Neutral Citation Number: [2019] EWFC 73

Case No: CM19C05012

IN THE FAMILY DIVISION

Royal Courts of Justice
Strand, London, WC2A 2LL

Date: 11/12/2019

Before :

MR JUSTICE NEWTON

- -

Between :

Essex County Council	Applicant
- and -	
A	1st Respondent
S	2nd Respondent
L	3rd Respondent
The Children through their Guardian	4th Respondents

Markanza Cudby (instructed by **Essex Legal Services**) for the **Applicant**
Mark Twomey QC and Catherine Piskolti (instructed by **Panesar & Co**) for the **1st Respondent**
Paul Storey QC and Ronan O'Donovan (instructed by Bindmans Solicitors) for the **2nd Respondent**
Julien Foster (instructed by BTMK Solicitors) for the **3rd Respondent**
Stella Young for The Guardian, Lucy Hopkins
Hearing dates: 28 October 2019 - 15 November 2019

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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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MR JUSTICE NEWTON

This judgment was delivered in private. The judge has given leave for this version of the judgment to be published on condition that (irrespective of what is contained in the judgment) in any published version of the judgment the anonymity of the children and members of their family must be strictly preserved. All persons, including representatives of the media, must

ensure that this condition is strictly complied with. Failure to do so will be a contempt of court.

Mr Justice Newton:

1. On the morning of 11 July 2017 baby J, who was almost 4 months old was discovered lifeless in his cot by his mother. He was taken to hospital where he was pronounced dead. A subsequent skeletal survey discovered a number of rib fractures of different ages. Yet further investigation identified subdural bleeding. The cause of death was, and remains, unknown. Over a protracted period, the local authority, whilst aware that it was thought that J may be the victim of inflicted injury, maintained the care of the other children in the family by their parents. An early assessment, a later s.47 Assessment, as well as earlier instigation of the Public Law Outline, led to considerable enquiry into the family's circumstances and functioning, and yet it was not until 2019 that proceedings were finally commenced.

2. In fact, the first child protection conference was not convened until 6 June 2018, when the children became subject to child protection plans. At the PLO meeting, held in August, forensic psychological assessment was sought, the report being available in December. A subsequent legal planning meeting (now almost 18 months after the death of J) decided that there was no immediate risk to the children, although the service manager appears to have directed that court proceedings should be implemented, I think because the mother was about to have another child, giving birth on 3 January 2019; it seems it was the pregnancy which finally galvanised the local authority into action. In the application the authority sought an interim supervision order. The matter came before the Designated Family Judge for Essex, HHJ Roberts, on 18 January 2019. When listed for a further (contested) interim hearing on 23 January 2019, because of the unusual history, Judge Roberts transferred the case to me.

3. The local authority maintain that J, prior to death, had suffered inflicted injury on at least two occasions, rib fractures, and the subdural bleed.

4. On 17 July 2017 a post mortem skeletal survey carried out by Dr Carmichael identified:

   i) Fractures with callus formation of the right lateral 6th and 7th ribs.

   ii) Fractures with callus formation of the left lateral 6th and 7th ribs.

   iii) Loss of vertebral height at T7 to T10 and L3 suspicious of fracture.

   Consistent with fractures having occurred between 10 days and 4 weeks prior to death.

5. Professor Mangham, Consultant Histopathologist, reported on 31 October 2017. He confirmed the fractures, and identified another, on the posterior left 7th rib – which was aged as having occurred between 2 and 5 days before death.

6. Professor Mangham concluded that there being no histological evidence of an underlying bone disorder which may have made the bones more susceptible to fracture, or plausible explanation, the number, distribution and different ages of the rib fractures indicated non-accidental injury.

7. Dr Du Plessis, Consultant Neuropathologist, reporting in December 2017, confirmed the earlier identified subdural scar tissue, which reflected a previous episode of subdural

bleeding over both sides of the brain and which was not birth related. Dr Du Plessis advised that the treatment of Dalteparin was highly unlikely to have caused the subdural bleeding. He concluded that:

> "the identification of evidence of previous likely thin film bilateral subdural bleeds which occurred after having seemingly recovered from jugular venous thrombosis combined with post mortem identification of healing rib fractures of a similar age and similar non-accidental association is of significant concern of a previous non-accidental injury."

8. Dr Cary, Consultant Forensic Pathologist, who was instructed as early as July 2017, issued his final report in March 2018, confirming the opinions of Professor Mangham and Dr Du Plessis, concluding:

> "There are healing rib fractures on both sides of the chest described in detail by Professor Mangham. I agree with Professor Mangham that based on the findings there is evidence of at least two separate episodes of trauma, one within a period of 2-5 days prior to death and the other significantly earlier and timed at 10 days to 4 weeks prior to death. I also agree with the opinion that the various rib fractures identified in this case are indicative of side to side compression of the chest typical of forceful squeezing. In my opinion this would be at a level of force significantly in excess of what might be termed rough handling considering that there was no evidence of any underlying bone disease. As well as obvious healing fractures there were other more subtle changes that may represent further signs of rib trauma. There is no pathological evidence of any abnormality of the spine in spite of the suggestions from radiological examination. I have encountered this problem before and would regard the detailed histopathological appraisal of the bones as being the "gold standard".
>
> Although Dr Du Plessis has not identified any signs of acute head or spinal cord injury in the period leading up to death, he has identified healing bilateral subdural haemorrhage timed as occurring around 2-4 weeks prior to death and not felt to be a residual sign of birth trauma. Whilst the eyes showed no evidence in support of head trauma around that time, this does not exclude it. It is noteworthy that the evidence of previous subdural haemorrhage overlaps with the timing of the older rib fractures. In my opinion on the balance of probabilities the deceased suffered a trauma in the 2-4 weeks prior to death characterised by side to side squeezing of the chest and bilateral thin film subdural haemorrhage. Such findings would typically be the result of shaking/impact trauma. I note that shaking is admitted to on the 2nd of May in association with apparent respiratory arrest. Of course, from a pathological point of view an episode of shaking would providence an explanation for respiratory arrest as well as rib fracturing and subdural

> haemorrhage. However, the shaking episode described, if it occurred, was said to have been significantly longer ago than the upper limit of timing for either the older rib fractures or the subdural haemorrhage. This, together with the finding of more recent rib fractures, raises the possibility that the deceased was gripped/shaken on more than one occasion previously."

9. As a result, the mother was interviewed under caution by the police on 24 April 2018. The parents did not and do not accept that J died other than from tragic natural causes.

10. The actions of the local authority need to be reviewed, however, from the evidence as it was then available, which presented, at the time, a worrying indictment against the family. J had a twin, as well as older siblings, and at about the time of Dr Cary's statement, the mother again fell pregnant. In such cases it can never be appropriate for the local authority to simply decide not to issue proceedings, it was a misguided abrogation of their statutory duty. It defies common sense.

11. The delay in examining the evidence has been especially difficult for the family, such cases are never easy, but it has not affected the detail of the comprehensive medical enquiry into what happened to J, indeed it has been greatly assisted by the subsequently extensive Court ordered expert evidence. What is clear however, is that those enquiries should have happened at the outset, and at the very latest by Spring 2018. I have been especially impressed by the helpful and dignified conduct of the family, counsel and solicitors in the case. The approach of the lawyers has yet again emphasised the absolute imperative of the Court having the benefit of the very best specialists in this field. But for their careful examination of the evidence, it would have been so easy for a really major miscarriage of justice to have occurred.

The Law

12. In determining the issues at this fact finding hearing I apply the following well established legal principles. These are helpfully summarised by Baker J (as he then was) in *A Local Authority v M and F and L and M* [2013] EWHC 1569 (Fam).

    i) The burden of proof lies with the Local Authority. It is the Local Authority which brings the proceedings and identifies the findings that they invite the Court to make. The burden of proving the assertions rests with them. I bear in mind at all times that the burden is fairly and squarely placed on the Local Authority, and not on either parent. Other case law (such as *Re B* 2013 UKSC and *Re BS* 2013 EWCA 1146) reinforces the importance of proper findings based on proper facts; the principles are the same for whatever the proposed outcome. Here there is, as in many cases, a risk of a shift in the burden to the parents to explain occasions when injuries might have occurred. Whilst that can be an important component for the medical experts, it is not for the parents to explain but for the local authority to establish. There is no pseudo burden as Mostyn J put in *Lancashire VR* 2013 EWHC 3064 (fam). As HHJ Bellamy said in *Re FM (A Clinical Fractures: Bone Density):* [2015] EWFC B26.

> "Where… there is a degree of medical uncertainty and credible evidence of a possible, alternative explanation to that contended for by the local authority, the question for the Court is not "has that alternative explanation been proved" but rather… "in the light of that possible alternative explanation can the Court be satisfied that the local authority has proved its case on the simple balance of probability."

ii) The standard of proof of course is the balance of probabilities (*Re B* [2008] UKHL 35). If the Local Authority proves on the balance of probabilities that baby A was killed by the mother or sustained inflicted injuries at her hands the Court treats that fact as established and all future decisions concerning the future welfare of B, based on that finding. Equally if the Local Authority fails to prove those facts the Court disregards the allegations completely.

> "the "likelihood of harm" in s31(2) of the Children Act 1989 is a prediction from existing facts or from a multitude of facts about what happened… about the characters and personalities of the people involved and things which they have said and done [Baroness Hale]"

iii) Findings of fact must be based on evidence as Munby LJ (as he was then) observed in *Re A (A child) Fact Finding Hearing: (Speculation)* [2011] EWCA Civ 12:

> "the elementary proposition that findings of fact must be based on evidence including interferences that can properly be drawn from the evidence, not on suspicion or speculation."

That principle was further emphasised in *Darlington Borough Council v MF, GM, GF and A* [2015] EWFC 11.

iv) When considering cases of suspected child abuse the Court must inevitably survey a wide canvass and take into account all the evidence and furthermore consider each piece of evidence in the context of all the other evidence. As Dame Elizabeth Butler-Sloss P observed in *Re T* [2004] EWCA Civ 558 [2004] 2 FLR 838.

> "Evidence cannot be evaluated and assessed in separate compartments. A judge in these difficult cases must have regard to the relevance of each piece of evidence to other evidence, and to exercise an overview of the totality of the evidence in order to come to the conclusion whether the case put forward by the Local Authority has been made out to the appropriate standard of proof."

v) The evidence received in this case includes medical evidence from a variety of specialists. I pay appropriate attention to the opinion of the medical experts, which need to be considered in the context of all other evidence. The roles of the Court and the experts are of course entirely distinct. Only the Court is in a position to weigh up the evidence against all the other evidence (see *A County*

Council v K, D and L [2005] EWHC 1444, [2005] 1 FLR 851 and *A County Council v M, F and XYZ* [2005] EWHC 31, [2005] 2 FLR 129). There may well be instances if the medical opinion is that there is nothing diagnostic of a non-accidental injury but where a judge, having considered all the evidence, reaches the conclusion that is at variance from that reached by the medical experts, that is on the balance of probability, there has been non-accidental injury or human agency established.

vi) In assessing the expert evidence, and of relevance here, I have been careful to ensure that the experts keep within the bounds of their own expertise and defer where appropriate to the expertise of others (*Re S* [2009] EWHC 2115 (Fam), [2010] 1 FLR 1560). I am anxious that the early obtained evidence might be guilty of coming to a peremptory and unsafe conclusion. I also ensure that the focus of the Court is in fact to concentrate on the facts that are necessary for the determination of the issues. In particular, not to be side tracked by collateral issues, even if they have some relevance and bearing on the consideration which I have to weigh.

vii) I have particularly in mind the words of Dame Butler-Sloss P in *Re U: Re B* [2004] EWCA Civ 567, [2005] Fam 134, derived from *R v Cannings* [2004] EWCA 1 Crim, [2004] 1 WLR 2607:

a) The cause of an injury or episode that cannot be explained scientifically remains equivocal.

b) Particular caution is necessary where medical experts disagree.

c) The Court must always guard against the over-dogmatic expert, (or) the expert whose reputation is at stake.

viii) The evidence of the parents as with any other person connected to J is of the utmost importance. It is essential that the Court form a clear assessment of their reliability and credibility (*Re B* [2002] EWHC 20). In addition, the parents in particular must have the fullest opportunity to take part in the hearing and the Court is likely to place considerable weight of the evidence and impression it forms of them (*Re W* and another [2003] FCR 346).

ix) It is not uncommon for witnesses in such enquiries, particularly concerning child abuse, to tell untruths and lies in the course of the investigations and indeed in the hearing. The Court bears in mind that individuals may lie for many reasons such as shame, panic, fear and distress, potential criminal proceedings, or some other less than creditable conduct (all of which may arise in a particular highly charged case such as this) and the fact that a witness has lied about anything does not mean that he has lied about everything. Nor, as *R v Lucas* [1981] 3 WLR 120 makes clear does it mean that the other evidence is unreliable, nor does it mean that the lies are to be equated necessarily with "guilt". If lies are established I do not apply *Lucas* in a mechanical way but stand back and weigh their actions and evidence in the round. I bear in mind too the passage from the judgment of Jackson J (as he then was) in *Lancashire County Council v C, M and F* (2014) EWFC3 referring to "story creep".

x) Very importantly, in this case in particular, and observed by Dame Butler-Sloss P in *Re U, Re B (supra)*

> "The judge in care proceedings must never forget that today's medical certainty may be discarded by the next generations of experts, or that scientific research will throw a light into corners that are at present dark"

That principle was brought into sharp relief in the case of *R v Cannings (supra).* As Judge LJ (as he was then) observed

> "What may be unexplained today may be perfectly well understood tomorrow. Until then, any tendency to dogmatise should be met with an answering challenge."

As Moses LJ said in *R v Henderson Butler and Oyediran* [2010] EWCA Crim 126 [2010] 1 FLR 547:

> "Where the prosecution is able by advancing an array of experts to identify non-accidental injury and the defence can identify no alternative course, it is tempting to conclude that the prosecution have proved its case. Such temptation must be resisted. In this as in many fields of medicine the evidence may be in sufficient to exclude beyond reasonable doubt an unknown cause. As Cannings teaches, even where, on examination of all the evidence, every possible known cause has been excluded, the cause may still remain unknown."

13. I bear in mind too the need to avoid speculation or jumping to a particular conclusion from an unknown cause: *R v Harris* 2005 EWCA Crim 1980 (in relation to the triad of head injuries); *Re R, Cannings and R v Henderson* all demonstrate situations where injuries singly or taken together could give rise to presumptive or misconceived findings, which I fear may have happened here in the initial advices), especially where there may be (as here), naturally occurring conditions that may have caused or contributed to, a particular medical finding.

14. Finally, I have in mind what Hedley J said in *Re R* [2011] EWHC 1715 (Fam), [2011] 2 FLR 1384:

> "A temptation described is ever present in Family Proceedings and in my judgment, should be as firmly resisted as the Courts are required to resist it in the Criminal Law. In other words, there has to be factored into every case which concerns a discrete aetiology giving rise to significant harm, a consideration as to whether the cause is unknown. That affects neither the burden nor the standard of proof. It is simply a factor to be taken into account in deciding whether the causation advanced by the one shouldering the burden of proof is established on the balance of probabilities… a conclusion of unknown aetiology in respect of an infant represents neither a professional or forensic failure. It simply recognises that we still have much to learn and…it is

> dangerous and wrong to infer non-accidental injury merely from the absence of any other understood mechanism"

The Background

15. The mother has had 5 children (including J), an older child with her husband, (though the marriage was short-lived); another son in 2015, J and his twin sibling, born in 2017; and a fifth baby, born in January 2019, which precipitated these proceedings. The mother lived with the father of the 4 younger children and with whom she has had a relationship with since 2013. They recently separated as a result of his drug use. Her eldest child has regular contact with his father, who is a party to the proceedings.

16. Whilst not uneventful, both describe a good and happy childhood. In adulthood the father has been to prison for cultivating cannabis and there have been the more recent difficulties in respect of drug use. The parents reported a good relationship as between themselves and the children, although no doubt in part because of these proceedings (as well as the drug misuse), currently their relationship has become somewhat strained, and they are currently separated.

17. The mother's pregnancy with the twins was completely uneventful until she caught a sickness bug in the 31st week. It seems that brought on contractions, and she went into early labour.

18. The twins were born by Caesarean section on 19 March 2017. They spent 29 days in hospital before being discharged home on 17 April 2017. Within 3 days, on 20 April 2017, J was readmitted to hospital with suspected sepsis. Just a week later on 2 May 2017, J was taken again to hospital with persistent vomiting, he had stopped breathing and turned blue. He was discharged, but early the next morning he was brought back to hospital; he was profoundly ill, vomiting, had a distended abdomen with episodes of apnoea. He had an obstructed large bowel which required surgery; his post operation recovery was complicated by the development of thrombosis within the neck veins. He was prescribed Dalteparin.

19. A computerised scan on 11 May 2017 showed a small volume of extra-axial fluid overlying the central hemispheres and bulging of the fontanelle. He was prescribed Frusemide on 12 May (to 24 May 2017). Chest x-rays on 20 May 2017 demonstrated no fractures. He was discharged home on 26 May 2017. Dalteparin was further prescribed for 3 months.

20. On 22 June 2017 he was again taken to hospital having stopped breathing on 2 occasions. On the morning of 11 July 2017, J was found dead in his bed. He was 114 days old (48 days after his expected birth date). Throughout his very short life he had been almost constantly unwell. Despite a recent rise in his weight, he was visibly very much smaller than his twin. As I have already recorded, the injuries with which I am concerned (fractured ribs and subdural bleed) are, it is thought, unrelated to his death, for which no cause has been established.

Expert Evidence

The Evidence of Pathology

Professor Mangham, Consultant Histopathologist

21. Professor Mangham is an important witness, he examined the ribcage, spine and spinal cord. He identified 5 rib fractures. Right and left lateral 6 and 7 ribs. In addition, a fracture of the posterior left 7th rib. He found well developed callus formation on the right and left lateral fractures. In respect of the posterior 7th rib fracture, he considered that it had occurred 2-5 days prior to death. He concluded as follows:

> "The number, distribution and different ages of the rib fractures indicate that, in the absence of a plausible explanation they were due to non -accidental injury. There is no histological evidence of an underlying bone disease that might have made the bones more susceptible to fracture."

22. Professor Mangham very recently was sent Dr Cartlidge's report and responded, remarkably briefly:

> "I can confirm that J's bones showed no evidence of disease in particular no evidence of osteopenia."

23. I have of course listened to the advices of Professor Mangham before, he is an expert with an excellent and well deserved high reputation. I am not sure what happened here but I cannot hide the fact that on this occasion I found his approach disappointing. I do not know whether it was the source of the original instruction, in other words by the Police as opposed to under the Family Procedure Rules (although that should have made no difference), but he was at best defensive and even, I am afraid, at times confrontational. On occasion he failed to answer simple and direct questions put to him.

24. At the time of his instruction Professor Mangham was not provided with J's medical records which would have been so invaluable; J, after all, had been a very poorly child. Rather, he had available just 3 documents, the MG21, the skeletal survey carried out by Dr Carmichael, and the provisional autopsy report, he could not bring the detail of those documents to mind. More worryingly, from the Court's perspective, was that there were clear factual inaccuracies, not unfortunately remedied by the absences of notes of the examination, or record of the step by step process of the assessment and observations of J's rib cage. Nor finally, in preparation for this hearing, had Professor Mangham reviewed J's medical notes, or the medical chronology.

25. The significance of the foregoing, especially in the light of the very brief email response recorded in paragraph 22 (ibid) was his evidence in relation to osteopenia. So that whilst he accepted:

    a) The link between diuretics and osteopenia

    b) The link between frusemide and osteopenia

    c) The link between intravenous feeding and osteopenia

d) The link between phosphate and osteopenia

e) The link between immobilisation and osteopenia

And accepted that each of the five factors applied to J, he did not appear to accept the World Health Organisation definition of osteopenia, neither could he offer an alternative. Nor surprisingly, consideration of a possible concession to the advices of Dr Watt (in relation to gracile bones and visible osteopenia), let alone Dr Cartlidge. So far as I could ascertain Professor Mangham did not measure J's bone mineral density by any objective method. He could not tell me J's bone mineralisation T score, commenting "that it was buried somewhere in the medical records". He acknowledged that he had not undertaken any histochemistry on J's bones so as to give a clearer picture as to their calcium content. The tests would also additionally have identified whether sufficient phosphate was present. Had I not been told by Dr Leadbetter how bones were prepared for examination, I would not have known that they are decalcified for examination.

26. I have deliberately paused to reflect on Professor Mangham's evidence. It was not, I am afraid to say, of the gold standard. He failed to provide the Court with a balanced view, not just in the sense of approach, but more seriously in relation to the important detail. Whilst he acknowledged the reasonableness of the approach of Dr Cartlidge, having read the reports the day before he gave evidence, he simply discounted them really on the basis of appearance without in fact addressing the detailed observations and several issues concerning de-calcification.

27. At the conclusion of his evidence, the Court was left wondering why there was such a stark and, to my mind, worrying contrast between this witness and almost all the others, who in such a complex case, gave time and thought to alternative diagnoses and mechanisms, and their likely effect on J. I have been left with a sense of unease that the approach here seems to me to have been blinkered and closed and ultimately therefore at least unhelpful. As I say, had I not been told by Dr Leadbetter that bones were deliberately de-calcified by acid for slide preparation, I would not have known that this had been done.

28. Failure to weigh the many alternatives potentially active in this case is an approach which could so easily have given rise to very great injustice. I cannot in the circumstances place weight on Professor Mangham's conclusions.

<u>Dr Du Plessis, Consultant Neuropathologist</u>

29. Dr Du Plessis carried out a neuropathological examination of J's brain. He did not find any acute discernible subdural haemorrhage, nor did he identify any extradural haemorrhage. He observed, however, that "the hemicranial and skull base dura showed evidence of previous subdural haemorrhage over both sides of the brain". That previous subdural haemorrhage was recognised in the form of a thin layer of scar tissue lining the inner aspect of the dura (the subdural aspect). The membranes showed microscopic appearances accommodating an age measured in terms of a few weeks (2-4 weeks) rather than days or months.

> "The identification of subdural scar tissue reflecting a previous episode of subdural bleeding occurring within weeks of J

> Sharp's death is of concern as such subdural bleeding cannot be attributed to birth related dura-associated bleeding."
>
> "Bilateral subdural bleeds in a child of J's age which certainly post-dated birth is of concern as this most likely reflects a consequence of a traumatic head injury".

There was no evidence of recent subdural haemorrhage.

30. In oral evidence, Dr Du Plessis made no attempt to disguise the fact that he had not read (nor indeed endeavoured to read) quite a lot (indeed I have a nagging suspicion any) of the core material sent to him, in particular Dr Keenan's report, the medical chronology or J's medical records.

31. He did acknowledge the importance of seeking a clinician's view (who have the benefit of seeing living patients, being able to see vastly greater numbers of people, and give their opinions in a wider context). Notwithstanding that view, when Dr Jayamohan's clear opinion was put to him (as to the role of Dalteparin acting with venous pressure), he thought that an unlikely explanation, although when pressed ultimately deferred to him. He acknowledged that even if trauma had precipitated the subdural bleed, given the treatment with Heparin, that trauma could be sufficiently minor to be described as normal handling. He additionally added another potential benign explanation for the subdural bleed, the possible extra-axial space. With the other issues the trauma could be very minor. He agreed that thrombocytopenia was an additional risk factor, and deferred to Dr Cartlidge in relation to interpreting the evidence of bleeding propensity from the chest petechiae.

32. Whilst it was unfortunate that he was not as prepared as might have been, nonetheless Dr Du Plessis gave due weight to the opinion of others and tempered his evidence accordingly.

<u>Dr McPartland, Consultant Paediatric Pathologist</u>

33. Dr McPartland examined J's eyes. She recorded that a screening for retinopathy of prematurity took place on 12.04.17 which revealed no such retinopathy. On examination she found:

> Left Eye "no retinal or vitreous haemorrhage is seen on naked eye examination or under the dissecting microscope."
>
> Right Eye ""no optic nerve sheath haemorrhage" "no retinal or vitreous haemorrhage is seen on naked eye examination or under the dissecting microscope."

Other findings are said to be of "questionable clinical significance.

> "both eyes show similar features with no evidence of significant retinal haemorrhages or retinal hemosiderin deposition."

Dr Andreas Marnerides, Consultant Perinatal and Paediatric Pathologist

34. Dr Marnerides performed the Post Mortem together with Dr Cary. Genetic testing detected no abnormalities. There were no findings to suggest an underlying metabolic disease. He reviewed the examinations and reports of Professor Mangham, Dr Du Plessis, Dr McPartland and concluded:

> "1. A detailed post-mortem examination has not demonstrated morphological evidence of an underlying natural disease, congenital or acquired, or medical condition to which J's death can be attributed.
>
> 2. Likewise, a detailed post mortem examination has not identified evidence of acute brain or other injury being the cause of death in this instance.
>
> 3. The combination of the osteoarticular and neuropathologic examination findings raises concerns regarding previous injuries; these injuries, in the absence of plausible explanation, would be due to non-accidental injury (injuries) of shaking/impact type"

35. He considered the mother's account of shaking J on 2 May when he stopped breathing, noting that J died 11 July 2017 some 6 weeks and 2 days after discharge from hospital on 28 May 2017;

> "it would not appear to me to be likely that J's mother's aforementioned account would be in keeping with the observed osteoarticular pathology and neuropathologic features."

36. These observations have to be seen in the light of the later and more considered investigation within the proceedings. Especially having regard to Dr Marnerides' concessions in relation to bone disease and osteopenia in immature babies.

Dr Cary, Consultant Forensic Pathologist

37. Dr Cary conducted the post mortem of J on 21 July 20017 with Dr Marnerides. His final report being available in March 2018. His findings are set out at paragraph 8 (ibid).

38. Once that evidence had all been received in early 2018 at the latest (and which no doubt led to the mother being interviewed by the police), it is difficult to comprehend the thought processes of the local authority. Whatever the Court's findings now, the matter should have been placed before the Court, not just as a matter of statutory child protection, but in fairness to the family. Crucially, either through ignorance or arrogance, the children were denied a proper investigation, which could have seriously impacted on their welfare. A proper assessment of the (complete) medical evidence was necessary in order to be able to evaluate an assessment of risk. What if another child had been injured?

39. Within these proceedings a number of important witnesses have now been instructed.

40. Dr Jayamohan, Consultant Paediatric Neurosurgeon at the Oxford Radcliffe Hospital considered that J had prominent sub arachnoid spaces. He considered that the bilateral subdural neo-membranes should be measured in a matter of weeks, but did not go back as far as birth. No bleeding was present on the imaging at the Royal London. Therefore, the onset of the subdural bleeding both radiologically and pathologically was after the occurrence of the venous thrombosis. Raised pressure can cause a back filling of the veins within the dural plexus, but this by itself would not necessarily be expected to cause subdural bleeding, more it is associated in clinical experience with hydrocephalus caused by a lack of drainage of the cerebrospinal fluid. However, the combination bilateral neck vein thrombosis, plus Dalteparin (causing a relative decrease in the blood's ability to clot), could have contributed to subdural bleeding, either by spontaneous mechanism (although he had not encountered this combination before), or made J more prone to subdural bleeding after trauma. He considered Dalteparin, even on its own, could predispose a subdural bleed from minor trauma during normal handling. He said that it is hard to gauge the spectrum that might be required in such a sick child to cause subdural bleeding, it was difficult to be definitive.

> "if the Court finds that the non-cerebral findings (e.g. the rib fractures) were caused by a shaking event, then the timing that I understand to be given to the bony injuries of the chest would fit with it potentially being the correct timing for the subdural bleeding. "

41. However, Dr Jayamohan concluded that the most likely explanation was the combination of obstruction of venous sinus thrombosis blood flow from the head to the neck. This was likely caused by some element of engorgement of the dura, and associated with the administration of Dalteparin, so that the combination of the two was sufficient to cause a small amount of subdural bleeding. He drew confirmatory support from the independent opinions of both Drs Keenan and Cartlidge.

42. Significantly he advised that J's pressure would not likely have reduced by the date of his discharge from hospital: "it takes months". He was on Dalteparin at home which would keep the blood thin whilst (the clot) breaks down". It may take between weeks and several months to complete.

43. On ageing the bleeding, he raised two issues: the first was that he was unable to say how Dalteparin might affect the usual time frame process (of 2-4 weeks to the age of any neo-membrane) to allow for the organisation and formation to begin. But secondly, significantly, he highlighted what it is that can (not) be seen on a scan. Not all subdural bleeding is identified on an MRI scan. Thus, the bleed might have been present on 18 May 2017.

44. He concluded that there was not just an absence of signs of trauma (and identified a long list) but rather the positive signs of medical treatment in action. He was he said on the same page as Dr Cartlidge. I cannot help but agree with the submission that the analysis of Dr Jayamohan demonstrates precisely the sort of considered, clear and independent thought that the Court expects. Having stood back, Dr Jayamohan was a powerful witness, bringing together rather well in a rational coherent and balanced way the many and different considerations. It is not without note that his evidence, but from a different specialism, echoes and complements the advices of Dr Keenan, Dr Cartlidge, Dr Leadbetter and others.

Dr Russell Keenan Consultant Paediatric Haematologist

45. J's venous thrombosis was treated with Heparin. Heparin increases the risk of bleeding, due to direct inhibition of blood clotting factors, and this extends to intracranial haemorrhaging. Not all blood clotting disorders have been tested for in this case. However, of the ones that were (tested for), an underlying blood clotting disorder was not identified. Heparin therapy when prolonged can cause thinning of the bones and increased risk of fractures. This is only significant with very prolonged administration over many months. Heparin, with one of the identified blood disorders, presents a much higher risk of bleeding (depending on the blood disorder). He recommended testing for such disorders (which has not been done).

Dr Cartlidge Consultant Paediatrician

46. Dr Cartlidge was an important witness, not just in his primary opinions, but also in his affirmation and explanation of the other expert opinions. He was clear that having reviewed the multiple health issues suffered by J, together with the drug treatment endured by him that it became

> "unnecessary to be looking for abusive explanations taking all of that together because he could foresee a scenario in which a child could be injured by an anodyne piece of parenting that would not be remembered."

47. He was specific:

i) J had been premature, and had been very ill.

ii) J had had respiratory failure and cardiocirculatory collapse.

iii) J had been through a long period with a low phosphate level and use of Frusemide.

iv) Frusemide additionally washes out calcium from the body.

v) A normal calcium reading is not indicative of an absence of osteopenia. The most important role for calcium is within the blood. The body is more concerned with maintaining the normal concentration of calcium in the blood, as opposed to the bones. If something has to give, it is the calcium in the bones. Measuring calcium in the blood is not a good way of knowing the level of calcium in the bones.

vi) Prolonged intravenous nutrition was another risk factor.

vii) Immobilisation (therapeutic paralysis), in combination with other factors, but not on its own, was another risk factor.

viii) During his serious illness and hospitalisation, risk and actual factors indicate that osteopenia would have likely developed during that period.

ix) Dr Cartlidge noted that Dr Watt's report (which he had not seen before submitting his own) recorded gracile bones and osteopenia. The two features not being conjunctive.

x) Low phosphate levels were found in J.

48. Dr Cartlidge was clear in relation to the fractured ribs that they would likely have occurred in the course of unremarkable, unmemorable and thus unremembered, handling. The cry of a child in such circumstances would not be different; a carer may not be able to discern the difference. Physiotherapy is a recognised cause of fracture in osteopenic ribs.

49. Dr Cartlidge was cautious about the ageing of the fractures as most of the radiological and pathological criteria have been gathered from children who were not ill in the same way as J. Recovering from osteopenia can take "quite a long time".

50. In relation to the identified membrane, he endorsed the analysis of Dr Jayamohan, given the individual and combination of factors (raised venous pressure, thrombocytopenia and treatment with Heparin). There was, he said, no need to search for other factors such as blood disorders.

51. Stepping back and looking at Dr Cartlidge's careful evidence as a whole, and in the context of the other expert evidence which supports his approach, I was struck by how thoughtful and considered his conclusions were. Deferring to others where appropriate, prepared to contemplate reasonable contentions or hypotheses; nonetheless reaching clear, coherent and balanced conclusions, upon which I rely.

Dr Stoodley Consultant Neuroradiologist

52. Dr Stoodley reviewed the CT scan performed on 11 May 2017 and MRI scan performed on 18 May 2017. The scans showed "no evidence of any acquired brain injury". The only bleeding evidence on the scans is of old sub-ependymal haemorrhages which are likely to have occurred in the neonatal period and are a common finding in premature infants."

Dr Watt, Consultant Paediatric Radiologist

53. In relation to propensity to fracture he advised that:

i) he agreed with Dr Cartlidge in respect of matters in which they share the same expertise and deferred to him when Dr Cartlidge's expertise exceeded his own.

ii) J had gracile bones and signs of osteopenia already in the hospital at 3rd May 2017 and thereafter.

iii) J's ribs were gracile, meaning thinner bones (not as wide in terms of measurement) than a normal term time child at that age.

iv) The commonest fractures seen in infants of this age are rib fractures, as bone density drops in first weeks and months of life. This is why, in general, fractures occur in the first few weeks or months of the premature cohort.

v) The chest radiograph dated 20th May 2017 showed that J's bones were thinner than other children by comparison when looking at radiographs of children of the same age.

54. In relation to level of force or type of handling required to fracture in J's case:

i) Both thinner bones and osteopenic bones are more susceptible to injury.

ii) Administration of Frusemide and chest physiotherapy is relevant to fractures.

55. In relation to timing issues:

i) There is a variation on how quickly fractures heal. The general consensus is that in the first 5 days fractures cannot be seen radiographically. Timing of fracture healing radiographically is itself an estimation.

ii) For radiologists to be able to detect osteopenia radiologically there needs to be a loss of mineralisation of 20-40%.

56. In relation to the more recent rib fracture, it is well accepted that rib fractures that show no bony healing are difficult to identify radiographically.

i) If there was a fracture present on 20th May 2017 when the scan was taken it might have been caused as early as 11 days prior without showing up on the scan.

ii) The time required to recover from osteopenia is unknown but with good nutrition osteopenia may have been alleviated and underlying bone fragility no longer visible when the skeletal survey was undertaken on 17th July 2017.

Dr Saggar Consultant in Clinical Genetics

57. Dr Saggar, well known to the Court, is a senior lecturer in medicine and with significant experience (35 years as a medical doctor) and subsequent specialisation (25 years in clinical genetics). Genetic testing shows that the twins were non-identical. At the time of his report Dr Saggar had not seen the report of Dr Cartlidge.

> " I cannot identify any unifying diagnosis, in J that would lead to an increased susceptibility to both bleeding and fracture"
>
> " I cannot identify any features that would explain the entirety of the clinical problems and injuries seen in J."

58. He agreed with the views of Dr Jayamohan and Dr Cartlidge. "I have no dissent at all. It is all quite reasonable. If there was any doubt genetic testing should be done." Deferring where appropriate, he gave his concurrent support to non-abusive injury. Whilst from a different perspective to the other experts, Dr Saggar, whom I have heard many times, brought a measured overview based on a lifetime's experience, and which in such a difficult case can be so valuable.

Dr Leadbetter, Consultant Forensic Pathologist

59. In some ways the necessity of Dr Leadbetter's instruction highlights the anxiety about some of the earlier evidence. However, having regard to the chasm between the clinical evidence and evidence of pathology, it was vital. His evidence was especially powerful because when he reported he had not seen the advices of Drs Cartlidge, Saggar and Watt. So, for example, whilst he could see no evidence of bone disease which would render the bones more susceptible to fracture, he raised whether a diagnosis of osteopenia could be excluded when the examined bone material subject to examination has been decalcified. He highlighted that prematurity and osteopenia are commonly linked. Given the history, together with the administration of Dalteparin, he was being cautious about attributing the injuries to trauma. He counselled caution also in relation to predisposition to fractures, ageing of fractures and increased excretion of minerals due to the administration of Frusemide. He deferred to Dr Cartlidge in relation to propensity to fractures, the force required to cause those fractures, and the difficulty in assessing healing rates in a child with osteopenia.

60. Overall, I was struck by Dr Leadbetter's contemplative, thoughtful and open-minded approach, frequently deferring to clinicians who have greater experience in many areas, the living, and on a day to day basis (for example as to the effects of Heparin), or the significance of raised venous pressure. Dr Leadbetter was an impressive witness. It is not without note that he brought together (but from the different discipline of pathology), the almost unified approach of the consultant clinicians.

The Parents

61. The mother gave brief but moving evidence. I thought her a comprehensively decent woman and mother, demonstrating that at all times she had the welfare of J, and her other children, at the centre of her life. The mother was a good witness. She portrayed a powerful picture of a loving, supportive family, both immediately, and more widely since the wider family all live nearby. They are a powerful force. More affectingly, I gained a real sense of J himself, and of their relationship over his very short life. Much of the detail now eluded her, which was no surprise, since we are talking of 2 years ago. Mr Twomey QC, in his closing submissions, has marshalled and prayed in aid the very considerable body of evidence from many sources supporting the mother's first rate capabilities. It should not be thought that I overlook that evidence in not setting it out here.

62. The father gave even briefer evidence. He is a straightforward man, I think devoted to the mother and the children, who has rather lost his way. Whilst it should not be thought that I have not had some anxiety about his recent drug taking, there is no evidence at all to connect that to the period when J died. Indeed, so far as I can tell, the father had almost nothing to do with J whose care was exclusively provided by the mother.

63. There was nothing in the parents' evidence that could assist the Court one way or another in this enquiry, except to record that they have been consistent in their account, that the mother has comprehensively put her children first (as demonstrated by all the many records and involvement with professional agencies), and as her immediate separation demonstrates, once she learned of the father's difficulty with drugs. In particular I was struck by the close attentive, empathetic bond the mother had with J, and recorded by everyone whom they encountered. The father's evidence was less

satisfactory in a number of areas, but evidential difficulties (of which there were a number), do not assist this enquiry without the foundation facts being established first. It is a danger which I think the local authority has misunderstood; the absence of a plausible explanation does not equate to a conclusion that the injuries are non-accidental – that after all would throw the burden of proof onto the parents.

Discussion

64. I bear in mind the rubric that today's medical certainty might be discarded by the next generation of experts, especially where, as here, medical experts disagree. I exercise caution in regard to opinions that a combination of conditions may be unusual, or even unique to J. Here the early witnesses in the immediate post mortem enquiry brought together the two areas of injury, concluding, as it were, that each supported the other. Whereas the later witnesses within these proceedings bring to bear wider and more uncertain considerations which are apparently absent, not just from the initial opinion, but some of the later evaluations made by them.

65. The medical evidence is only one part of my enquiry, but mindful of the submissions of Messrs Twomey QC and Storey QC, of the danger of the possible trap: it is not of course for the parents to prove anything, no burden rests on them, and whilst these are care proceedings, if the medical evidence of itself raises no issue, no bar, then there cannot in fact be any case for the parents to answer.

The approach of the medical witnesses

66. I have already recorded the thrust of each witness, all from appropriate specialisations, those who gave evidence appropriate to their professional standpoint. All are specialists within their own disciplines and largely respecting the frontier of their knowledge or expertise. There is a remarkable degree of agreement between the clinical experts. It is the evidence of the pathology which is in discord (although not Dr Leadbetter who from a different standpoint acknowledges the significance of the clinical opinion). Most were willing to acknowledge the perspectives of the others. I bear in mind the danger of looking at each piece of evidence in isolation, a linear approach, as opposed to looking at the wider canvass. That is not the same as consideration of each area separately, otherwise it becomes impossible to navigate the fog.

The fractured ribs

67. Bringing the evidence together, Dr Cartlidge said he would "stake money on J suffering from osteopenia". The rib fractures most likely were caused by compression force from whatever source.

68. Dr Watt advised that J had gracile (thinner) bones and signs of osteopenia on 3 May 2017, i.e. before his illness in May (and confirmed by x-ray on 20 May 2017). The commonest fractures which are seen are rib fractures and which occur most commonly in the first few weeks of the premature cohort. Frusemide and chest physiotherapy are both relevant. Thinner and osteopenic bones are more susceptible to injury.

69. Dr Cartlidge, agreeing with Dr Watt raised these factors:

    i) osteopenia at the beginning of May

ii) developing osteopenia (because of serious illness and the factors before).

iii) Low phosphate levels

iv) Forced clinical immobilisation (a contributory, but not causative factor)

v) Prolonged intravenous nutrition

vi) Frusemide – washing calcium from the body.

vii) Chest physiotherapy.

viii) If the fractures were caused by a parent, in the light of the above, they would not constitute a memorable event and the cry would be no different.

ix) Recovering from osteopenia takes "quite a long time" and caution is needed in ageing such fractures in osteopenic bones.

70. Dr Leadbetter advised care in relation to a) predisposition to fracture; b) the age of fractures; and c) excretion of minerals due to the administration of Frusemide. In conclusion he urged caution in attributing the injuries to trauma (given the administration of Dalteparin for 62 days prior to death). He additionally confirmed the link between prematurity and osteopenia.

The Subdural Bleed

71. Dr Keenan confirmed that Heparin, an anticoagulant, increases the risk of bleeding, extending to intracranial haemorrhaging. Being both premature and a very young baby increases the risk of intracranial bleeding. Treatment with Heparin with an existing blood disorder can present a much higher risk of bleeding. No such testing has been carried out.

72. Dr Jayamohan advised that the venous sinus thrombosis could have potentially caused the subdural bleed, the more so with the administration of Dalteparin. Indeed, Dalteparin alone might have been causative.

73. Having weighed the alternatives Dr Jayamohan concluded that the most likely explanation was the combination of venous sinus thrombosis blood flow from the head to the neck, caused by some element of engorgement of the dura and associated with Dalteparin. The combination of the two being sufficient to cause the small amount of bleeding seen by the pathologist.

74. He concurred with Drs Keenan and Cartlidge. Dr Jayamohan was clear to point out that the Court should not assume that any blood vessel needed to be breached first before the Dalteparin came into play as it is not known whether there is on-going but usually self-controlling leakage, aggravated by such a drug.

75. Importantly and understandably, heavily relied on (by Mr Storey QC in particular) there are no other signs of trauma which would normally be expected (in fact there are no other intracranial findings at all).

76. Dr Cartlidge, agreeing with Dr Jayamohan, concluded starkly that given the Thrombocytopenia, Heparin and raised venous pressure, that there was no need to look for other factors such as blood disorders.

77. Dr Leadbetter highlighted the use of Dalteparin, and the difficulty of dating. In particular the time taken for the cells to organise and thence to form a membrane. That time is unknown.

Conclusion

78. Bringing the central evidence together I find there were many unusual factors concerning J which are relevant:

    a) J was unwell and sick throughout his short life, requiring frequent emergency treatment in hospital.

    b) J died in a manner apparently unconnected with the identified injuries.

    c) J was born prematurely, 80% of bone mineralisation occurs in the third trimester of pregnancy.

    d) J had gracile bones and visible osteopenia from the x-rays taken on 3 May 2017.

    e) The position of the fractures would be unusual for a "shaking".

    f) J was prescribed Frusemide in hospital.

    g) J was prescribed Dalteparin from 10 May 2017 until his death.

    h) No visible contusions, bruises or marks were identified.

    i) There was no spinal or axonal damage, no damage to the eyes, no encephalopathy, no subarachnoid haemorrhaging, no "triad of injury".

    j) J had thrombocytopenia between 5 and 8 May 2017.

    k) J had suffered previous venous thrombosis

    l) J had prominent subarachnoid spaces.

    m) J had hypophosphatemia

79. The rib fractures. I conclude that it is overwhelmingly more likely that J had osteopenia before he became very unwell and was admitted to hospital. I conclude that it is more likely that his treatment with Dalteparin and Frusemide would have aggravated that condition. I conclude that each of J's fractures occurred at a point when he was suffering from gracile and/or osteopenic bones, and could have been caused unnoticed by anyone during normal handling. I am unable to date the age of the fractures.

80. The subdural bleed. I conclude that the subdural bleed was more likely to have been caused by a combination of raised venous pressure, and/or the administration of

Dalteparin. I conclude that I am unable to age the bleed because of the individual and combined effects of the effect of Dalteparin and the bleeding process, the difficulty of relying on a relatively small thin bleed as evidence of there being no earlier bleed at the date of the scan, and the commencement of membrane organisation, and the time required to give rise to this type of membrane formation. Even if I were wrong about the above, I could not rule out (and therefore the local authority fails to establish) a combination of blood disorder and Dalteparin and/or enlarged extra-axial spaces as causative.

81. Accordingly, I conclude that J's injuries arose from his very poor health, no doubt linked in part to his prematurity. It follows that I exonerate the parents from any culpable behaviour or responsibility. It follows that the threshold triggers are therefore not activated (including a failure to protect). Accordingly I discharge the proceedings.