay has sought to separate the two patterns of axonal injury; the classic form (the form recognised in unconscious patients for decades) and the more recent form, called the pleomorphic form, that reveals not only in trauma, but in other situations of brain injury unrelated to trauma, much more widespread but subtle damage to the axons. He said Jayden had both patterns, the pleomorphic form which did not surprise him due to the extent of the brain injury and swelling. The classic axonal swellings were restricted to the brain stem. He said save for one site they have an odd appearance which suggests they may have been around for some time, or there could be some other process that hasn't been recognised in the brain to account for them. In one site he said there were classic axonal swellings in one of the white matter tracks low down in the brain stem. He said the absence of siderosis and astrocytes, which he would expect to see if there was traumatic axonal injury, means that whilst trauma can't be excluded as a possibility these features are not seen. Dr Smith in his written and oral evidence was cautious about the significance of these as supporting a traumatic cause, he said in his statement and his evidence that they are an aspect that concern him as to a '*possible'* traumatic aetiology.

(4) The concern about the significance of the axonal swellings to support trauma is supported by the view that his experience as a neuropathologist (which is supported by the experience of the clinicians) is that one would expect that after an injury is inflicted on a child that causes the triad and which causes axonal swellings localised to the brain stem is that the child is immediately and permanently unconscious. He said the clinical history does not fit with his experience of inflicted head injury in infants. He accepted that there is a debate in the literature about this *'but it's not very convincing yet'*.

(5) In relation to the significance of the axonal damage in the lumbosacral part of the spinal cord he said the general idea is that when a child is violently treated that the rapid flexion, twisting and extension of the vertebral column stretches these nerve roots, causes the axons to be injured and therefore allows one to identify the sites of axonal injury. But he cautions against reliance on this on two grounds: Firstly, you need to know what the normal state of affairs in the infant's spinal cord is; it may be a normal finding, it has not been adequately explored in the medical literature. Secondly, you need direct evidence of trauma (e.g. fractures to the vertebrae, bruises to the tissue around the vertebral column, tearing of muscles), which were not present. The red cells in the nerve roots are common non-specific findings with an absence of an inflammatory reaction. Again Dr Smith is cautious about this aspect too in his written and oral evidence, referring to it as an aspect that concerns him as to a *'possible'* traumatic aetiology.

(6) He expresses general caution about using cerebello changes to date injuries. He said whilst there are some features to suggest there are older injuries they could also be the result of acute events over the last three days of Jayden's life.

(7) In relation to the cause of the SDH being other than traumatic in cause Dr Ramsay said, referring to the Cohen/Scheimberg paper, that possibility still remains theoretical. His own view was that it is an evolving part of the medical literature, that there were good grounds to believe that under certain circumstances in severe hypoxic-ischemic injury such haemorrhages can take

place, but that the acceptance of that as a mechanism is not wide. Dr Smith in his evidence said it was a *'great leap'*.

125. Dr Ramsay summarised his position in his oral evidence as follows. He said doctors function effectively by recognising patterns and if in recognising a pattern there are a number of unusual features that don't quite fit with the pattern there is a complete red flag to look at your diagnosis. In this case he regarded the main odd features making this not a straightforward case of seizures, trauma or hypoxic-ischemic encephalopathy; in an unusual and complex case like this he said ready answers may not be available. The unusual features include the vitamin D deficiency; multiple fractures most of which may be related to weakness of the bone; clinical presentation of the child with waxing and waning seizure state and then rapid deterioration; the presence of inflammation of the meninges; the odd axonal swelling in the brain stem. He concluded *'Taking these findings together though they certainly warn me that this is a complex case in which a single explanation, any of the single explanations, cannot hold water.'*

Opthalmologists

Professor Luthert and Dr Bonshek

126. The court had the benefit of having the expert evidence of Professor Luthert and Dr Bonshek, two of the leading ophthalmic pathologists in the country. They, together with John McCarthy, gave a joint presentation to the meeting convened in December 2009 by the Royal College of Pathologists.

127. In this case they had both seen the details of the examination conducted by Dr Nischal on the afternoon of 23.7.09 and the RetCam images taken just before that examination. In terms of their respective examinations there was little dispute between them. Any difference between them was limited to their interpretation of their findings in the context of their independent experience and (as somewhat graphically described by Professor Luthert) their *'interpretation of what is, bluntly, a rather inadequate or incomplete knowledge base of what goes on in these cases'*.

128. The observations from their examination can be summarised as follows:

(1) Both described the eyes as being somewhat autolytic caused by the delay in the removal of tissue for fixation, the degenerative process causes the tissue to fragment and it can make it somewhat harder to interpret change.
(2) They confirmed the presence in both eyes of scattered retinal haemorrhaging that went as far as the equator and beyond, into the ora serrata. Professor Luthert said he *'found it difficult to be completely confident of precisely what was where, because things had moved around because of the disintegration'* although the RetCam images clearly show that the haemorrhages are going beyond the equator and neither disputed the findings made by Dr Nischal.
(3) They agreed there was optic nerve sheath haemorrhage.
(4) The existence of papilloedema; this is a swelling of the optic disc (where the optic nerve comes into the back of the eye). Professor Luthert said its existence suggested to him that there has been *'significant and reasonably sustained increase in intracranial pressure'*. There was an issue between the

experts as to timing. There was no evidence of papilloedema in the RetCam images. Professor Luthert said in his view it takes time for papilloedma to develop so its absence would not exclude the existence of increased intracranial pressure, he said *'I do not find the absence of evidence of papilloedema when the RetCam images were taken as an indication that the retinal haemorrhages have not resulted from a mechanism, if you like, therefore secondary to raised intracranial pressure.'*. Dr Bonshek agreed it was evident post mortem but said *'..it is mild, and the pattern of bleeding seen pre-mortem at Dr Nischal's examination, and also even at post mortem actually, is not the pattern of bleeding one associates with papilloedema, at least not all of it.'*

129. They both agreed that retinal haemorrhages could be caused by raised intracranial pressure but disagreed as to the extent and type. Professor Luthert said that the clinical picture would be '*entirely consistent with the presence of high, and sustained high, intracranial pressure, which is what I believe would be required to generate the papilloedema that I saw.'* He continued '*..it supports, I suppose, with a greater degree of clarity that would otherwise have been possible, the fact that the intracranial pressure appears to have been elevated before we had the first evidence of retinal haemorrhaging.'* In his evidence Dr Bonshek said *'...I believe that in some cases very, very sudden and very, very severe increases in intracranial pressure, and this is basically over a period of minutes or less than a minute, rather than hours to days, and very, very high spikes of pressure, I believe that situation may lead to retinal haemorrhages. But raised pressure in the situation where one gets a gradual rise over a period of hours to days, I do not believe produces that pattern. If it does cause retinal haemorrhage, the pattern it produces is that seen with papilloedema, and at the time of Mr Nischal's examination of these eyes, there was no papilloedema detected by him, and the pattern of retinal haemorrhaging was not that of what one associates with papilloedema in any case. So I think my area of disagreement with Professor Luthert is on his statement where he says he believes that the retinal haemorrhages are best considered secondary as to any cause....raised intracranial pressure of any cause. It is the 'of any cause' that I disagree with.'* He said when he went through the history he did not see any information which indicated there had been a sharp rise.

130. However, when Dr Bonshek was taken through the detailed clinical history (in particular when Dr Runnacles and Nurse Mogridge became involved and the clinical observations and medication administered by the retrieval team) he agreed that it showed Jayden was showing very pronounced signs of a very steep rise in intracranial pressure and seizure activity.

131. One of the points made by Dr Bonshek is that the pattern of the retinal haemorrhages seen here is not that that you would expect for raised intracranial pressure. Professor Luthert said *'The classical distribution of haemorrhages following raised intracranial pressure is for the haemorrhages to be quite close to the optic nerve head and to not have the distribution that we see here where they extend a long way forward in the retina. My first concern in drawing, if you like, an absolute conclusion around that is, firstly, a lot of our perspective of the pattern of retinal haemorrhaging in raised intracranial pressure comes from older children and adults, and there is reason to believe that the infant retina can behave in a*

rather different way. I also believe there are challenges around context and so the very particular context in which we see increased intracranial pressure in these kinds of cases is not of, for instance, a rapidly growing brain tumour or as a result of a severe infection within the brain, but it is in the context of a child who has got a lot of other problems as well. For those reasons, I think that, if you like, the classical pattern of retinal haemorrhaging associated with intracranial pressure is not necessarily informative in this context. Elaborating and pursuing from the opposite direction, I think there is, for myself and colleagues in general, a view that, conversely, a more distributed pattern of haemorrhages is not diagnostic or specific for trauma. So looking at it both ways, I think it is appropriate to approach interpretation of causality from pattern of haemorrhage with caution.' He said he had personal experience of a case of a child of a similar age as Jayden with pneumococcal meningitis who had extensive haemorrhages from the optic nerve all the way to the ora serrata, were much more severe than in this case, and there was no suggestion of trauma. He pointed to the fact that it is known that about one third of newborns have retinal haemorrhages, but it is not known what the mechanism and formation of those are. Professor Luthert said it suggests to him that there is a *'certain ease'* with which retinal haemorrhages can occur in this age group and the younger the child (especially 6 months and under) the more extensive the haemorrhages are. For Professor Luthert this increases his concern about extrapolating evidence from older children and adults to a young child.

132. Dr Bonshek maintained his position that the pattern of bleeding was more likely to be caused by trauma (inflicted or otherwise) he said they were *'..more likely to fit into the pattern of bleeding that one sees in non-accidental injury, or severe accidental injury, or other medical conditions…'* but did accept that retinal haemorrhages could be caused by the rapid increase or spikes of intracranial pressure.

133. They both agreed that the research evidence demonstrated that seizures can be a cause of retinal haemorrhaging but that it is rare and there is no evidence where retinal haemorrhages to the extent as seen in Jayden's eyes are caused solely by seizures alone. This view is formed on a very limited research evidence base relative to Jayden's age and the type and duration of seizures experienced by Jayden. Professor Luthert suggested that if there was a link between seizure activity and retinal haemorrhages it was through the mechanism of brain swelling, not through something completely independent of brain swelling.

134. They both agreed that the increase in carbon dioxide levels in Jayden could have an impact. It would cause the blood vessels to dilate and they may become more fragile due to the stretching and may be more susceptible to bursting. This factor could contribute to the existence of the retinal haemorrhages.

Histopathologist

Professor Malcolm

135. Professor Malcolm was asked by Dr Scheimberg to look at the bone samples taken at the post mortem. He is a Consultant Histopathologist at the Shrewsbury and Telford NHS Trust and is honorary Professor of Pathology at the medical school in

Keele. He has been a consultant pathologist for over 30 years and performed about 4,000 autopsies. He described a histopathologist in his evidence at the CCC as a specialist who looks at human tissue down a microscope to diagnose what is wrong, either with a live patient or with tissue from a dead patient. He has a particular expertise in the histopathology relating to bones.

136. There is no issue in this case about the existence of the fractures; they are listed in the agreed facts. In his evidence Professor Malcolm divides them into two categories, one relates to timing and the other relates to likely cause.

137. Dealing with timing first. His evidence is that the existence of fractures does not alter the initial healing process, as calcium is not required. So the time frame he gives for the fractures (other than in the right tibia greenstick fracture and right humerus) range between 5 to 14 days. He took those time frames from the stage the healing process had reached. In relation to the healed greenstick fracture in the right tibia and the healed fracture in the right humerus he said they were at least six weeks old, but accepted they could be older, up to 10 weeks.

138. Turning to likely cause he considered that a number of the fractures could be attributable to rickets and possibly rough handling. These principally related to the fractures of the hand and foot. He said that having been weakened by rickets these fractures could be caused by mechanisms like trying to put the foot into a babygrow or something like that. He also considered the incomplete fracture of the right tibia could be caused in the same way; he thought it was a possibility. In relation to the three metaphyseal fractures, the fractures to the right humerus, the recent fracture to the right tibia and the skull these could not be explained, in his opinion, by the presence of rickets.

139. He agreed a number of matters:

(1) The effect of the vitamin D deficiency goes beyond simply the impact on the bones.
(2) Calcium is controlled by the endocrine system and is borrowed from the bones to support the rest of the body.
(3) When he wrote his textbook in 2001 rickets was rare in developed countries.
(4) In the last few years there has been a significant increase in rickets, both congenital rickets and potentially nutritional rickets.
(5) Congenital rickets would appear to be the most severe because it starts pre-birth.
(6) There is a difference between low vitamin D or vitamin D deficiency as shown by testing the blood and rickets, which is an alteration of the growth and structure of a bone. One leads to the other but the two are not the same.
(7) Jayden had moderate to severe rickets.
(8) In cases Professor Malcolm has come across this case is as severe as he has seen since the 1970s when rickets were very common in children in Glasgow, where he worked. Since then he has only seen one of the severity seen in Jayden, although gives the caveat that his experience is distorted by the fact that he looks at bones from dead children so it cannot reflect the severity in the population as a whole.

(9) He agreed with other witnesses that expressed surprise at the presence of this type of rickets in a child as young as 4 ½ months. He said there has to be a reason for identifying rickets. As can be seen in this case not all clinicians are attuned to the presence or absence of rickets
(10) There is no objective way of evaluating the strength of the bone, you don't know how much force is required to inflict any damage; it can only be inferred.
(11) He found it very surprising that the mechanisms causing the fractures had not produced any visible signs such as bruising.
(12) He did not see any fractures to the ribs.

140. The main challenges in his evidence in the CCC and during this hearing were focussed on the following areas:

(1) In the CCC a central plank of the Crown's case (and Dr Cary and Dr Peter's evidence) was that the recent haemorrhage around the skull fracture and the fissure were advanced as evidence of recent injury of some kind to the healing fracture site prior to admission to UCLH. In his evidence at this hearing Professor Malcolm confirmed that this haemorrhage took place within 24 hours of death; namely when Jayden was at GOSH. He agreed the relatively fresh haemorrhage implied trauma of some kind to those particular sites which on that timing cannot be related to the parents care.
(2) The evidential foundation for his opinion on timing. He accepted that rickets affected the healing process, although not in the first stage as calcium was not needed. In relation to the timing of the older fractures he accepted that the evidential foundation for his timing (6 or 10 weeks) when looking at Jayden's particular circumstances was not very strong although he maintained his position that none of the fractures could be birth related.
(3) The failure to conduct any tests on the strength of the bone. He accepted the bones may be less robust than in normal infants and that rickets affects all the bones, but he could not see how the healed right humeral fracture and the recent tibia fracture could be down to handling Jayden. He said the tibia is probably the strongest bone in the body and he considered it was difficult to see that a transverse mid shaft fracture of the tibia in a non mobile infant could be down to rough handling and rickets. He said a cortical fracture of the humerus requires significant trauma; he said looking at the cortex of the bone there is sufficient bone there that he thought it would take quite a degree of force to actually cause it to fracture completely. His rationale for that view was that he has examined the histological sections; he looks at the bone thickness, both in children and in adults. He did not apply that analysis to the skull fracture.
(4) He agreed there were a number of factors that affected bone strength; diameter; cortical thickness; and collagen strength. He said the only difference when you look at the strength of a bone with and one without rickets is the amount of calcium present within that bone. Beyond that one cannot assess the strength of the bone without doing a whole series of mechanical tests which he was not aware of ever having been done.
(5) In relation to the skull fracture he said rickets would have very limited effect on the strength of the skull, if anything it would make it more malleable. He did not agree that the existence of craniotabes would make the skull more

likely to fracture. Firstly, because if you reduced the calcium in the skull you make it more flexible, therefore less brittle. Therefore, in his view, it would require greater force to fracture the skull in a four month year old, but he accepted there was no sign of external injury. Second, he relied on a paper by Teresa Chapman and others in 2010 when they reviewed 47 children in Seattle with proven rickets and found no skull fractures. He said this was one of the few papers in the world that has looked at this. They were all under the age of 2 years, there were 6 under 6 months and there were no fractures noted in any non-mobile child. However he accepted that he did not know whether the study differentiated between congenital or acquired rickets, as he agreed that would make a difference to the severity of rickets. He thought they would all have to be moderate to be diagnosable on an x-ray. Also the study did not make clear what the difference in time was between diagnosis and taking part in the study as the parents or carers may have behaved very differently if they knew their child had rickets. Finally, it is not clear what images were done on the children, whether there were full skeletal surveys.

(6) The metaphyseal fractures are not, in his opinion, affected by rickets as calcium is not present in the growth plate. He said they are caused by a twisting or rotational mechanism. In relation to their existence he said much of the literature was based on radiological evidence rather than pathological evidence. He said on several occasions he had been asked to look at a metaphyseal fracture that appeared on an x-ray which, in fact, wasn't a metaphyseal fracture. Again relying on the Chapman study he said none of the children in that study suffered from classic metaphyseal fractures. He was asked about a 2008 study by Russell Chesney in 2008 which stated that *'Patients with metaphyseal lesions and/or rib fractures are sometimes said to have 'pathognomonic findings' of either rickets or abuse. Fractures at these locations are found in a wide variety of inherited and metabolic disorders as well as in cases of trauma',* he said that was written in 2008 and prior to the Chapman paper he would have accepted that as it was the best they had, but he doesn't accept it now because of the Chapman paper.

(7) He did not accept Dr Cohen's view regarding the existence of the traumatised fissure. He said she did not mention or see the granulation which he said is the crucial diagnostic feature, it is the healing tissue which is what he sees in the fissure and why he believes it is traumatised. He said it is not particularly obvious and he may have seen it because of his expertise.

(8) He was not able to say whether the massage conducted by the parents on Jayden could be responsible for any of these fractures.

(9) He said he could not gainsay the paediatric evidence that such fractures may not cause pain and so may not be detected by the carer. He said he found it surprising that there were no bruises relating to mechanism by which the fractures occurred but did not profess expertise in this aspect.

Dr Cohen

141. Dr Cohen is a Consultant Histopathologist based at Sheffield Children's Hospital. She has been a Consultant for about 20 years specialising in paediatric neonatal and perinatal histopathology. Her department conducts about 320 perinatal and paediatric post mortems each year, of which Dr Cohen carries out about 100. She said about half of her work is clinical and the other half post mortems. Her clinical

work involves under taking biopsies for all soft tissues, including bones. She said she has dealt with a few cases that involved vitamin D deficiency but none as severe as in this case. She also lectures in pathology at the University of Sheffield and had published a number of research papers.

142. She confirmed her view that the cause of Jayden's death was global hypoxic ischemic encephalopathy. In her written reports (which were in places difficult to follow because of their confusing format) she was unable to give a cause for this and considered there were a number of possibilities, including trauma and natural causes. She was unable to say which of them was the primary cause and there may have been a number of them together that lead to his collapse.

143. She was asked about the internal bruising to the skull, she said that was in keeping with some trauma although she would have expected to see bruising on the skin. She said no swelling was noted. She considered other causes such as hypocalcaemia and seizures triggered by an infection. She did not suggest Jayden had an overwhelming infection but it could have contributed to the whole picture and been a cause for the seizures.

144. In relation to the subdural haemorrhages she said her view was that there can be several sources of the bleeding. She said she noted fresh bleeding in the dura, within this membrane covering the brain. She said that could be the source. The quantity of blood supported that as the other source could be rupture of the bridging vein, if it was from there she would have expected a larger amount of blood rather than a thin film. She said she had prepared a paper with Dr Scheimberg on this subject. She said it was an observational study. The dura is a fibrous membrane that contains blood vessels and nerves. In the study she did with Dr Scheimberg they looked at 25 foetuese and 30 neonates, the age of the neonates was no more than 19 days old. She said they chose a population all had some degree of intradural bleeding and in that population they found an association between subdural haematoma and hypoxic-ischemic encephalopathy. She said she has also subsequently conducted a study with Dr Sprigg and Dr Whitby in 2010 where they did MRI comparisons and found 13 cases with subdural haemorrhage; in the 13 cases 12 had features of hypoxia and all had intradural haemorrhage. She said that further supports the association in children older than the ones in their earlier study. She accepts it's unusual but says it can happen.

145. Dr Cohen supported the procedure adopted by Dr Scheimberg during the post mortem in seeking to establish whether the bridging veins are intact, she did not regard it as a futile exercise. She said if you are dealing with a subdural haemorrhage that is a large amount of bleeding she agrees it would be very difficult to see the bridging veins, but that is not the case with a thin film subdural as in this case. Then, in her view, it is possible to see if there is a torn bridging vein, although she agreed it is not possible to explore them all. She accepted that the source of subdural bleeding could equally be (according to her previous studies) haemorrhage exceeding the boundaries of the dura or ruptured veins that could not be seen. She prefers the source of blood coming out of the boundaries of the dura as that is what she has observed. The difficulty is that despite this having been her view (together with Dr Scheimberg) for some time it has little support, either by way of direct

observation or further study, from others who specialise in this field. As one witness put it they are seeing what others don't.

146. In relation to the traumatised fissure there is an issue between Dr Cohen and Professor Malcolm about the presence of granulation tissue. Dr Cohen said she didn't see it. She said when you look at granulation tissue it is defined by the presence of very young blood vessels. They are very delicate blood vessels and they are seen as little holes surrounded by endothelial cells because they are very young. In her view Professor Malcolm mis-interpreted these as granulation tissue. She relies on a paper by Mack, Squier and Eastman where they describe the presence of these holes they call channels and did a stain to demonstrate that these are not blood vessels. She accepts if they were blood vessels there would be granulation tissue and that would mean this is an old lesion, more than three or four days old. To follow through her argument she said if this is not granulation tissue and this is just fresh intradural blood then it can be the blood because of the hypoxia into the dura which can go through to the subdural space. She said there was no haemosiderin reported, this is iron from the red cells that is processed by the macrophages, which can be identified by a stain. She said if there was granulation tissue you would expect to find haemosiderin. This aspect was not explored with Professor Malcolm other than in very general terms. However, Professor Malcolm accepted the recent bleeding seen in this area was less than 24 hours old. Dr Cohen did not take issue with Professor Malcolm's view that none of the fractures can be dated back to Jayden's birth.

147. She was asked about the sub scalp bruising, she considered it could be related to the skull fracture and you need a degree of trauma to cause the bruise. But she noted from the external post mortem he didn't have any swelling or external bruise. If you have an impact the force comes from outside to inside. In this case the lesion to the skin was missing. It appears to come from the inside out, from the fracture out. It could be because of the lack of external bruising the force would not have been so big. In terms of timing she thought the bruise was 72 hours plus.

Paediatric Neurologist

Dr Jansen

148. Dr Jansen was instructed on behalf of the father. She is a paediatric neurologist. She qualified in 1999 and has been specialising in paediatric neurology since 2006. She is based at the University Hospital in Brussels. Her fields of expertise are early child development, epilepsy, neurogenetics and learning disorders. She has done research where she focuses on brain development and the genetics of epilepsies and she also teaches in the field of child health. Her day to day practice consists of outpatient clinical work (40 to 50 patients a week, covering a wide spectrum including acute neurological problems such as seizures) and she also sees patients on the intensive care unit, on neonatology and on the wards.

149. In her written report and evidence at the CCC and in this court she examined the history of presentation of Jayden whilst in his parents care and also at the GP and UCLH, the medication he was given and his response. She concluded that from her examination of that background it was clear that Jayden was having tonic/clonic

seizures, probably intermittent seizures. She acknowledged it may be difficult to tell the difference in a child of Jayden's age between a tonic seizure and tetany. She also acknowledged that in such a young child the clinical expression of seizures can be very subtle. She said the grunting noises described can also be seizures. She considered Jayden had prolonged seizures (status epilepticus) and the fact that he was insufficiently responsive to anti-epileptic treatment means that it was refractory status epilepticus. The effect of ongoing seizures, especially in a young baby, has a higher risk of mortality and a poor prognosis. She said the consequences are that there is a higher metabolic demand on the brain, and prolonged seizures are also associated with inadequate blood flow and decreased oxygen supply to the brain, which contributed significantly to the hypoxic ischemic injury to the brain. She said the description of the decerebrate posturing became clear a bit later after he was admitted to hospital. At 2.03 he was described as being very rigid, the arms are completely outstretched and twisted outwards, and he is described as flat and unresponsive and not moving any more at all; when he was admitted he was still more active. She said the change in the GCS is a reflection of the fact that there is increasing injury to the brain and it might be an expression of rising intracranial pressure. She said even taking into account the concerns expressed about the use of GCS she considers the difference between 13 and 6 really reflects the clinical deterioration.

150. She said at 14.03 the drugs that were administered to Jayden would paralyse his muscles so they are no longer going to contract but it does not stop the underlying firing in the brain. If you want to treat that it should be treated with anti-epileptic drugs. On admission he was given two doses of lorazepam (an anti-epileptic drug), he did not respond and was loaded with phenytoin, another anti seizure drug. But then, after that there were no additional anti-fitting drugs until much later in the afternoon (around the time of the transfer to GOSH when benzodiazepines and phenobarbitone are administered) and there is no record of the monitoring of his fitting or adjustment in his treatment. As a consequence it was her view that it could not be excluded that fitting carried on during the afternoon, undetected due to the other drugs given to Jayden. She said *'..it's very difficult to formally assess because of the fact he was paralysed and the fact there is no EEG. And it is clear from the fact that once he was fitting later on in the afternoon…when the retrieval team arrived, one, and both also because his pupils dilated at that time, I think these two factors are signs of the fact that his condition is still worsening at that point.'* Later she said *'..after that [the anti fitting treatment on admission], it was most likely there were ongoing seizures, also the fact that afterwards there are signs of more intracranial pressure and breakthrough seizures. I think it very likely that there have been seizures also during these four hours.'* Dr Peters said this would be monitored though blood pressure and temperature, however it is of note that there was raised blood pressure at about 17.45 reported to Dr Runnacles which she advised was treated by way of a saline bolus.

151. In relation to Jayden's low calcium levels she said that although there is no frank hypocalcaemia considering Jayden had a Vitamin D deficiency it is known that his calcium metabolism is altered; it would make him more prone to seizures or more refractory to the treatment of the seizures and it could be linked to tetany. His Vitamin D was measured at 18 nmol per litre which is very low (deficiency is less than 30 nmol/L) and this would have impacted on the way his body reacted to

stresses; it gave him a latent vulnerability and it had an impact on his immunity system. The very low Vitamin D level affected his whole metabolism.

152. She said the other contributing causes for the seizures were (i) febrile seizures caused by his flu like symptoms, she recognised he did not have a temperature but said the administration of calpol may have affected that and she is not suggesting that it is a typical febrile seizure. She relies on the CRP levels and white blood cell count (with a high neutrophil count) being raised, which are objective indicators of a degree of infection. Later blood tests may be affected by the medication administered on admission and the tests cannot cover all agents; (ii) meningitis, although she recognised that his clinical presentation was not typical for this but only a lumbar puncture would rule this out (it was agreed by all that this was not an appropriate procedure due to Jayden's condition). The impact of (correctly) immediately on admission putting Jayden on antibiotic and anti viral medication could impact on the subsequent investigations for infection; (iii) trauma although the fractures were dated older than five days and in those circumstances she considered it very unlikely that they were a direct cause of the seizures with which he presented. If it had been trauma she considered he would have presented much closer in time to the fracture; it is likely he would have been in a state of immediate collapse.

153. Dr Jansen considered there could be more than one trigger for the seizures and the fact they went on for a long time and were refractory to treatment would give rise to or be an aggravating factor for hypoxic ischemic injury and would be consistent with what was seen on the imaging on 22.7.09. She confirmed this was in her clinical experience and in the medical literature.

Endocrinologist

Professor Nussey

154. Professor Nussey is a Professor of Endocrinology at St George's Hospital Medical School. He was called by the mother. He has been involved in this field since 1971 and was appointed senior lecturer and consultant in 1987. He has held the posts of clinical and medical director and set up a specialist endocrine unit. His work covers a lot of specialities as just about every tissue in the body either makes or responds to endocrine signals. He has published extensively on the subject. He has worked extensively with his paediatric colleagues and had experience of rickets many years ago but, like the other experts, limited direct or indirect recent experience.

155. He described the significance of calcium and Vitamin D. Calcium is an ion (small charged particle) and is involved in a very large number of processes within the body and as a consequence is very tightly controlled in the body. A measure of the importance of something in the body is the tightness with which it's controlled. It's controlled by two hormones, one of which is called parathyroid hormone, which is made by glands in the neck, they have an effect on activating vitamin D, and also mainly have effects on the bone and the kidney. Vitamin D gets its name because it was thought to be necessary in the diet in small quantities, that's why it's called a vitamin. He said it is best regarded as a prohormone. That is something which gets

converted to an active hormone and the active hormone had some minor chemical changes done to it which make it active. Vitamin D has effects on, for example, the gut absorption of calcium. They regulate the wastage of calcium out of the body. Low concentration of blood calcium is called hypocalcaemia. It can be asymptomatic or it can be severe if there is a sudden lowering of calcium or very marked low calcium. Severe symptoms include seizures or spasms of the muscle, as many of the body cells are dependent on calcium for doing things. A deficiency of vitamin D in adults tends to cause mild hypocalcaemia and weakness of muscle and softening of the bones. In children where the bones are still growing it classically causes the condition called rickets, this gets its name from an old English word rick, which means twist. It also has other effects; it affects 3% of all the genes in the body. As a consequence the picture of the disease can be very variable; if you have 750 genes which are potentially affected by vitamin D, and each of those response elements within that gene are affected by 10 or 15 other factors, called transcription factors, these result in a very variable phenotype.

156. He described some of the papers being used as being used *'a bit like academic grenades and thrown into the argument to justify points rather than informing the discussion'*. He said it is very difficult to make generalisations when you are dealing with something at one extreme of the spectrum. He also referred to the limited evidence that was available as to the effect of vitamin D deficiency on the brain. He said there were some studies that showed vitamin D has anti epileptic fits in animal models. He was not aware of any studies on humans.

157. He said it is known that certain populations are at risk of vitamin D deficiency. That includes breastfed babies who are entirely breastfed and particular ethnic groups. Although vitamin D was originally named because it comes from the diet it is actually made much more in the skin and so pigmented skin populations have low vitamin D. The combination in this case with the radiological presentation, mild hypocalcaemia, low phosphate with the alkaline phosphatase measurement (that measurement comes from the cells in the bone associated with fractures) and vitamin D deficiency were in Professor Nussey's view, so classical that there didn't need to be any further tests; Jayden was a *'high risk baby'*. His view was had further tests been done the parathyroid hormone would have been high because the phosphate was low.

158. Turning to studies on the impact on a baby's body of vitamin D deficiency to withstand, for example, infection are limited. There have been limited double blind randomised control trials, most studies look for associations which tend not be as robust as the double blind trials. He agreed, on the information that is available, with Dr Jansen's view that vitamin D deficiency would make a child's body more liable to stresses and that it inhibits the child's ability to fight infection.

159. In relation to the calcium levels recorded for Jayden his opinion was that it is important not to apply the ranges rigidly, the person being studied might not be normal from the population that provided the information to found the ranges. In addition the clinical context is important too.

160. His opinion was that hypocalcaemia alone could not have caused Jayden's condition when he arrived at UCLH. He said it was highly likely, because Jayden

was born in March he was born vitamin D deficient and the calcium was likely to have been low for a very long time and because it was only mildly low, he did not think the calcium level on its own would have caused any problems. He said if there's another factor that causes seizures, then a low calcium level would exacerbate that. The statistics on infants is that at least 10% of infants will have some seizure; infection is one factor that increases the chance of that. This could have been a trigger, together with slightly low calcium to generate the fits. He observed that when doing samples for infection the fact that you don't find the infection doesn't mean to say it's not there, because there are all sorts of reasons why it's not in the sample you have taken. In addition, the results can be masked by administration of antibiotic and anti viral drugs. He does not rule out meningitis as no lumbar puncture was carried out. He said there was other evidence of possible infection in Jayden; a raised white blood cell count of 20 a large proportion of which (three quarters) were neutrophils (which is a cell that attacks bacteria). The normal range is 6 – 18; he said the significance was the high proportion of neutrophils. Plus the marker called C-Reactive protein ('CRP'), which responds to all sorts of stimuli (infection would be one) was also elevated (normal 0.25, result 9.9). These are indicators of potential infection. The infection, per se, would be the trigger and then the borderline calcium would be an additional factor. He took into account the clinical presentation that there was no raised temperature but said the absence of fever does not exclude this. He does not suggest an overwhelming infection was required to set off this process.

161. He said fits are caused by the constant firing of the brain cells, this uses up the nutrients of the brain and the brain is not very good at storing nutrients. It has a constant requirement for things like glucose and if the firing goes on and on it exhausts the cells and the cells die. So constant seizure activity essentially kills neurons. He was asked whether that could have been the cause of the hypoxic ischemic injury that was seen in the brain and he said it would be a fairly *'high contender'* for that and fairly likely to have done it as the seizure activity had been going on for many hours. He said infarction is a term that's used when the blood supply (or blood oxygen) is reduced and the tissue dies, so it's infarcted. It can be venous infarction, where the pressure in the drainage sample is elevated so it stops the blood going in or it can be a cutting off of the supply on the arterial (the supplying side) which is more common. He was aware of the consultant paediatric neurologists description, following the MRI scan at 16.45, of Jayden having 'extensive infarction'. His opinion was that there was evidence of the prime problem being the seizures that had gone on for many hours, which were not properly treated.

162. This was contributed to, in his view, by the changing CO2 levels. Professor Nussey relied on an article called 'Hypocapnia and the injured brain: more harm than benefit' (CritCareMed 2010 Vol 38 No 5 p 1348). He said that demonstrated the detrimental effects of doing this as it may aggravate secondary ischemic injury by diverting blood from the injured brain, even if it had been done to seek to reduce intracranial pressure by reducing the blood flow to the brain. Dr Peters response to that is the pH levels were satisfactory (Dr Peters said the article confirms this where it states *'the effects of CO2 are primarily on the cerebral arteries, are pH-mediated rather than CO2-mediated'*)**.** The difficulty with this aspect of the evidence is that what is being sought to be done is re-evaluate the situation on limited information.

The conclusion of the article relied on by Professor Nussey states *'...hypocapnia can certainly cause or worsen cerebral ischaemia, worsen outcome, and cause (direct or indirect) injury to other organs. The decision to institute hypocapnia for therapeutic purposes in the setting of acute brain injury should be undertaken only after careful consideration of the risks and benefits….it might be best limited to the emergency treatment of ICP or to acutely reduced brain bulk in the operating room…..in these settings, normocapnia should be re-instituted as soon as is feasible.'*

163. There was an issue between Professor Nussey and Dr Peters about the effect of the oxygen/CO2 readings whilst Jayden was at UCHL. This issue is not assisted by the lack of any report of information from an anaesthetist. According to the clinical records there is an oxygen recording of 100%. Dr Peters considers these recording relate to pulse oxymeter readings (which usually are a device fixed to the finger measuring the oxygen in the haemoglobin as it circulates). Professor Nussey said if they are from pulse oxymeter readings it tells you the index finger is well perfused; it doesn't tell you anything more than that. He concluded the readings support his view that reducing the CO2 has reduced the vascular supply (vasoconstriction which can result in the brain being starved of oxygen and cause further cell death) to tissue beds, including the brain, and that is what has provoked this severe metabolic acidosis. This has a tissue damaging effect, all of the natural processes of getting rid of acid (through kidneys or by breathing out CO2) have been circumvented; Jayden is markedly acidotic which has detrimental effects on a lot of body systems. It is necessary to take into account the anion gap acidosis. Dr Peters states that this evidence demonstrates that Jayden was not hypoxic whilst in hospital and that together with looking at the reduced CO2 levels in conjunction with the pH readings, they support that too. Professor Nussey's opinion is that Dr Peters is only giving half the picture of the impact of the 'Bohr effect'. Bearing in mind the complications regarding Jayden's metabolic condition it would be difficult to rely on the pH level as an accurate monitor of any secondary ischaemic injury and the effect of acidosis.

164. There was also an issue about the conclusions to be drawn from the lactate levels. Professor Nussey said in his view they would not necessarily be supportive of a traumatic brain injury. He agreed if you stopped breathing for some time you would put up your lactate level but that was not recorded anywhere; there was no record of a cessation of breathing. He had noisy breathing but has never been blue. He said if it was being suggested that this took place prior to hospital he said generally if you are apnoeac there isn't usually a re-starting process. He said his explanation, being related to the seizure activity, had a logical consistency about it. He said as follows when dealing with the counter arguments that the lactate levels were related to trauma '*...what you are saying is blunt injury to the head causing, which was parietal..the breathing centres are quite well hidden down the brain stem…my reading of the experts reports was that this was not seen as causative of the fitting or the subsequent events. I'm trying to understand what you're suggesting happened to cause the apnoea, which caused the lactate.'*

165. He considered what was noteworthy about the documented drugs at UCLH was that the clinical records indicate some were drawn (morphine and midazolam) but not given. He said you can't stop the fits by paralysing someone, because then the

muscles just don't contract so you don't twitch. If you don't do something about the brain cells firing, by sedating them then potentially that was ongoing during the afternoon.

166. In relation to the fractures he was asked to consider whether or not vitamin D deficiency delays the healing process. He said the evidence is unequivocal that it does but the problem is deciding how much; he could find no evidence base to help determine by how much. There are some short term avian and rodent studies, he was not aware of anything in humans.

167. On the degree of fragility of the bones he said the calcium level is independent of bone fragility, or strength, because it's a measure of what's in the blood. What matters for bone strength is what's in the bone and the only histological studies that were done were on bone that was decalcified. He said no one conducted any tests of the strength of the bone, in particular calcified bone.

168. He considered if any of the fractures could have been caused by normal handling of Jayden. Professor Nussey said he took into account the following matters:

(i) The absence of any bruising noted, confirmed by the regular contact with various health professionals.

(ii) The variable ages of the fractures would be compatible with bone fragility of a degree that normal processes (such as putting an arm through a babygrow with a fractious infant) might produce the fractures, not because necessarily the force was excessive but because the bones were fragile.

(iii) The only injuries were to bone. Experimental evidence going back many years illustrates the extreme fragility of vitamin D deficient bones. Some of the studies describe the skull of babies who have got severe rickets as being parchment like.

(iv) There is some evidence (from a study conducted in Queensland) that entirely breastfed babies are less likely to be at risk of abuse.

(v) Vitamin D deficiency has an impact on fibroblasts and collagen type 1. It is not just limited to the growth plate and calcification. He considered it could have some effect in weakening the growth plate but was not aware of any study that had looked at that.

(vi) There was no reliable way of determining why some bones may fracture due to rickets and others don't as *'you would have to make some assumptions as to the torsional strength or cross strength of the bone and I don't see – because if you make that assumption then you're making an assumption about the strength and that's a perception.'* He said there are cases in the literature of black American teenagers breaking the main bone of their legs during a seizure caused by the hypocalcaemia of rickets.

169. One of the factors in considering trauma being a cause for Jayden's death was that it was suggested by the Crown to Professor Nussey in his evidence at the CCC that Professor Malcolm found evidence of fresh bleeding at the time of this child's collapse and it was in that context he accepted that could be evidence of recent trauma. However, the evidence has been clarified in this hearing from Professor Malcolm who dates that to 24 hours before death, so whilst Jayden is in hospital.

170. In relation to the current prevalence of vitamin D Professor Nussey referred to the actions taken after the second world war to address this issue. He said more recently there have been recommendations that in certain circumstances supplements of vitamin D should be taken but they have not, in his view, been effectively actioned.

Paediatric Neuroradiologist

Dr Barnes

171. Dr Barnes is a paediatric neuroradiologist, he is the Chief of Paediatric Neuroradiology and co-director of the Paediatric CT and MRI centre at the Lucile Packard Children's Hospital and Professor of Radiology Stanford University Medical Centre. He a co-founder of the Northern California Child Abuse Task Force and member of the Suspected Child Abuse and Neglect team. He has been practising in the field of paediatric neuroradiology and radiology since 1977. His practice over the last 35 years has not only been in paediatric radiology and then in paediatric neuroradiology but also as a child protection professional, participating with child protection teams and child abuse scan teams at a number of children's hospitals. He has been at his current hospital for 12 years.

172. He carried out a review of the CT and MRI scans on behalf of the mother in this case. His written report concluded that the brain imaging findings were most consistent with global hypoxic ischaemia, but he was unable to conclude any specific causation.

173. There is no issue in this case about the existence of the fractures. Having surveyed the various images he found a number of features which he said were consistent with rickets, in particular:

(1) Incomplete ossification in the skull called craniotobes (cranio means skull; tabes means softening of the skull).
(2) The front part of the ribs near the breast bone were abnormally thickened and widened at the growth centres. This is another finding characteristic of rickets, often referred to as 'rachitic rosary'.
(3) The growth centres in the upper part of the humerus at the level of the shoulder on both sides are irregular and fragmented, no bony or other reaction or swelling.
(4) In the lower portion of the baby's right humerus there is a transverse defect in the bone showing no swelling or healing reaction. This is consistent with a fracture or pseudo fracture. A pseudo fracture is a type of defect in the bone which is not considered to be a true fracture; it's a weak area and due to rickets that can cause a defect in the bone that is known as a 'Looser zone.'
(5) In the left humerus there are areas of irregular ossification or bone development in the shaft of the left humerus; a defect in the bone in abnormal areas of development of the bone that are weak.
(6) In the forearms on both sides there is bowing or irregular curving of the two bones in the radius and ulna. The growth centres of the ulna and the radius are very irregular.
(7) Within the hands there are irregularities, in particular the left fourth metacarpal which can be a fracture, pseudo fracture or a looser zone.

(8) Irregularity at the top end of the left femur, the growth centre is irregular.
(9) In the lower leg fibula and tibia in both legs the growth centres at the knee and ankle are irregular where there is incomplete calcium and phosphorous mineral deposition.
(10) The tibia and fibula are wavy and curved.
(11) In the right tibia there is an area of defect that could be a fracture, it has a very unusual appearance for a fracture. There is no swelling; there is little or no reaction to the bone or callous formation on the x-ray.
(12) There are a number of irregularities in the bones in the feet, particularly on the left.

In his evidence in this court Dr Barnes was able to point out many of these features to the court and the parties on the x-rays on his laptop. Some of matters listed above illustrate the point made by Professor Malcolm of the differences between what is seen on the images and what is seen under the microscope. For example the changes noted on the tibia and humerus, at (4) and (11) above are accepted to be fractures.

174. In relation to the brain findings he said there is a differential diagnosis as to the cause. This list includes a lack of oxygen or blood flow to the baby's brain, infection, swelling due to a baby who is having seizures, convulsions or fits, blood disorders, nutritional or metabolic imbalances, trauma (accidental or non-accidental) and there may be a combination of these factors at play. He said there were no features of the MRI scan that were specific to non accidental injury. He considered the lack of any injury to the neck, spinal column within the neck or the spinal cord as seen on the images militated against a shaking type injury. He would expect to see evidence of haemorrhaging or oedema in the muscles or soft tissues of the baby's neck, or injury to the bones of the baby's cervical spine or traumatic injury to the spinal cord in the neck where it is connected to the brain stem and neck. He did not consider the axonal damage recorded by Dr Smith to be relevant to this aspect as it was in the lower part of the spinal cord, away from the neck area.

175. In considering the particular history of Jayden if he did undergo a long period of seizures over many hours prior to the images taken on the afternoon of 22nd July he did consider that could be a cause to what is seen regarding the brain injury pattern. If the skull fracture is between 7 – 14 days old he said he would have expected to see more focal injury in the brain, deep in that fracture, that he could time with the fracture using the CT and MRI, which he couldn't.

176. His view was that there is little evidential base or scientific study for radiologists to be able to time fractures in an infant under six months of age, particularly when there is a bone fragility disorder. In relation to the healing process he said there is not a substantial evidence base to be able to provide any kind of parameters other than a general impression that it takes longer, particularly in babies who continue to be handled which may impact on healing. He made clear this view was given from the perspective of imaging examination. He questions the criteria used by Professor Malcolm to date the fractures when there are no standards for timing fractures in the presence of rickets.

177. Turning to the strength of the bones he agreed that was not possible to predict from x-rays. He said it is known from the scientific literature that rickets not only affects the growth centres but also the shaft of the long bone or shaft of a rib where those can be weak. No one has done the work or the research, either with normal bones in this age group or with abnormal bones to know what the minimum force is that can cause a fracture; such work, he said, was desperately needed in this age group. He was not aware of any literature that would support Professor Malcolm's ability to distinguish between some fractures being caused by rickets/rough handling and some being non-accidental. He refuted too much reliance on the Chapman paper as he said they did not x-ray all the bones. They x-rayed the bones with the fractures on but did not x-ray all the growth plates to see if there were classic metaphyseal lesions present. It is difficult to tell from the paper precisely what their method was. It states it was a *'A Retrospective study of children younger than 24 months was performed. Clinical data and radiographs were reviewed. Radiographs obtained within one month of the diagnosis were evaluated for the presence or absence of osteopenia, presence or absence or fraying-cupping and presence and characterisation of fractures.'* It appears from that only radiographs of the fractures were reviewed. He said the same applied to another study that was put to him in cross examination (Rachitic Changes, Demineralization, and Fracture risk in Healthy Infants and Toddlers with Vitamin D Deficiency Jeanette Perez-Rossello and others 2011), they did not do skeletal surveys, the only images they did was the knees and some of the wrists. They didn't evaluate them for asymptomatic 'fractures' that can often be seen with rickets.

Paediatrics and Obstetrics

Professor Miller

178. Professor Miller is a Professor of Paediatrics and Obstetrics and Biomedical Engineering at the Wright State University Boonshoft School of Medicine, Dayton, Ohio. He is the director of medical genetics at Dayton Children's Hospital. He has held two major academic paediatric positions, first at the University of Rochester from 1980 to 1992 and since 1992 to date at his current location. His major clinical research interest is in paediatric bone health and bone disease. His medical practice is mainly medical genetics and he does a small proportion of in-patient general paediatrics every year. He has published extensively.

179. The main points he makes are as follows:

(1) Of the main fractures identified on Jayden he considered if they were as a result of NAI there would be a high likelihood there would be bruising. He relies on two articles to support that view, McMahon and Matthew, both concluded that there was a strong association of soft tissue injury, including bruising, in infants who were abused and had fractures.
(2) Rickets is a systemic disorder that affects every bone in the body. Jayden vitamin D deficiency was very severe from the tests conducted. He questions the ability to accurately assess bone strength under a microscope in an infant who has rickets in the absence of proper tests being done.
(3) In his own clinical experience dealing with unexplained fractures and in reading the literature his view was that infants can incur fractures with

minimal trauma because they have an intrinsic bone disorder, such as rickets. He relies on an article by Koo which concluded that physical therapy may result in fractures, although that study concerned only premature infants. He disputes the ability of Professor Malcolm to be able to distinguish between fractures as to which may be caused by rickets and what are not.

(4) He does not accept the view that classical metaphyseal lesion is specific to non accidental injury. In his experience he said he cannot tell the difference between what causes a metaphyseal fracture or what sometimes is not really a fracture but a healing metabolic bone disease or a variant or an irregularity, caused by the vitamin D deficiency. He distinguished the study conducted by Chapman as it did not include many children with congenital rickets and many of the children were older than Jayden, with the majority being over a year. The four children under 6 months did not have congenital rickets. He did not accept Professor Malcolm's analysis that as the growth plate did not have calcium there was no compromise in its strength. He said in rickets the metaphysis is grossly disorganised and there's abundant amounts of what is called non-mineralised osteoid that in his opinion puts that part of the bone at increased risk of being fragile.

Jayden's birth

Professor Page

180. Professor Page is the President of the Maternity and Newborn Forum of the Royal Society of Medicine and she was recently elected as President of the Royal College of Midwives. She qualified as a nurse in 1965, became a midwife in the 1970's and a supervisor of midwives in Oxford in 1988, a teacher of midwives in the same year and a Professor of Midwifery in 1991. She is currently based at Kings College London and has an honorary clinical contract with the Oxford Radcliffe Hospital Trust. She has also published widely and edited three books. Her clinical work consists of being attached to a free standing birthing centre within the NHS which provides antenatal care for women and post natal care. The midwives attend births at the out of hospital birth centre, home births and she also spends some time in the labour ward of the John Radcliffe Hospital. She has a regular commitment to this broad range of clinical work.

181. In her report and oral evidence she carefully considered the circumstances regarding Jayden's birth which she summarised by saying that the labour was both slow at the beginning and fast towards the end. She focused on the propulsive nature of the contractions that even within the womb can press the baby's shoulder against the pubic bone or the back of the pelvis to the extent that they can damage nerves in the baby's neck. This in her view demonstrates how strong the contractions are. It was her view, having considered the circumstances of Jayden's birth that the skull fractures and the axonal changes. The axon is a part of the nerve or the neuron, and the swelling of the superior cerebral peduncle in the mid-brain might be affected particularly by the speed of the birth but also, possibly, by the pressure of the midwife attending the birth and pulling down the baby's head and helping the baby out.

182. She described the McRoberts procedure; the baby's head twists around to slip out from the birth canal and at that point the expectation is that the baby will untwist, will turn slightly. Sometimes when the baby's head has been born she said you wait a while for the shoulders to be born, if they don't appear, the very first step that is taken is the McRoberts procedure, and that's simply helping the mother to put her legs into a very extended position which opens the pelvis, the outlet of the birth canal. Before that the midwife will usually help the baby to be born by applying what's called lateral flexion (putting the hands on the baby's head and pulling down so the top shoulder comes out). If the McRobert's procedure then doesn't work the lateral flexion is tried again. This is considered to be an acute obstetric emergency; she said it is one of the most acute because the baby can die very quickly.

183. She said skull fractures could (although rare) be caused in two ways; first when the baby is descending into the birth canal when the head can hit parts of the mother's pelvis or second, if the birth is very rapid there is a shearing force that shears the skull away from the surrounding of the skull causing bleeding and in a number of cases has been associated with fractures. When asked whether there would be any signs of this she said sometimes there are no signs. Sometimes there can be a swelling or change in behaviour of the baby. She agreed Jayden seemed to be in good physical health in the period after the birth.

184. In relation to rickets she said there was little current awareness of rickets in the maternity services. At the time when she did her training it was more commonly encountered.

Biomechanics

Dr Van Ee

185. Dr Van Ee is a Professor of Biomechanical Engineering at Wayne State University, working part time. The balance of his time he works for a commercial consulting agency. He has a PHD in Biomechanical Engineering and his area of academic and scientific research is in impact and orthopaedic biomechanics. He studies the influence of forces or impacts on the human body and the injuries the body can sustain. The focus of his research is to define the level of forces or the nature of forces that result in specific types of injuries. The orthopaedic biomechanics is more the study of bones and joints and orthopaedic interventions, such as artificial joints, braces etc which can protect the person from injury during an impact.

186. I can take his evidence quite shortly as it doesn't contain anything that is not already known to these courts or is already before the court from other witnesses. His evidence both at the CCC and in this hearing made clear there is little data to be derived from experiment and the enormous difficulties in drawing conclusions because of the complexity of a baby's brain. He concluded from his tests and expertise that the mechanism of shaking is unlikely to result in the angular accelerations necessary to tear the cranial vessels resulting in intradural haemorrhage including traumatic SDH, however he accepted that trauma to the head is associated with SDH. He said violent shaking is much more likely to result in neck and torso injuries than an isolated head injury; a child with decreased bone

strength would be more likely to incur chest, rib and cervical injury from a shaking mechanism and there were no signs of external trauma consistent with a shaking injury or impact.

Mechanism

187. One of the difficulties in this case is that the medical evidence does not suggest a mechanism. In the CCC the Crown's case was that there had been a traumatic event, including impact, just prior to the hospital admission. The evidential foundation for that was the recent bleeding in the skull fracture and fissure. That has since changed as Professor Malcolm's evidence in this hearing has been that that bleeding took place 24 hours prior to death, namely when Jayden was in hospital. The LA in their threshold statement relied on shaking and/or shaking impact but all the experience of the clinicians and experts who gave evidence is that this would result in an immediate collapse and there was no evidence of any contemporaneous injury.

188. Each case turns on its own facts and the lack of a clear mechanism does not prevent, in certain cases, a finding of NAI but it is a factor to take into account.

The parents

189. Another feature of the evidence that I have to weigh in the balance is my assessment of the parents. In doing that I have considered their police interviews, their evidence at the CCC, their statements filed in these proceedings and their oral evidence in this hearing. I have considered the other evidence that was called at the CCC and in this hearing the MGM, Catrina Al Alas, has also given evidence. The parents have been supported by the presence of their mothers and a member of the wider maternal family during this hearing.

190. It is clear to me that whilst there was initial (and understandable) anxiety expressed by the parents respective families at the time when it was discovered the mother became pregnant, once it became clear that the parents wanted to proceed with the pregnancy they received considerable emotional and practical support from the wider family.

191. The parents took up and welcomed all the support they were offered during the pregnancy. The father and maternal family were present when the mother gave birth to Jayden and following their discharge the next day have remained living in the MGM's home where she was on hand to provide support. The MGM described her daughter in evidence as being someone who has always been quiet and gentle and does not lose her temper. Her evidence described in detail that Jayden was very much a wanted baby. She gave a number of examples of the activities the parents would undertake with Jayden and from her observations were always loving and careful with Jayden, always sought advice either from her or the HV/GP when needed. She was at home full time. She didn't work and would help with Jayden when needed. I have no reason not to accept the evidence that she gave as being an honest and truthful account.

192. It is right that the only time she stayed away for the night was the night before Jayden was taken to hospital and I have had to consider whether there was anything

that happened that night, in the context of the history, that would lead me to consider that there was some form of momentary loss of control, which is often said to be the context for shaking type injuries. The MGM said she left about 9pm, after putting Jayden down in his cot. She said there was nothing unusual that she noticed as between the parents or with Jayden other than the fact that he was still unwell.

193. The mother gave oral evidence. She came across as someone who was quiet and reserved. Although clearly finding it very emotional at times, she gave evidence about the circumstances leading up to her pregnancy with Jayden, her relationship with the father, her wish to continue with the pregnancy and her wish for knowledge about what she needed to do to prepare. She welcomed the involvement of Lorna Waite and took up the support and advice she was offered. She described the father being involved in this too.

194. She again became very emotional at times when describing aspects of their life once Jayden was born. She described in detail the joy he brought them both, they took numerous photographs and short video clips of him (some of which I have seen) as he progressed through different stages. She breast fed from the start, as she said she had been told that was what was best for him and it is what she wanted to do. She was feeding at 3 and 4 hour intervals (in the latter stages he went a bit longer between feeds during the night) and said sometimes he took up to an hour to feed. They established a routine which included often going out in the afternoons, bath-time was early evenings and apart from one evening (when she went out for her birthday) she did not leave him in the care of anyone else apart from short periods, for example when she had a bath. There were no concerns raised by anyone about any of Jayden's developmental progress.

195. When describing the events in the early morning of 22.7.09 she said her main concern was that he was not able to feed, after they had dressed him and tried again they contacted the hospital and then the GP. There was no significant challenge by the LA of the account she gave.

196. The father gave evidence. He was somewhat flatter in emotion than the mother. This was described by the clinicians in the records. He described a similar history as the mother had in relation to the progress of their relationship, the pregnancy and the birth of Jayden. He too described his delight at Jayden's arrival and their life after that. He shared many of the tasks, such as changing nappies and bathing and this is supported by the health professionals and the MGM. Apart from the period when he was on his back to work course, the parents were together with Jayden all the time. The father features on many of the photographs and both parents can be seen and heard on the video clips. He attended many of the appointments with the mother and took an active part on them. They all attended an open day at the end of June run by the FNP and had a baby massage class and following that massaged Jayden each day. They both described to me the movements and the technique they used to do that. They described Jayden enjoying this and not being distressed.

197. He was cross examined about an argument he had with his mother, he said it was about who was coming to his mother's birthday party. He said they didn't speak for a short while but then resumed communication and attended her birthday party, which is included in the photos I saw.

198. He was asked about a reference in the GP records to Jayden crying all day when they attended on 20.5.09 about a scab under his nose. He said Jayden had cried but not all day, but had cried on the bus. The mother was not asked about this in these proceedings and it needs to be looked at in the context that they returned to the GP two days later.

199. Although the father did come across as someone who was somewhat flat and unemotional I did not regard that as significant or indicative of any evidence in support of abusive behaviour towards Jayden. It needs to be looked at in the context of what this couple have had to deal with in the last 2 ½ years.

200. They both deny any behaviour that could have caused harm to Jayden. That is supported, in so far as it can be, by the observations of others, both family and health professionals. The only event they can point to where Jayden could have injured his head is banging it against the side of his cot. They said he was starting to roll, he preferred sleeping on his front. There are pictures of him lying on his front holding his head up.

201. My assessment of the evidence of the parents is that despite their youth and the fact the pregnancy was unplanned Jayden was very much a wanted baby, they shared the care of him and there were no concerns about the way they undertook those tasks. They sought advice appropriately and Jayden was progressing well in their care. This is confirmed by the evidence they gave to me, the evidence from Catrina Al Alas (which I accept) and the observations of the numerous professionals who came into contact with the parents whilst Jayden was in their care. Against this background I don't attach any weight to the comments about the parents' behaviour or demeanour at UCLH and GOSH. That was not explored in cross examination and I can see from the CCTV recordings at UCLH the devastating effect, in particular on the mother, on hearing the news about the seriousness of Jayden's condition.

Discussion

202. The court has had the benefit of hearing many experts who are experienced and have wide expertise in their field. What is of note is that despite their considerable expertise and experience there is very limited direct experience of dealing with babies of Jayden's age with congenital rickets similar in severity to the circumstances of this case. Most have not seen an example of this severity or if they have, it was many years ago. It is against that backdrop the court has to analyse this evidence and consider whether the threshold criteria are established. I shall consider the evidence under two separate broad headings: the fractures and events leading up to Jayden's death. I undertake each of these tasks by reminding myself that the burden of proof is on the LA and the need for the court to be satisfied on the balance of probabilities.

The fractures and the fissure

203. It was not discovered until after Jayden died that he was vitamin D deficient, as was his mother. As a consequence there is no issue he had congenital rickets and (as

I understand it) nutritional/acquired rickets as he was exclusively breast fed by his mother who was vitamin D deficient herself.

204. From the evidence I have heard (and I accept it is to some extent a developing picture) it is a curious feature of this condition that on occasion it can be readily seen on some imaging on others not; it is only in the context of post mortems and/or histological examination can the full extent be seen. This case was a classic example of that. There is an issue as to the extent to which it was apparent on the images taken when Jayden was alive and through the blood tests, it was only when the post mortem place that the true extent was seen. Jayden was at the severe end of the spectrum in the direct experience of both Dr Scheimberg and Dr Smith, who both have many years experience between them. Another feature of this case, which makes it so unusual, is the severity of the deficiency and manifestations of that in the bones on a baby aged only 4 ½ months. It was effectively outside the clinical experience of any of the medical witnesses.

205. There is no evidential dispute about the existence of the fractures. The dispute arises in relation to some of them where it is said by the LA that they fall outside the 'rough handling' explanation given by Professor Malcolm. These can be subdivided into three categories:

(1) Classic metaphyseal fractures
(2) Skull fracture and fissure
(3) Fractures of the right tibia and humerus

206. Professor Malcolm says there are three metaphyseal fractures which can only be caused by a rotational mechanism which is excessive, which is beyond normal handling of a baby. As was made clear in his evidence his view in relation to these fractures where there is rickets has changed. Prior to the Chapman study he accepted that patients with metaphyseal lesions are sometimes said to have pathognomonic findings of either rickets or abuse. His view changed as a result of the Chapman study which he said demonstrated that this type of fracture was not observed. There are, in my view, real difficulties in placing so much reliance on the Chapman study in support of his opinion in this case. This is for the following reasons:

(1) It appears the imaging relied on in that study was limited and did not include skeletal surveys which would capture any asymptomatic fractures;
(2) There is no detail given as to when the parents were made aware of the diagnosis of rickets and whether that would have affected the way they handled the child or the way the child behaved;
(3) There is no detail in the study of whether any of the children had congenital as compared to acquired rickets and the level of severity of the Vitamin D deficiency.
(4) The study acknowledged that some of the metaphyseal fractures seen had features 'closer to' the usual classic metaphyseal fractures.
(5) In an area where there is limited research and limited clinical experience in situations similar to those found in Jayden the court should be cautious in placing too much reliance on this one study.

207. If the Chapman study is not a reliable foundation for Professor Malcolm's view then, in my judgment, it is reasonable to place more reliance on to his previous view about these fractures, prior to the Chapman study, namely that these changes could be a feature of rickets.

208. Turning to the skull fracture it is his view that this was probably caused by trauma. To support that conclusion he relies on the following matters:

(1) the sub scalp bruise;
(2) his view that the lack of calcium would make the skull more pliable and therefore less easy to fracture;
(3) the conclusions of the Chapman study that no skull fractures were noted.

209. The difficulty with this analysis is that, in my judgment, it too lacks firm foundation for the following reasons:

(1) In addition to the matters outlined above about the Chapman study it did not, according to the report, include images of the skulls of those who took part;
(2) There is no evidential foundation, as I understand it, for saying that following a diagnosis of rickets some bones are more likely to fracture than others;
(3) The evidence in relation to previous brain injury is equivocal as between Dr Smith and Dr Ramsay;
(4) There is no evidence of any external mark consistent with such an injury;
(5) There is evidence as to the fragility of the skull bone during the post mortem which has not been challenged;
(6) The sub scalp bruising although indicative of a minor trauma did not support major trauma;
(7) Dr Cary's view was that such a fracture would require significant impact from the blunt force but there is no other evidence to support that.
(8) The timing of the bleeding around the fissure is within 24 hours which is when Jayden was in hospital.

210. Turning to the fractures of the tibia and humerus Professor Malcolm's conclusion is based on the fact that having looked at the cortex of the bone, he said *'My opinion, looking at the cortex of this bone [humerus] there is sufficient bone there that I think it would still take quite a degree of force to actually cause it to fracture completely.';* a little later he said *I am used to looking at the bone thickness, both in children and in adults. It is part of my job.'*

211. In my judgment, in the circumstances of this case, that opinion lacks firm foundation for the following reasons:

(1) Whilst acknowledging his considerable experience and expertise Professor Malcolm lacks a comparator in this case due to the very limited number (only one other in the last 30 years) of a case as severe as the rickets were in Jayden in such a young baby;
(2) His analysis was of the uncalcified bone and therefore he was not able to consider ways of testing its strength and the extent of the calcium, if any, that was present in the bone. It is unfortunate that Dr Scheimberg's email request was not clearer as to what she required.

212. In addition to the specific matters I have outlined in relation to each category I have also taken account of the following factors:

> (1) There is no external evidence to support or indicate that Jayden was being assaulted by his parents; if anything the evidence all points the other way. That evidence is importantly not just self reporting by the parents but was witnessed by a range of professionals with whom the family had contact with on a regular basis over the four and half months he was in their care. Whilst such behaviour can't be ruled out it is unusual with this degree of 'visibility' by the parents and Jayden for something not to be noticed.
> (2) There was no evidence of marks or bruises that could indicate that such abusive actions have taken place. Within the timeframes indicated by Professor Malcolm the child was very 'visible' to the professionals through the appointments he attended.
> (3) The relatively unusual nature of Jayden's condition in the experience of the relevant experts and the literature which did not give them any reliable comparison with other similar cases.
> (4) Whilst not scientific Dr Scheimberg, who is experienced in conducting post mortems of children of Jayden's age considered the bones (the rib and the skull) to be very fragile, in her experience and she compared them to newborn baby bones.
> (5) I acknowledge that Professor Malcolm is the only expert histopathologist who specialises in bones but due to the particular circumstances of this case and for reasons outlined above I consider there are grounds upon which I can depart from his opinion.
> (6) The evidence in relation to the traumatised fissure is now far from clear in the light of the timing of the fresh bleeding within 24 hours of Jayden's death. There is an issue about whether granulation was present which was not fully explored with Professor Malcolm. But in the light of the timing of the recent bleeding and my findings about the fractures I do not consider the traumatised fissure is more likely than not to be a result of inflicted injury.

213. I have therefore reached the conclusion for the reasons outlined above that I can't be satisfied, on the balance of probabilities, that any of the fractures or the traumatised fissure were as a result of inflicted deliberate harm caused to Jayden by either of these parents. In my judgment, on the evidence, the fractures could have been caused by the day to day handling of a young baby due to the particular fragility of Jayden's bones as a result of the severity of his undetected rickets. It has been suggested that the skull fracture could have been caused at the time of Jayden's birth. On the evidence I have that is unlikely due to the passage of time and Jayden's presentation after birth, but it can't be ruled out.

214. Although I am not asked specifically to find this by the LA I consider the other fractures that Professor Malcolm said could be due to rickets rather than any abusive act by the parents. I am satisfied on the balance of probability that that is correct.

215. I have considered the evidence given by Dr Smith and Dr Barnes in reaching my conclusion but not attached significant weight to it. Dr Smith's evidence mainly did no more than draw the court's attention to the relevant literature, some of which supports the general point that children with rickets can sustain fractures other than

through inflicted injury. He was able to give some helpful direct clinical evidence although it was somewhat generalised. Dr Barnes was helpful in outlining the relevant parts of the imaging which, in his opinion, showed signs of rickets. But I have to consider his evidence in the context that by the time he was involved vitamin D deficiency and rickets were an accepted feature of the case. As I have said earlier this case has demonstrated the difficulties in identifying rickets just by images. In this case it was queried by the radiologist at UCLH, missed by the radiologists at GOSH and picked up by the paediatric pathologist.

Events leading to Jayden's death

216. I now turn to the difficult task of seeking to unravel the events leading up to Jayden's death and look at the evidence relating to the differential diagnosis.

Seizures or not?

217. One of the first issues I need to determine is whether Jayden was seizing prior to his admission to hospital or whether he was to all intents and purposes unconscious and/or demonstrating decerebrate posture.

218. I have reached the conclusion that it is more likely than not that Jayden was suffering from seizures prior to his admission to UCLH. I have reached that conclusions for the following reasons:

(1) The description given by the parents of what they observed from about 5am on 22.7.09 has not seriously been challenged and I accept it. In their written closing submissions the LA set out what they said were inconsistencies in the various accounts given by the parents. Very little of what was set out was put to the parents in cross examination, or it was put in part to one but not the other. Considering the various accounts they have given (their police interviews, their oral evidence at the CCC, their statements in these proceedings and their oral evidence during this hearing) their account has remained broadly consistent. They describe how they found Jayden in his cot, laying to one side with his arms through the bars; the difficulties in feeding with his tongue stuck to the roof of his mouth; trying to engage him with his toys; the descriptions of him reaching out for the keys and drawing them to his mouth and that his eyes followed the toys and his mother. Their primary concern was that he was not feeding. Their prompt phone call to the hospital and the GP has not been disputed. Neither has their description of being able to feed and change him and not noticing any stiffness in his limbs. Their description of him shaking/shivering on their way to surgery accords with the description they gave the GP.

(2) The evidence from the GP both from the records and his oral evidence is not consistent with a child who has lost consciousness or exhibiting decerebrate posture. He conducted a full examination, observes Jayden as alert and observes him being undressed and dressed. They were with the GP for about 25 minutes, although he was not with them all the time. Even making allowance for the inexperience of the GP the description he gives is consistent with what was observed by the parents. The lack of any sense of urgency (although inexperience must have played a part in this decision) is graphically

illustrated by allowing the parents to take Jayden to the hospital by public transport.

(3) The CCTV and stills of Jayden on the bus are consistent with the parents account and the GP's observations.

(4) The arrival at UCLH and Nurse Edward's observations are consistent with seizures. She is an experienced nurse, trusted by the Consultant Paediatrician. She immediately noticed something was wrong and observed tonic/clonic seizures and alerted Dr Sutcliffe. When he first saw Jayden and the parents he did not observe anything obviously wrong and finished off his other appointment. I consider it more likely than not that he gave reassuring words to the parents and that is what prompted the mother to phone her mother to say everything was fine. If Jayden had been unconscious or exhibiting decerebrate posturing Dr Sutcliffe as an experienced paediatrician would have noticed that. It is more likely that he was having fluctuating seizures. Nurse Edwards continued to observe what she considered were seizures and Jayden was seen by Dr Sutcliffe. He observed the abnormal movements and immediately made the referral to the resuscitation unit. It is of note that when he spoke to Dr Salt to warn her Jayden was on his way he said it was because of fitting. The subsequent suggestion by him that he observed decerebrate posturing at that time needs to be looked at in that context of the contemporary notes and recordings and what Nurse Edwards observed, which she was clear were seizures. I do not accept the suggested criticism of sending Jayden to the resuscitation unit rather than administering anti convalescent medication there (which they did not have) or requesting the resuscitation team to come to them. The unit was not far away and he sent a doctor with them.

(5) They were met by Nurse Leech in the resuscitation unit and she clearly realised something was wrong. She immediately realised Jayden needed to be weighed for medication and did so and took off his clothes. She did not observe any stiffness when she did that but did observe the seizures. Dr Salt soon arrived and between them they conducted their assessments. There has been a lot of evidence about the various scales to measure consciousness (AVPU and GCS) and how reliable they are particularly in such a young child. Standing back from that I consider what is important is that the form for the measure of the GCS being used they were familiar with using, they work as a team and are both experienced clinicians. Making all due allowance for the subjective element of these assessments I am satisfied, on the balance of probabilities, that they are broadly accurate. The significance is the deterioration between 11.35 and 12.20 when the score falls from 13 to 6.

(6) The fact anti-convulsing medication was administered immediately, followed by two more doses until the fitting appeared to subside.

(7) I have carefully considered Dr Peters evidence in relation to this and have weighed up his undoubted clinical experience in dealing with these cases but I am concerned that there is an element of his evidence that seeks to continue to justify his firm opinion formed at a very early stage as to the cause being due to some form of non accidental trauma. In my judgment he has had to try and account for the clinical picture ex post facto which in part does not sit comfortably with the early opinion he reached. Even making all due allowances for the difficulties in assessing consciousness the clinical evidence, from different sources, points towards repeated seizures.

(8) Dr Jansen, who has expertise in this field, both in terms of her qualifications and her clinical experience was instructed on behalf of the parents. She considered the clinical picture very carefully and had access to all the records. Her report carefully analyses the issues as she did when she gave her evidence. Her evidence is clear, and I accept it, that Jayden was suffering from seizures from at least 5am, that it rapidly became status epilepticus and was clearly refractory due to amount of medication required to control it.

219. The next matter I have to consider is the chronology of treatment received by Jayden whilst he was in UCLH. It is submitted on behalf of the parents that the treatment between about 2 – 6pm on 22.7.09 was 'sub-optimal'; I agree. Great efforts have been made by the legal teams to seek to establish what took place, in particular the events between 2 – 6pm. The juniors met with Nurse Jeffery to take a statement to provide extra information and Dr Salt was recalled to give what information she could. There is no doubt that it was an extremely busy day at the hospital; it is accepted that it was soon after the outbreak of swine flu and Dr Salt said she had one other child on the unit who was as seriously ill as Jayden was. I have reached the conclusion I have about Jayden's care at this time for the following reasons:

(1) Dr Salt accepted that once Jayden was intubated the anaesthetists took effective control. The difficulty with that, and it may be in part due to the particular pressures of that day, is that there was no effective paediatric clinical oversight or driver in relation to the decisions made and events that afternoon. This was particularly when the effect of the muscle relaxants given to Jayden for the purposes of intubation meant that it would have been very difficult to monitor any continuing seizure activity. Dr Salt said when she was recalled that the dose of phenytoin given at 12 noon would have lasted 24 hours. That did not really deal with the point that there could be no certainty that it was actually working with Jayden, bearing in mind that he required three doses of anti convulsing medication prior to being intubated. Dr Peters suggested that this could be monitored by any changes in blood pressure or temperature. The difficulty with that is that there is evidence to support the fact that he was probably still fitting; Dr Runnacles notes in her statement that when she was contacted by Dr Salt about 5.45 pm there was raised blood pressure and she advised a further saline bolus. The clinical notes record morphine and medazalam (an anti convulsant) being prepared earlier on in the day but not administered. They were administered at 17.55. At 18.45 Dr Runnacles noted further seizure activity and administered lorazepam and a loading dose of phenobarbitone to control the seizure activity. All this points towards Jayden's fitting not having been adequately controlled during the afternoon. He was out of the effective direct clinical management of the Consultant Paediatrician.

(2) When he was intubated the second time the tube went too far down into his right lung, causing his left lung to partially collapse, as recorded on the post intubation x-ray taken between 14.08 and 14.12pm. Dr Shaw very helpfully produced the hospital records which show that she viewed the images at 14.34 and said she immediately phoned Dr Shaw to inform her about this. It appears more likely than not it was corrected soon after that (although his CT scan is timed at 2.39pm so it is unclear whether he would have been enroute at the

time Dr Salt was alerted). In any event it appears this compromised his ventilation for at least 20 minutes. Dr Peters sought to play this down saying it happens regularly and suggested that it was in part due to gases from the stomach. The blood gas measurements taken at 14.24 showed Jayden's CO2 level had increased dramatically to 11.3 (twice the normal range). It is accepted that this will adversely contribute to his condition. It is unclear how or when this was corrected as there are no contemporaneous records about the management of this. The next blood gas was taken at 15.38 but that recording shows there was insufficient to measure and no attempt was made to take it again. The next one was not until 16.13 when the CO2 had dropped to 2.44 and remained at around that level for the next two tests taken at 16.17 and 17.17. Dr Peters has sought to rationalise this in two ways. First, by suggesting that there was no evidence that Jayden was hypoxic as the pulse oxymeter level was 100%. Professor Nussey said that only gave part of the picture. Second, that when considered in conjunction with the pH level it demonstrates that the body was self adapting to this change. Professor Nussey said that again only gives half of the picture. I prefer the evidence of Professor Nussey, whilst he may not have the same clinical experience as Dr Peters his expertise in considering the consequences of such changes is clear. As Dr Peters said *'It sounds like he [Professor Nussey] understands the chemistry better than I, but that translating into a clinically meaningful difference I just don't understand'*. It is more likely than not, on the information I have, there was not proper management of Jayden's CO2 levels during this period and that both the high and low level are both likely to have contributed to his deteriorating condition.

220. I shall now turn to consider the evidence as to how Jayden's ICP developed once the retrieval team arrived. Dr Runnacles and Nurse Mogridge assessed him after the handover at 18.40. Dr Runnacles immediately noticed the eyes were unequal, which had been recorded as equal 30 mins previously and he was fitting. She said she was concerned about the raised ICP and administered a saline bolus. After taking further advice, his condition did not improve and over the next hour or so she administered the equivalent of 9 3% saline bolus (the equivalent of 6 were administered over 15 minutes, between 7 – 7.15pm). This was solely done to try and reduce the raised ICP, which was clinically apparent by the changes in the eyes. Nurse Mogridge agreed in her evidence this was due to a very sharp rise in Jayden's ICP.

The Triad

221. This is an area of some controversy with strong feelings on both sides of the medical profession. Following a number of high profile cases the President of the Royal College of Pathologists convened a meeting to explore what consensus there was on the vexed topic. Following that meeting a document was produced which has been referred to in this hearing and at the CCC. It is an extremely helpful document and many of those who contributed to that meeting have given evidence in this case. I note the caveat given at the beginning of the document that not everyone responded with any amendments to the document. It is of concern that in this hearing Dr Bonshek (and I think Dr Cary) mentioned that in relation to retinal haemorrhages it was never intended that macular folds should be listed in the way

that it has in the conclusions. I bear in mind the observations in *Henderson v R (ibid).*

222. The role of the court in considering each component part is to consider, in particular, any differential diagnosis. As Dr Cary said, any break in the link, means the whole picture needs to be re-evaluated.

Retinal Haemorrhages

223. As one would expect there were, in reality, limited issues between Dr Bonshek and Professor Luthert. I obviously consider their evidence in the clinical context as seen by Dr Nischal and as evidenced by the RetCam images. They don't dispute his findings save that in their experience they are not at the severe end of the spectrum they see.

224. Having considered their evidence I have reached the conclusion that they are more likely than not to be secondary to the hypoxic ischemic injury, as concluded by Professor Luthert. I have reached that conclusion for the following reasons:

(1) Although Dr Bonshek in his report concluded that the findings were supportive of a traumatic cause he accepted in evidence that they could be caused by a very steep rise or spike in ICP. Unfortunately he had taken the history from a summary in the GOSH clinical papers, which did not have the full picture, which he was not aware of. When he was taken through it, in particular, the events around the time the retrieval team were dealing with Jayden he agreed there was a very steep rise in ICP that could cause the retinal haemorrhages. That was confirmed by Nurse Mogridge in her evidence.
(2) I accept Professor Luthert's analysis about the significance of the papilloedema and the optic nerve swelling.
(3) Professor Luthert had direct experience of a case where there were retinal haemorrhages similar to the pattern seen in Jayden where there had been no trauma.
(4) I preferred Professor Lutherts more cautious approach to the conclusions that can be drawn from the existence of retinal haemorrhages and their formation. He drew a very clear distinction between cause and mechanism.
(5) The case is complicated by the impact of the changing CO2 levels between 2 – 6pm on 22.7.09 and the effect those changes would have had on the blood vessels, possibly making them more likely to burst. Both Professor Luthert and Dr Bonshek agreed this could have been a relevant factor.
(6) Finally, it is important that the time of Dr Nischal's observations are factored in. He saw Jayden nearly 30 hours after he had been admitted to hospital and the detailed clinical picture was not known by Dr Nischal.

Encephalopathy

225. This aspect has been the most troubling and difficult to unravel. However, having considered the evidence I have reached the clear conclusion that, on the evidence I have seen, I am not able to conclude that it is more likely than not that the hypoxic ischemic encephalopathy had a traumatic cause. In my judgment it is more likely to have been caused by a combination of different factors, some of which may be

unknown, but not including inflicted trauma, either by way of impact or shake or any other mechanism. In reaching that conclusion I have considered the following matters:

(1) It is agreed by all the experts who operate directly or indirectly in this area, and Dr Peters who is an experienced clinician, that the expected and typical presentation in this type of case if it had been caused by trauma of some kind would be immediate collapse. As Dr Peters said in his evidence in the CCC "*one would expect that after an injury is inflicted on a child that causes the triad and which causes axonal swellings localised to the brain stem that the child is immediately and permanently unconscious."* There have been references to different scenarios in the literature but that is not the experience of those who gave evidence. On any view Jayden's presentation and history in the first part of 22.7.09 was not an immediate collapse or loss of consciousness.

(2) In the light of my findings about the impact of Jayden's rickets on his bones, the lack of any injury to the ribs (or any other sign of relevant external trauma), in the particular context of this case, where the mechanism of shake and/or impact has been the mechanism that has been put forward (both in the CCC and in this hearing) is relevant. As Dr Scheimberg said "*..we know from the fresh bleeding in the fracture site which happened while he was in hospital. So obviously he is a very fragile child"*

(3) I accept both the evidence of Dr Jansen and Professor Nussey that Jayden's low calcium levels would affect him in the two ways they describe; adverse impact on his immunity system and make him more likely to have seizures.

(4) I have made my findings about Jayden's presentation during the morning of 22.7.09. The primary cause of his seizure is unknown; the most likely contender is probably a form of febrile seizure which although there was no raised temperature due to his calcium levels, he was more likely to seize with a much lower level of infection. That could either have been related to his flu or possibly meningitis; the latter was never ruled out (through a lumbar puncture) although the clinical signs were not readily apparent. The low level infection cause is supported from a number of other sources: raised white blood cell count, raised CRP, the inflammation of the brain. Dr Ramsay considered an odd feature was the degree of leptomeningeal inflammation in the subarachnoid space purely in association with hypoxic-ischemic brain injury. This indicates that something was stimulating or irritating that part of the brain raising the possibility of an infection and specifically with a pattern like this, an infection by bacteria. Whilst none of these features would be enough on their own, taken together they provide support for that being the primary cause for the seizures. Jayden had been unwell with flu type symptoms for a number of days.

(5) The seizures were not effectively treated until just before 12 noon, some 7 hours after changes were first noticed in Jayden.

(6) It is more likely than not that Jayden carried on seizing, undetected for some, or all, of the afternoon between 2 and 6pm on 22.7.09. I have outlined my reasons for that above. This is likely to have contributed to the infarction of the brain.

(7) The difficulties with both the intubation tube and the changed CO2 levels are also likely to have contributed to the hypoxia and the adverse consequences that flow from it.

(8) I accept the evidence and analysis of Dr Jansen and Professor Nussey in relation to these aspects as they bring together the important twin features of the clinical expertise and also the knowledge of the wider impact of these changes on the different systems that operate in the body.

(9) The evidence in relation to axonal damage is complex. Both Dr Smith and Dr Ramsay have considerable expertise in this area, Dr Ramsay gave a detailed account of his experience. Dr Smith said that he had not seen axonal swellings outside motorbike accidents or admitted shakes however he conceded a non–traumatic cause was a possibility, he said it needed to be considered in the context of the overall ischemic damage Jayden suffered. He agreed axonal damage is not diagnostic of inflicted trauma. He said in his oral evidence and his section 9 statement the existence of the axonal damage raised the *'possibility of trauma'*. In relation to damage in the lumbosacral part of spinal cord being an indicator for trauma Dr Ramsay had two important caveats to that: First, you need to know what the normal state of affairs in the infants spinal cord is and it has been seen in infants who die in situations where there is no suspicion of trauma, which Dr Ramsay had direct experience of. Second, traumatic injury to the spinal cord would leave signs of direct evidence of trauma (fractures to the vertebrae, bruises in the tissue, tearing of muscles) which is not present in this case. Red cells were present in the nerve roots; that is, in his view, a non-specific finding. In relation to the other axonal injury Dr Ramsay said that Jayden had both the classic axonal injury (which is established to be linked to trauma) and the plemorphic form (which can be related to other situations of brain injury unrelated to trauma). The presence of the pleomorphic form is not surprising in the context of severe ischemic injury but the classic axonal swellings are restricted to the brain stem and had, what he considered to be, rather peculiar appearance in all but one site. This suggested to him they had been around for some time. He said the only classic axonal swellings were those in the inferior cerebellar penduncle, which he agreed could indicate trauma, but would be associated with the child becoming *'immediately and permanently unconscious'*. Dr Ramsay also considered the absence of siderosis (haemosiderin at the sites of the haemorrhages) and astrogiolsis (no reaction to the astrocytes) are relevant as contra indicators from the cause being trauma. Bearing in mind the caution Dr Smith gives to these indicators and the caveats outlined above from Dr Ramsay I do not consider they do any more than indicate the possibility of trauma but have to be looked at in the context of all the other evidence in this complex case.

Sub Dural Haemorrhage

226. There was a bilateral thin film subdural haemorrhage over the frontal left hemisphere. The source of such bleeding is a matter of considerable debate. Those present at the Royal College of Pathologists meeting in December 2009 (including Dr Cary, Dr Bonshek, Dr Cohen, Prof Luthert, Dr Scheimberg and Dr Smith) agreed that: "*in very young children, subdural haemorrhage must be interpreted with considerable caution because (whether due to hypoxia or direct mechanical trauma) it could be due to damage at birth."* The meeting could not produce

agreement on whether the source of the bleeding was venous plexuses or bridging veins *"though the majority of those present seemed to regard both sites as possible"*.

227. The debate in this case has centred on two particular aspects. First, the observations made by Dr Scheimberg during the post-mortem that all the bridging veins were intact. Second, what other causes there could be for SDH other than trauma.

228. As set out above Dr Scheimberg describes the process she undertakes in looking to see if the bridging veins are intact. The point made by many of the other experts is that such a process cannot exclude torn veins that can't be seen by the process adopted by Dr Scheimberg and in any event any torn or damaged vein may be masked by the blood. Dr Scheimberg said in this case it was only a thin film and she felt she was able to see but didn't vigorously suggest that she could see all the veins. Her intention in undertaking this process is to seek to contribute to the debate about the source of such bleeding.

229. The observations made by Dr Scheimberg and Dr Cohen in 2008 in their observational study *"Evidence of Occurrence of Intradural and Subdural haemorrhage in the context of HIE"* report that the *"occurrence of SDH on the convexities of the cerebral hemispheres is not an unusual finding in the setting of intrauterine, perinatal or neonatal deaths, the haemorrhage usually presenting as a thin film…."* Their findings demonstrated that*: " …SDH and cerebral hypoxia are common associations of …. SDH (often seen as a thin film)."* They confirmed these observations in their oral evidence, however they have to be looked at in the important context of their method of selection (where there may have been birth related trauma present) and that the oldest child they saw this in was 19 days old. Further observations were reported in the Cohen, Sprigg and Whitby paper published in 2010 which concluded *"Our results exhibit an association between IDH, SDH and hypoxia in children dying of natural causes."* This reported association has not been noted by any other practitioners operating in this field.

230. Another factor that needs to be considered is the impact of the raised CO2 level in this case. It is accepted that the consequences of that would be to cause dilation of the blood vessels which does increase the likelihood of them bursting.

231. All the other relevant expert evidence did not support the association made by Drs Cohen and Scheimberg that SDH can be caused by hypoxia. Some expressed more strident views than others, Dr Cary considered their papers *'fundamentally flawed'*. Dr Smith and Dr Ramsey adopted a more moderate tone. Dr Smith said it represented a *'great leap'* from established literature and day to day experience of many practitioners, he continued *'I am not saying that the literature published by Cohen and Scheimberg is wrong. They just seem to be seeing something different from everyone else and there clearly needs to be more research around this area.'* Dr Ramsay said "*The evidence in the literature for such haemorrhages developing in the context of hypoxia and ischemia subsequently is in a state of evolution. There is some evidence this may occur, but then there are other papers that I do refer to in my report - specifically those by Bayard and Hurley - that suggest that pure hypoxic-ischemic injury itself does not cause these haemorrhages. My own view is*

that this is an evolving part of the medical literature, that there are good grounds to believe that under certain circumstances in severe hypoxic-ischemic injury such haemorrhages can take place but that the acceptance of that as a mechanism is not wide."

232. On the evidence I have in this case I have reached the conclusion that the SDH is more likely to have been caused by trauma. However, that has to be looked at in the context of all the other evidence and, in particular, my findings about the other component parts of the triad and the *'wider canvas'*

Conclusion

233. Having now considered all the evidence, and my analysis of each part as set out above, I have reached the conclusion that even though the presence of the SDH points towards Jayden's injuries being caused by trauma the balance of the evidence points the other way. As Dr Scheimberg said '*So, could it be torn bridging veins? Yes. But does it fit with the rest of the picture? No. That's the problem. When you tried to put it all together is when the thing starts crumbling all over the place. Each individual step, it's possible. But when you put it all together, it [trauma] stops being probable.'* I agree.

General matters

234. The complexity of this case is perhaps obvious by the length of this judgment and breadth of expert evidence the court has heard from. There are a few general observations I would like to make.

235. Despite the extent of the dispute between the various experts, the one aspect they were all agreed upon was the need for further research. In particular research in relation to the different aspects of the triad and the impact of Vitamin D deficiency and rickets on babies under 6 months. I wholly endorse that view.

236. I am very aware that this court has had the opportunity, as did the CCC, to consider the events of the 22.7.09 – 25.7.09 in exhaustive detail, with the benefit of expert evidence over a number of weeks. I am acutely aware that the clinicians operating on the ground, dealing with such urgent situations as occurred in this case, simply don't have that luxury. However, one thing has been clear from this case is the importance of the clinical notes and a proper understanding of the clinical history. As Dr Peters described to me, when I asked him about the system in place for the transfer of notes when the retrieval teams are used he described them as *'chaotic'*. He agreed that should be reviewed.

237. The issues surrounding vitamin D deficiency have dominated this hearing. Evidence has been given that it is on the increase, leading possibly to an increase in congenital rickets. I am unclear as to the evidential foundation for that however it is a condition that if identified at an early stage can usually easily be resolved. The identification of it is not easily done, as this case has so graphically demonstrated. Dr Peters readily accepted it was not picked up by GOSH as even requiring further investigation. I echo the observations made by Professor Nussey and Professor Page

of the need to understand this more and ensure that early identification of this takes place in those groups most at risk.

238. The Children's Guardian took no part in this hearing. Those were her instruction to her solicitor, Mr Sharma. He hinted at one of the early directions hearings that there had been difficulty in securing funding from the LSC for instructing counsel. In the light of the fact that he proposed to take no part in the forensic process Mr Sharma did not attend after the third day of the hearing, other than to hear specific witnesses on the instruction of the Children's Guardian. The role of the Children's Guardian can include drawing to the court's attention to all relevant matters to assist in the fact finding exercise [see *Lancashire v DE [2010] 196 at para 19]*. In my judgment in cases as complex as this that remains a valuable role for the Children's Guardian to have. With the benefit of hindsight it is perhaps a role that should have been give more robust encouragement in this case at a case management hearing

239. One of the matters that made the preparation for this hearing so complex was the close proximity between the conclusion of the criminal trial and the start of this hearing. It meant effective case management by this court was limited to making sure the parties assembled all the relevant material into a manageable form, ensured an effective witness template was completed and the practical arrangements for witnesses were in place. All counsel played an important part in undertaking this work. I would like to record the courts gratitude, in particular, to Ms Sambrooks-Wright and Ms Purkiss for the tireless way they undertook this difficult task.

240. Finally, on a practical note, it has been of great assistance in this case to have a number of the relevant trial bundles (in particular the clinical records) in electronic format so they can be sent to the witnesses who can then readily refer to them if they give evidence by video link.