Case No: B3/2014/3978 & A

Neutral Citation Number: [2016] EWCA Civ 1249
IN THE COURT OF APPEAL (CIVIL DIVISION)
ON APPEAL FROM High Court, Queen's Bench Division
Mr Justice Goss
HQ14X01568

Royal Courts of Justice
Strand, London, WC2A 2LL

Date: 13/12/2016

Before :

LORD JUSTICE JACKSON
LADY JUSTICE BLACK
and
LADY JUSTICE GLOSTER

- -

Between :

Baynham (a child and protected party by her Litigation Friend, Sarah Jane Baynham)	**Appellant/ Claimant**
- and -	
Royal Wolverhampton Hospitals NHS Trust	**Respondent /Defendant**

- -
- -

Satinder Hunjan QC & Dr Jonathan Punt (instructed by **Anthony Collins Solicitors LLP**) for the **Appellant**
Paul Rees QC (instructed by **Browne Jacobson Solicitors**) for the **Respondent**

Hearing dates: Tuesday 15th, Wednesday 16th, Thursday 17th and Friday 18th November 2016

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Judgment

Lord Justice Jackson:

1. This judgment is in five parts, namely:

Ground (ix) – Finding one pathology was against the weight of the evidence	Paragraphs 90 – 98
Ground (x) – Stable neonatal blood pressure	Paragraphs 99 – 101

Part 1 – Introduction

2. This is an appeal by a young woman suffering from cerebral palsy against a decision that a 30 minute delay in delivering her by Caesarean section did not cause or materially contribute to her disabilities. The principal issue in this appeal is whether the trial judge erred in assessing the expert evidence.

3. The claimant's mother was Ms Smith at the time of the events in issue. She has subsequently married the claimant's father. In order to avoid confusion, I shall refer to her as Ms Smith (the name in the medical records) rather than by her married name.

4. The defendant is the NHS Trust responsible for New Cross Hospital, Wolverhampton. That is the hospital which Ms Smith attended during the antenatal period and the birth.

5. It may be helpful if I define some of the terms which I shall use.

   A placental abruption occurs when the placenta peels away from the inner wall of the uterus, either partially or completely. That deprives the baby of oxygen and nutrients.

   Hypoxia means an insufficient supply of oxygen.

   Hypoxia-ischaemia means an insufficient supply of oxygen to an organ by reason of inadequate blood flow.

   Hypoperfusion means insufficient flow of blood to an organ.

   Reperfusion means the restoration of the blood flow to that organ.

   Reperfusion injury means injury caused to an organ when there is hypoperfusion, then reperfusion causing a surge of blood to the organ.

   Ventricles are interconnected cavities within the brain where cerebrospinal fluid is produced.

   An intraventricular haemorrhage ("IVH") is, as its name suggests, a haemorrhage within a ventricle. A placental abruption before birth may cause hypoxia-ischaemia leading to a reperfusion injury after birth, namely IVH.

There are four grades of IVH, with grade I being the least serious and grade IV being the most serious.

Bradycardia means that the heart is beating too slowly. If the foetal heart rate ("FHR") drops below about 120 beats per minute, that is bradycardia.

White matter means the layer of nerve fibres lying beneath the cerebral cortex.

Expansion of the ventricles consequent upon IVH may cause damage to the surrounding white matter. Such damage is also called periventricular leukomalacia ("PVL").

6. Finally, I shall use the abbreviation "CPR" for the Civil Procedure Rules.

7. After these introductory remarks I must now turn to the facts.

Part 2 – The Facts

8. On 22nd August 2000 Ms Smith was pregnant at approximately 28 weeks gestation. At about 9pm she felt pain in her stomach. She had a bath and went to rest in bed. At about 10.50pm she suffered extreme pain in her stomach. Her parents took her to hospital, while her partner looked after the children. Ms Smith arrived in hospital at 11.25pm.

9. Ms Durkin, a midwife, examined Ms Smith. She noted a foetal heart rate of 85 beats per minute. She appreciated that there was a serious problem. Ms Durkin called for medical assistance. She established intravenous access and sent for a full blood count, anticipating that the patient would probably need a Caesarean section.

10. Dr Ekpo, the registrar, attended at 11.30pm. After examining Ms Smith and taking a history, he diagnosed pre-term labour. He noted that there was foetal bradycardia and decided that an emergency Caesarean section was required.

11. Unfortunately Dr Ekpo's diagnosis was incorrect. The cause of Ms Smith's pain was placental abruption. Thus the need for a Caesarean section was even more urgent.

12. Having decided to carry out a Caesarean section, Dr Ekpo put in hand the necessary arrangements, but matters did not progress with proper expedition. Ms Smith arrived in the operating theatre at 00.18 hours on 23rd August. Attempts to administer a local anaesthetic were unsuccessful. Ms Smith was given a general anaesthetic at 00.32 hours. The claimant was born at 00.40 hours.

13. During the operation the placental abruption became apparent. There was a 200 ml retroplacental blood clot.

14. The paediatric registrar, Dr Lee, was present, assisted by the paediatric senior house officer. Dr Lee recorded that the claimant weighed 1.22 kg at birth and she was in a poor condition. Her Apgar scores were 4 at one minute, 8 at five minutes and 9 at ten minutes. The claimant was intubated at birth. Her chest expansion was good and equal. She took one to two minutes to pink up. At the age of ten minutes the claimant

was transferred to the neonatal unit with a diagnosis of "preterm 28/40 with respiratory distress syndrome".

15. Umbilical cord blood samples were analysed. The readings were as follows. In the arterial blood pH was 6.99, carbon dioxide 9.5 kPa, bicarbonate 17.3 mmol/L and base excess -16.3 mmol/L. In the venous blood pH was 7.03, carbon dioxide 9.3kPa, bicarbonate 20.7 mmol/L and base excess -14.2 mmol/L. Base excess denotes the amount of acid which needs to be added or subtracted in order to achieve a normal level. Taking matters shortly, this set of readings indicated that the baby was suffering from acidosis.

16. Over the next two days the claimant received intensive care in the neonatal unit and her blood gas levels were regularly monitored. The details of the claimant's treatment are not in dispute. Nor are her blood gas readings. A helpful chart setting out those readings at 30 different times between 1.50 am on the 23rd August and 4.50 pm on the 25th August is included in the claimant's expert evidence.

17. On the afternoon of the 25th August 2000 medical staff noted jerky movements of the claimant's limbs. An ultrasound scan was carried out. This revealed grade II IVHs, the right being larger than the left. The claimant was treated with phenobarbitone.

18. Unfortunately the IVHs increased in size. Their final state, as established on 29th August, was grade III on the left and grade IV on the right. Subsequent ultrasound scans showed cystic degeneration and evidence of ventricular dilatation.

19. Over the following months it became clear that the claimant had developmental problems. Further examination and tests revealed that she had asymmetric cerebral palsy, learning difficulties and epilepsy. Her parents took the view that those disabilities were attributable to negligence by the medical staff who attended at the time of the claimant's birth. Accordingly, Ms Smith acting as litigation friend for the claimant, commenced the present proceedings.

Part 3 – The present proceedings

20. By a claim form issued in the Birmingham District Registry, Queen's Bench Division of the High Court of Justice on 17th December 2010, the claimant claimed damages for personal injuries caused by the defendant's negligent delay in delivering her. The claimant contended that she ought to have been delivered by midnight on the night of 22nd-23rd August 2000 and that the subsequent period of delay made a material contribution to her injuries.

21. By its defence served on 8th July 2011 the defendant admitted that there had been 25 minutes of negligent delay, but denied that the delay had caused or contributed to the claimant's injuries. It was the defendant's case that the claimant would have suffered precisely the same disabilities, regardless of whether she was delivered at midnight or at 00.20 hours or at 00.40 hours. Thus the central issue at trial was one of causation. The question was whether the negligent period of delay (whether the judge held it to be 25 minutes or 40 minutes or some period in between) had caused or contributed to the claimant's injuries.

22. The action came on for trial before Mr Justice Goss ("the judge") in October 2014. The trial lasted for three weeks. The claimant called the following witnesses: Mr Robert Hammond, a consultant gynaecologist; Dr Wellesley Forbes, a consultant neuroradiologist; Professor Malcolm Levene, a consultant paediatrician/neonatologist; Dr Sian Harding, a specialist in neonatal paediatrics. The defendant called the following expert witnesses: Mr Derek Tuffnell, a consultant gynaecologist; Dr Neil Stoodley, a consultant neuroradiologist; Dr Janet Rennie, a consultant in neonatal medicine; Dr Colin Ferrie, a consultant paediatric neurologist.

23. It was common ground between Mr Hammond and Mr Tuffnell that the placental abruption probably started at about 9pm on the 22nd August 2000, when Ms Smith suffered abdominal pains. There was a significant extension of the abruption at around 10.50pm when Ms Smith's pain increased.

24. In relation to the foetal heart rate, Mr Hammond and Mr Tuffnell examined the various records and CTG traces. They agreed that the FHR probably remained constant at about 85 to 90 beats per minute between Ms Smith's admission at 23.25 hours and the claimant's birth at 00.40 hours.

25. The following further matters were agreed between the parties, as recorded by the judge:

> "a. Megan's disabilities are asymmetric quadriplegic cerebral palsy, with better right upper limb function, epilepsy and learning difficulties, having between moderate and severe intellectual impairment. She functions as a 6 or 7 year old. No improvement is anticipated;
>
> b. Her neuro-developmental impairments and/or neurological disabilities are due to the following abnormalities, which are to be seen on imaging: -
>
> i. Right sided porencephalic cyst (which explains Megan's left sided cerebral palsy)
>
> ii. Ventriculomegaly (which is a descriptive term referring to the abnormal enlargement of the ventricles)
>
> iii. White matter injury to both sides of the brain (which is why Megan has cognitive impairment).
>
> c. Megan suffered a parenchymal germinal matrix haemorrhage –intraventricular haemorrhage ('GMH-IVH') that was bilateral and probably occurred between birth and the first cranial ultrasound scan on 25th August 2000 as a hypoxia-reperfusion injury. The substance and tissues within the cerebral hemispheres are the brain parenchyma and is the relevant area in this case. What happened is that at the start of the hypoxic-ischaemic event – in this case the abruption – insufficient oxygen and blood flowed to her brain due to hypoperfusion. After she was born, sometime prior to the first ultrasound scans

on 25th August 2000, these nutrients were restored and acted as a spark, starting the reperfusion injury which accounted for the GMH-IVH. Loss of autoregulation leaves the brain unprotected against changes in blood pressure. The greater the change in cerebral blood flow in relation to a change in blood pressure, the more likely GMH-IVH is to develop – see Dr Rennie's textbook

d. It is agreed that the early and sequential cerebral ultrasound scans show the development of the bilateral IVHs, the right larger than the left, with haemorrhagic venous infarction by the time Megan was scanned on 25th August, when she was over 48 hours old, and that subsequent ultrasound scans show progressive ventricular enlargement and the development of a porencephalic cyst in the region of the haemorrhagic infarction and of cystic periventricular leukomalacia ('PVL' which is softening of the white matter around the ventricle – 'white matter damage') elsewhere. The cerebral white matter forms part of the cerebral hemispheres and is comprised of the nerve cell fibres that pass between the nerve cell bodies in the cerebral cortex and other nerve cells and structures in the brain and spinal cord. The subsequent CT and MRI scans show features consistent with a combination of PVL and superimposed porencephaly.

e. No abnormality was caused or contributed to by any insult in the pre-natal period or in the post natal period; it is the events of the intrapartum period that relate to causation and require critical examination."

26. The defendant's case was that the agreed mechanism set out in subparagraph (c) above, was the sole cause of the claimant's injuries. The placental abruption followed by loss of autoregulation of the foetal blood supply made it inevitable that reperfusion injury would occur after birth. That injury became inevitable well before midnight. Any negligent delay between midnight and 00.40 hours did not have any impact on the extent of the IVH or the extent of the white matter damage which was bound to occur after birth.

27. The claimant's case was that a second pathology was also operating as follows. The placental abruption caused hypoxia-ischaemia, which caused progressively increasing injury until 00.40 hours, when the claimant was born. The hypoxia-ischaemia during the period of negligent delay had two consequences:

    i) It caused a large part of the white matter damage which the claimant sustained.

    ii) It increased the severity of the reperfusion injury.

28. It is implicit in the claimant's analysis that some of the white matter damage occurred before birth as a result of hypoxia-ischaemia. It is implicit in the defendant's analysis that all of the white matter damage occurred after birth.

29. It was accepted on all sides that this was a complex case. The experts on both sides accepted that there were factors pointing in favour of both analyses.

30. The judge handed down his reserved judgment on 14th November 2014. He held that the period of negligent delay was 30 minutes. The claimant ought to have been delivered by 00.10 hours on 23rd August 2000. In relation to causation, the judge preferred the analysis advanced by the defendant's experts. Accordingly, he held that the period of negligent delay had not caused or contributed to the claimant's injuries. He therefore dismissed the claim.

31. In paragraphs 23 to 49 of his judgment, the judge set out a concise summary of the expert evidence on causation and the competing arguments. In paragraph 50, he set out his conclusion as follows:

> "Conclusion
>
> I have to decide this case to a standard of a balance of probabilities. What is known and agreed is that Megan was compromised by reason of the abruption. As a result, it is accepted that she lost her autoregulation and suffered a reperfusion injury after birth resulting in a complicated high grade GMH-IVH that was bilateral. That, in itself, could account for the totality of the damage and her disability. It is accepted by Dr Rennie and Dr Ferrie that it is possible that there was an additional ischaemic insult to the brain causing additional WMD [white matter damage], however, for the reasons they gave and which I accept, it is not established to the requisite standard that there probably was additional ischaemic insult in the 30 minutes prior to delivery. I reach this conclusion for the reasons set out above and, in particular, on the basis of the features of a stable FHR and the absence of evidence of deterioration of her condition over that last period, her condition upon delivery being significantly acidotic but having reasonable renal function, relative ease of resuscitation, stable neonatal blood pressure, together with the fact that Megan is not microcephalic and that her injury is markedly asymmetrical. All these features are consistent with the consequences of her complicated high grade GMH-IVH, which was bilateral and event, not time mediated. There is nothing of significance that can be relied upon as clear evidence of a separate, time related hypoxic ischaemic event prior to birth. A difficulty of the Claimant's case has been exemplified by its evolving nature, as is apparent from paragraphs 28 and 32-34 inclusive of this judgment. Tempting though it is to adopt what is, in one sense, a simplistic approach, namely the gas tap analogy, the evidence does not, in my judgment, establish to the requisite standard of a balance of probabilities that the delay in Megan's delivery of 30 minutes resulted in her suffering an additional ischaemic insult that caused further damage and disability to that which she would, unhappily, have sustained in any event."

32. The claimant (represented by her mother) was aggrieved by the judge's decision. Accordingly she appealed to the Court of Appeal.

Part 4 – The appeal to the Court of Appeal

33. By an appellant's notice filed on the 4th December 2014, the claimant appealed to the Court of Appeal on grounds which I would summarise as follows:

    i) The judge failed properly to assess the expert evidence. In particular, he failed to hold that the claimant's experts were more "consistent, authoritative, reliable and independent" than the defendant's experts.

    ii) The judge wrongly held that the claimant's case had been "evolving".

    iii) The judge failed to take account of changes in the defence case.

    iv) The judge failed to take account of inconsistencies in the expert evidence.

    v) The judge failed to take into account that the claimant's experts had greater expertise in the relation to the central issues than Dr Ferrie.

    vi) In reaching his conclusion the judge wrongly placed reliance on stable FHR, absence of microcephaly, absence of significant renal impairment and asymmetry.

    vii) The judge failed to appreciate the significance of the different types of IVH on the right and left sides.

    viii) The judge failed to appreciate the significance of the fact that reperfusion occurred on the 25th August, rather than earlier.

    ix) The judge's conclusion that there was one pathology rather than two pathologies causing white matter damage was against the weight of the evidence.

    x) The judge wrongly treated stable neonatal blood pressure as supporting the defendant's case.

34. In distilling this summary of the claimant's grounds of appeal, I have excluded paragraph 6(i) of the appellant's notice which seems to me to be a repetition of paragraph 1. I have excluded paragraphs 6 (iv) and (ix) which are no more than assertions that the judge came to a wrong decision on causation. I have treated the surviving parts of paragraph 6 of the appellant's notice as three separate grounds of appeal, as they relate to three separate topics. References in this judgment to grounds of appeal are references to the grounds as identified and numbered above.

35. On 13th February 2015 the claimant issued an application to adduce fresh evidence. The proposed fresh evidence relates to the fact that Dr Ferrie was in possession of cocaine on 17th December 2014. He pleaded guilty to that offence in the magistrates'

court and received a conditional discharge. Dr Ferrie subsequently faced the following charges before the Medical Practitioners Tribunal, namely:

i) being un-contactable while on-call on 17th December 2014 between 16.30 and 18.27;

ii) being convicted of possessing cocaine on 17th December 2014.

Dr Ferrie admitted the charges and expressed his deep regret. The tribunal ordered that his registration be suspended for four months. The claimant also sought to rely upon newspaper articles critical of Dr Ferrie concerning that incident and related matters.

36. The appeal to this court came on for hearing on 15th November 2016 and lasted for four days. We heard the application to adduce fresh evidence at the outset of the hearing. In a ruling given on the morning of 15th November, we said that we would give a decision on the fresh evidence at the end of the appeal, but in the meantime would look at the evidence *de bene esse*.

37. Under CPR Rule 52.11(2) the Court of Appeal has discretion to receive fresh evidence. The guidance given in *Ladd v Marshall* [1954] 1 WLR 1489 is relevant, but not binding. In the present case the claimant fails by a wide margin to satisfy the second limb of the *Ladd v Marshall* test. She cannot show that the fresh evidence would probably have an important influence on the result of the case. In my view, Dr Ferrie's misuse of cocaine on 17th December 2014 (some two months after the trial) is not relevant to the quality of his expert evidence in the present case or to any of the grounds of appeal. I would therefore refuse the fresh evidence application.

38. Having dealt with that preliminary matter, I must now address the grounds of appeal.

Part 5 – Decision on the grounds of appeal

Grounds (i) and (v). Assessment of expert witnesses

39. Mr Satinder Hunjan QC for the claimant contended at trial, as he contended before this court, that his expert witnesses were more consistent, authoritative, reliable and independent than the opposing witnesses, in particular Dr Ferrie. Mr Paul Rees QC for the defendant contended at trial, as he contended before this court, that all of the experts on both sides were witnesses of long experience with appropriate expertise to assist the court; there was nothing to choose between them in terms of calibre and expertise.

40. An appellant can of course argue in an appropriate case that the judge has misunderstood expert evidence or made findings which were not open to the court on the expert evidence. But when it comes to appraising the quality of the expert witnesses as expert witnesses, this really is within the province of the trial judge.

41. The judge addressed the rival contentions in paragraph 23 of his judgment as follows:

> "There is fundamental disagreement between Professor Malcolm Levene, Consultant Paediatrician/Neonatologist, and Dr Sian Harding, whose specialist field is neonatal paediatrics, on behalf of the Claimant, on the one hand, and Dr Janet Rennie, Consultant in Neonatal Medicine, and Dr Colin Ferrie, Consultant Paediatric Neurologist, on behalf of the Defendant, on the other. All are highly qualified, experienced and respected practitioners with considerable expertise in this field. Although some cross-examination of Dr Ferrie was directed to his alleged lack of day to day management of babies in the neo-natal period and therefore disadvantaged position compared to a neonatal paediatrician, I accept his expertise in the determination of causes of cerebral palsy, which he described as his "bread and butter" work, and qualification to give his opinion on the timing, causes and consequences of the IVHs. It has been submitted on behalf of the Claimant that, in themselves, the authoritative standing of Professor Levene and Dr Harding and the asserted defensive way in which Dr Rennie and Dr Ferrie gave their evidence, together with Dr Ferrie's expertise being in epilepsy and not the central neonatology issues in this case, should lead me to the conclusion that Professor Levene and Dr Harding's evidence as a whole should be preferred and the Defendant's experts' evidence should be rejected. I reject that approach; rather there has to be a calm and objective assessment of all the evidence and the respective opinions on that evidence in order to reach conclusions. No significant distinction can be drawn between them in their expertise or ability to assist in relation to the core issues in this case. Each has provided a report and signed an Agreed Note of a meeting held between them on 23rd April 2013 as well as giving evidence in the trial."

42. The judge heard the expert witnesses give oral evidence over many days and he made the assessment which I have just set out. In my view, absent some serious and obvious error on the part of the judge, this court cannot go behind the judge's assessment. Indeed I would go so far as to say that grounds of appeal (i) and (v) are based upon a misconception of the proper functions of this court.

43. Having said all that, in view of the gravity of the present case, I have read all the expert reports, the experts' CVs and the material parts of the transcripts of their evidence. They are all experts of high standing who gave their honest opinions about the issues arising in a complex and difficult case. I can see no basis for differing from the judge's assessment set out in paragraph 23 of his judgment.

44. I therefore reject the first and fifth grounds of appeal.

Ground (ii). The judge's comments about evolution of the claimant's case

45. As previously noted, in the course of paragraph 50 of his judgment, the judge commented:

> "A difficulty of the Claimant's case has been exemplified by its evolving nature, as is apparent from paragraphs 28 and 32-34 inclusive of this judgment."

Mr Hunjan contends that the claimant's case was not evolving. Therefore the judge took into account an erroneous matter in reaching his conclusion.

46. Mr Rees submits that the views of the experts on both sides evolved as the evidence emerged and they thought about the issues. That does not detract from the quality of the expert evidence of either party. Indeed it is to be expected in a complex case such as the present. Accordingly if and in so far as the judge treated the evolution of the claimant's case as a reason for rejecting it, Mr Rees does not support that line of reasoning. Mr Rees argues, however, that really the sentence quoted was no more than a passing observation which did not influence the judge's decision.

47. In addressing this ground of appeal, I must first consider whether the judge's observation is factually accurate. In my view it is, principally for four reasons:

i) In paragraph 7.4 of his initial report Professor Levene wrote:

> "In summary, I am unable to determine whether the placental abruption was the major factor in Megan developing PVL or whether the preceding intraventricular haemorrhage exacerbated or caused all her PVL."

He subsequently maintained positively that hypoxia-ischaemia prior to birth and consequent upon placental abruption caused the white matter damage.

ii) Dr Harding's oral evidence went some way beyond what she had put in her report, for example relying upon a paper by Professors Gunn and Bennett ("The Gunn Paper").

iii) In his report at paragraph 3.2 Professor Levene opined that the IVH occurred "sometime between birth and prior to the first scan at two days of age".

During the trial the claimant's case was that IVH occurred during resuscitation immediately after birth.

iv) Dr Forbes' evidence evolved as set out in the appendix to the defendant's closing submissions at trial.

48. In my view the fact that the thinking of the claimant's experts evolved during the course of the litigation should not be held against them. During the hearing Black LJ drew attention to a telling answer which Dr Harding gave on day seven of the trial at page 51 of the transcript:

> "So, of course, one's thinking about the case develops all the time as you listen to the evidence."

49. Let me now draw the threads together. The judge was correct in his observation that the claimant's case evolved. On the other hand that was not a factor which detracted from the claimant's expert evidence. In my view it would have been better if the judge

had not made the observation complained of. On the other hand, reading paragraph 50 as a whole, I do not think that the judge's unfortunate observation played any significant part in his decision. It was not one of the judge's identified reasons for holding that there was no "separate, time related hypoxic ischaemic event prior to birth".

50. I do not therefore accept that ground (ii) provides any basis for interfering with the judge's analysis of the expert evidence.

Ground (iii). Changes in defence case

51. The claimant's case on this ground rests upon inconsistencies between the defendant's pleaded case and the expert reports which the defendant subsequently served. In particular, the defence asserted that hypoxia-ischaemia resulting from the placental abruption caused white matter damage before birth. That position was directly contrary to the defendant's case at trial.

52. Mr Hunjan explained to the trial judge (day 1, pages 14 to 15) and he explained to this court (day 4 of the appeal) that he did not contend that the defendant was shut out from pursuing its case on the causation issue without a formal amendment of the defence. The claimant's contention was that the inconsistency between the defendant's original defence and the expert evidence which the defendant subsequently served and relied upon was a relevant factor, which the judge failed to take into account. The lack of consistency weakened the defendant's case. This was particularly so, since the defendant had instructed its experts before serving the defence.

53. Mr Hunjan put this point to Dr Rennie in cross-examination on day 9 on pages 22 to 24. Her response was that she did not agree with the defence; there was never a time when she believed that the claimant's injuries occurred in the manner pleaded in the defence; the barristers decided what to plead. Mr Hunjan did not put the defence to Dr Ferrie in cross-examination, but I surmise that Dr Ferrie's answers would have been similar.

54. Mr Hunjan complains that the judge did not mention in his judgment the inconsistency between the pleaded defence and the defendant's case at trial.

55. Mr Rees was in some difficulty in dealing with this point. Other counsel had drafted the defence and Mr Rees was instructed later. He pointed out that the case presented at trial was essentially the same as that foreshadowed by the expert reports. Throughout Mr Rees' involvement the defendant's case had been consistent. Possibly previous counsel misunderstood the expert evidence.

56. Mr Rees submitted that all the experts were giving evidence in good faith. The judge was deciding the case on the expert evidence adduced at trial. Earlier inconsistencies between the pleadings and the experts' reports were not relevant.

57. In my view, the inconsistency between the defence served in July 2011 and the defendant's expert reports dated September 2012 does not give rise to an inference that the defendant's experts were being dishonest. Mr Hunjan, for good reason, did not suggest that in cross-examination. The purport of Dr Rennie's evidence was that

counsel had pleaded the defence wrongly. Presumably Dr Ferrie would have said much the same, if he had been asked about the matter in cross-examination.

58. Since the judge chose to mention the evolution of the claimant's case, it would have been better if he had also referred to the evolution of the defendant's case. Nevertheless this omission does not undermine his analysis of the expert evidence. The judge was engaged upon the task of assessing the conflicting opinions of medical experts at trial. He was not making findings of primary fact about who said what or who did what. In my view it is clear that the judge did not gain assistance from the extent to which the views of the experts did or did not evolve since they were first instructed.

59. I conclude that the judge's failure to mention that inconsistency in his judgment is not a ground for disturbing the judge's conclusions on the issues between the experts.

Ground (iv). Inconsistencies in the defendant's expert evidence

60. Mr Hunjan raises a host of different points under this heading. I shall deal briefly with the main points.

61. First, he submits that the defendant's experts originally alleged that there had been a uterine artery spasm. That theory was discredited during the trial. It did not feature in the defendant's closing submissions. The judge erred in making no reference to this. Mr Rees ripostes that the presence or the absence of a uterine artery spasm is an irrelevant matter. It was simply a possible explanation of the undisputed fact that the placental abruption stabilised.

62. In her report Dr Rennie put the matter like this:

> "Mr Tuffnell is of the view that there was probably a significant abruption before Sarah Baynham was admitted, which then stabilized. This is a pattern of abruption which is well recognised, and in this case the alteration in placental function was sufficient to cause a fetal bradycardia which was present from at least 2325. A sudden significant abruption can be associated with spasm of the uterine arteries, followed by some recovery and stabilization, and this is quite a likely explanation for the situation in this case. I agree with the Particulars that once the CTG began the asphyxia was constant and it is not likely that the abruption was progressing between 2325 and 0040."

It will be noted that Dr Rennie's reference to a possible uterine spasm was tentative and not critical to her analysis.

63. Dr Harding in her oral evidence agreed that the abruption stabilised. She believed that this was by way of a tamponade: see day 7, page 15.

64. In cross-examination Mr Hunjan vigorously attacked Dr Rennie's suggestion of uterine spasm. Dr Rennie fairly acknowledged that it was speculation whether a uterine spasm occurred. She also stressed that this was not a matter of any

significance. The important point was that the placental abruption stabilised. That fact, which was critical to the defence, was common ground between the experts.

65. Having regard to the way the expert evidence developed, I am not surprised that the defendant made no reference to uterine spasm in its closing submissions. Likewise I am not surprised that the issue does not feature in the judgment. It was, perhaps, an interesting byway for the experts to ponder, but it was irrelevant to determining the causation issue. In paragraph 31(ii) of his skeleton argument Mr Hunjan submits that the occurrence of a uterine spasm was an essential element of the defence case. That proposition is plainly wrong.

66. Next, says Mr Hunjan, there was a significant change of the defendant's position in relation to hypercarbia. He says that during the trial the defendant's experts developed a novel theory. This was that at reintubation on the 25th August there was a surge of carbon dioxide and that triggered the reperfusion injury. This theory was not put in cross-examination to the claimant's experts. Mr Hunjan offered to recall his expert witnesses for that purpose, but Mr Rees declined the invitation.

67. Once again Mr Rees submits that this is a non-point. It was common ground between the experts that reperfusion occurred at some point between the 23rd and 25th August. Each of the experts had their own view as to precisely when the reperfusion occurred and what triggered it. Possibly the trigger was resuscitation at birth. Possibly the trigger was reintubation on the 24th or 25th August. Once again, this was no doubt an interesting byway, but it was not relevant to the court's decision on causation. The important points for present purposes were that (a) reperfusion occurred within two days of birth and (b) the reperfusion was a consequence of the earlier placental abruption.

68. In the course of argument we pressed Mr Hunjan as to why it mattered whether reperfusion occurred on 23rd or 25th August. He could not give any satisfactory answer beyond asserting that the judge needed to consider the mechanism by which the IVH or IVHs occurred.

69. In my view the judge cannot be criticised for failing to discuss the hypercarbia issue in his judgment. The judge rightly concentrated on those disagreements between the experts which were relevant to the decision on causation.

70. Mr Hunjan also took us through the evidence concerning the presence or absence of bilateral flares. This does not constitute an inconsistency by the defendant's experts. The judge carefully considered the evidence on this issue at paragraphs 27 to 29 of his judgment, but concluded that it did not lead anywhere. At paragraph 29 he said:

> "On any view, the evidence relating to bilateral flaring is mixed (the apparent conflict between the radiologist's report and the available images) and of limited value. There is no clear evidence of bilateral flaring; had there been, it would have assisted the Claimant's case. However, its absence, if such was the case, is not determinative. Accordingly, its presence or absence is an aspect to which I consider no significant weight can be attached."

71. I am unable to see how examination of the bilateral flares issue and similar matters is of any relevance to the present appeal.

72. None of the suggested inconsistencies in the defendant's expert evidence provide any basis for interfering with the judge's decision. I therefore reject the fourth ground of appeal.

Grounds (vi). Factors wrongly relied upon by the judge

73. Mr Hunjan submits that the judge erred in relying upon the stable FHR as a factor supporting the defendant's analysis of causation. He submits that even if the FHR remains stable, there could be a drop in the carotid blood flow to the brain. In support of that submission he relies upon figure 1 in the Gunn paper. This records the results of experiments on lambs, whose umbilical cords were clamped. Figure 1 shows that the carotid blood flow dropped more sharply than the FHR.

74. The Gunn paper was subject of much discussion at trial. The judge concluded in paragraph 41 of his judgment that the results on lambs whose umbilical cords were totally occluded could not be transposed to the claimant's situation. He was entitled to reach that conclusion.

75. The defendant's experts maintained that the stable FHR was a strong pointer against any white matter damage occurring before birth. In cross-examination on day 7 at page 63, Dr Harding very fairly admitted that there was some force in this point although she did not think it was determinative.

76. Although it is no part of the appellate function to scrutinise the evidence in detail, I can say that having done so in this case I agree with the judge's conclusion that the stable FHR was a factor supporting the defendant's case on causation.

77. I turn now to the absence of microcephaly. The judge treated this as another factor supporting the defendant's analysis. Mr Hunjan submits that this was wrong; the absence of microcephaly is irrelevant.

78. The short answer to this point is that there was a clear conflict between the expert witnesses on the significance of microcephaly or its absence. At day ten, page 48, Dr Ferrie said:

> "Microcephaly is usually defined as a head circumference less than the first centile. If the principal cause of the claimant's brain damage was hypoxic ischemia due to decreased cerebral perfusion in the run up to delivery, then the principal type of brain damage would be a loss of white matter. That would lead to almost inexorably microcephaly. Put simply, the head growth is going in the wrong direction. If there was of course progressive ventricular dilatation and the development of hydrocephalus, this is a complex situation here but if the principal type of brain damage had been antenatal or intrapartum hypoxic ischemic brain damage, I would have expected microcephaly to have developed."

79. The judge preferred the evidence of Dr Ferrie on this issue and he was entitled to do so.

80. I turn next to lack of renal impairment. The background to this issue is the claimant was acidotic at birth. The extent of her acidosis is apparent from the blood gas readings which I have set out in Part 2 above.

81. The judge held that despite the acidosis the claimant had not suffered white matter damage during the period before birth. One of the factors supporting his conclusion was the lack of any significant renal impairment.

82. The judge was quite entitled to reach that conclusion on the basis of Dr Rennie's and Dr Ferrie's evidence. Indeed even Professor Levene conceded that in the majority of cases where white matter damage occurred before birth there was some renal impairment: see day five, pages 58 and 59.

83. Finally, there is the issue of asymmetry. Dr Ferrie stated that if the claimant had suffered white matter damage before birth because of hypoxia-ischaemia, there would be relatively symmetrical cerebral palsy. The marked asymmetry of the claimant's condition was typical of cases where the cause was IVH. The judge accepted that evidence.

84. Mr Hunjan submits that the judge "failed to weigh up the expert witnesses" in reaching his conclusions on asymmetry. In my view this a hopeless point to advance on appeal. The rival views were deployed in the expert reports and the oral evidence. It was for the trial judge to decide which view he preferred. There is no basis for saying that the judge was wrong. I reject the sixth ground of appeal.

Ground (vii). Different IVHs on left and right

85. Mr Hunjan contends only the right ventricle sustained reperfusion injury. The blood in the left ventricle had trickled through via connecting passages. The fact that there was bilateral white matter damage means that it was not caused, or extensively caused, by the IVH on the right hand side. The judge failed to appreciate the significance of these matters.

86. Mr Rees' response is that there were bilateral IVHs and these caused bilateral white matter damage. He draws attention to paragraph five of the joint statement signed by Professer Levene, Dr Harding, Dr Rennie and Dr Ferrie, which reads:

> "We agree that the first ultrasound 25th August (day 3) showed bilateral IVH right more than left and the second ultrasound on the 29th August (day 7) showed a large parenchymal infarcation (the notes use the term "massive") on the right and IVH with ventricular dilation on the left. There was evidence of increasing ventricular indices (significant enlargement by 6th September) which stabilised by late September.
>
> We agree that Megan then developed asymmetric cerebral palsy, learning difficulties and epilepsy (see above)."

87. That paragraph makes it clear that there were bilateral IVHs. The judge was quite entitled on the evidence to find that both IVHs constituted reperfusion injuries. The asymmetric cerebral palsy reflects the fact that the IVH was worse on the right than on the left.

88. The judge's conclusions seem to me to fit neatly with the expert evidence. I therefore reject the seventh ground of appeal.

Ground (viii). Failure to appreciate the significance of reperfusion occurring on 25th August, rather than earlier

89. When and how the reperfusion commenced was a subject of understandable interest to the experts. It was therefore explored in the evidence. Nevertheless, for the reasons mentioned in relation to ground (iv), this was not an issue which affected the decision on causation. I therefore reject the eighth ground of appeal.

Ground (ix). Finding only one pathology was against the weight of the evidence

90. This ground invites the Court of Appeal to read through the bundle of expert reports, the medical literature and the transcripts of evidence in order to reach our own conclusion on the causation issue. In the ordinary way this court would not countenance such a course.

91. On the other hand, the appellant is a gravely disabled young person who contends that the judge has reached flawed conclusions, thereby depriving her of huge damages. In the circumstances, and since (most unusually) four days were allocated for the hearing of the appeal, we have read through all of the written evidence and most of the trial transcripts.

92. The short answer to this ground of appeal is that, having read through all that material, I can detect no error in the judge's weighing of the evidence. Unsurprisingly in a case of this complexity there were features pointing in both directions. There were legitimate differences of opinion between reputable and experienced experts. The judge assessed all of that material and reached a perfectly reasonable conclusion.

93. I will not extend this judgment further by trawling through all of the individual issues. But I should refer to a paper by Kayani, Walkinshaw and Preston, since Mr Hunjan attaches great importance to it. The paper was published in volume 110 of the International Journal of Obstetrics and Gynaecology (July 2003). The paper sets out the results of a study of 33 women with singleton pregnancies over 28 weeks gestation, who had placental abruptions. The overall conclusion was that a 'decision to delivery interval' of 20 minutes or less was associated with substantially reduced neonatal morbidity and mortality. This demonstrates, says Mr Hunjan, a clear relationship between length of delay and severity of injury.

94. The judge concluded that the Kayani paper was of very limited relevance to the present case. He gave two reasons. First, the paper relates to only 33 cases, of which only 7 were under 32 weeks gestation. Secondly, the paper stated that the authors were unable "to directly link the duration of the bradycardia to outcome".

95. In relation to this issue, Mr Hunjan invites us to consider Professor Levene's evidence-in-chief at day 5, pages 36 to 37 and his cross-examination at day 6, pages 17 to 18. In the first passage Professor Levene said that the Kayani paper was relevant to a hypothetical question posed by the judge. That hypothetical question was what would be the outcome if Ms Smith had been in hospital at the time of the placental abruption and the baby had been delivered 20 minutes later. In the second passage Professor Levene accepted that the small sample size made it impossible to draw firm conclusions. The judge joined in the questioning and suggested that the Kayani paper was of no great assistance.

96. Mr Rees submitted to this court that there were other shortcomings in the Kayani paper which make it of limited value. In particular, table 1 does not make it clear which cohorts the various babies listed fell into. There is no account of each abruption and its consequences. It is not possible to extrapolate conclusions concerning a baby at 28 weeks gestation. Furthermore, there is no discussion of the pathology or pathologies operating in those cases where the baby died or developed cerebral palsy.

97. In my view the judge was entitled to conclude that the Kayani paper was of very limited relevance to the circumstances of the present case.

98. Let me now stand back from the Kayani paper and look at the whole picture. I am quite unable to say that the judge reached decisions against the weight of the evidence. I therefore reject the ninth ground of appeal.

Ground (x). Stable neonatal blood pressure

99. The final substantive paragraph of the grounds of appeal states:

> "In reaching his conclusion, relied upon the "stable neonatal blood pressure" without any consideration or analysis of the relevance of the same and when, in fact, the stable neonatal blood pressure was supportive of the Appellant's case."

100. This issue received little attention during the hearing. Mr Hunjan has not put forward any coherent argument as to why the judge erred in treating the stable neonatal blood pressure as one factor (amongst many) which supported his conclusion on causation. I therefore reject the final ground of appeal.

101. Let me now draw the threads together. For the reasons set out above, I do not accept any of the grounds of appeal. The judge made findings on complex issues which were in dispute between the expert witnesses. All of those findings were well open to the judge on the evidence before him. I would therefore dismiss this appeal.

Lady Justice Gloster:

102. I agree.

Lady Justice Black:

103. I also agree.