Case No: HQ14X01568

Neutral Citation Number: [2014] EWHC 3780 (QB)
IN THE HIGH COURT OF JUSTICE
QUEEN'S BENCH DIVISION

Royal Courts of Justice
Strand, London, WC2A 2LL

Date: 14/11/2014

Before :

MR JUSTICE GOSS

- -

Between :

MEGAN NICOLE JANE BAYNHAM (A CHILD AND PROTECTED PARTY BY HER LITIGATION FRIEND, SARAH JANE BAYNHAM)	**Claimant**
- and -	
ROYAL WOLVERHAMPTON HOSPITALS NHS TRUST	**Defendant**

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Satinder Hunjan QC and Dr Jonathan Punt (instructed by **Anthony Collins Solicitors LLP**) for the **Claimant**
Paul Rees QC (instructed by **Browne Jacobson LLP**) for the **Defendant**

Hearing dates: 6th – 24th October 2014

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Judgment

MR JUSTICE GOSS:

Introduction

1. Megan Nicole Jane Baynham ('Megan/the Claimant') was born at 00.40 hours on 23rd August 2000 at the New Cross Hospital, Wolverhampton ('the hospital'). Her birth was premature, at 28 weeks gestation, and she was delivered by an emergency caesarean section. She weighed 1.22 kg and was in a poor condition in consequence of her mother, Sarah Jane Baynham (then Smith, having since married) ['mother'], having suffered a partial placental abruption (the premature separation of the placenta from the wall of the womb). She responded quickly to resuscitation. She was mechanically ventilated for 3 days and then on continuous positive airways pressure ventilation ['CPAP'] for 39 days. On the third day of her life, 25th August, she developed seizures; scans of her brain revealed a large parenchymal infarction on the right and intraventricular haemorrhage ['IVH'] with ventricular dilatation on the left. Megan developed asymmetric cerebral palsy, learning difficulties and epilepsy. By her mother and litigation friend, she now claims damages for personal injury and consequential losses arising out of the circumstances of her birth, alleging there was negligence by those acting on behalf of the hospital in their treatment of her and her mother in delaying delivery by at least 40 minutes and that this delay caused significant additional damage to her brain, thereby resulting in a more severe form of cerebral palsy and greater cognitive impairment than would otherwise have been the case. The hospital admits that delivery was delayed by around 25 minutes due to fault in its systems but disputes that there was an additional period of some 15 minutes attributable to fault on the part of the examining Registrar, Dr Ekpo. However, it is the hospital's case that none of the time differences had any impact on the outcome and that the IVH suffered by Megan was a reperfusion injury that was solely primed by the placental abruption, which injury, its extent and consequences were inevitably going to be sustained as a result of the abruption whether Megan was born at 00.00 hours or, as she was, at 00.40 hours or at some time in between. Although the case was listed for trial on both liability and quantum, during the course of the hearing it was agreed by both parties that the issue of liability should be tried first.

Background

2. This was Mrs Sarah Jane Baynham's fourth pregnancy. She has two sons, Jack Stafford, who was born prematurely at 34 weeks on 7/1/96, and Kallum Stafford, who was born prematurely at 32 weeks on 19/10/98 with abnormalities relating to his bones and a growth disorder, rhizomelic limb shortening, though this is irrelevant in relation to Megan's case or condition. A third pregnancy had ended in miscarriage in 1998.

3. The antenatal history to 22nd August is not controversial. Fetal ultrasound scans at around 16, 18 and 20 weeks revealed no abnormality and the pregnancy was proceeding unremarkably. She was admitted to the delivery suite on 28 June at around 21+1 weeks gestation due to 'tightenings', she was noted to have 'backache and period type pains'. She was discharged the following day. On 20th July 2000 at 24+2 weeks she was seen in the antenatal clinic and, apart from 'abdo pains' which were considered to be possible Braxton Hicks, was otherwise very well. On 11th August at 27+3 weeks she was admitted to the delivery suite with abdominal pains; they, or irregular tightenings, continued intermittently over the ensuing days until she

was discharged on 14th August. She had no further abdominal pains until 22nd August.

4. In August 2000, Mrs Baynham (then Smith) was living with Adam Baynham and the children in a flat on the 13th floor of a block of flats close to her parents, David and Margaret Smith.

Issues

5. Although there was a good deal of agreed evidence, the following central issues require resolution: -
    1. The timings and circumstances of the events of 22nd – 23rd August 2000 up to the moment of delivery, the findings in relation to which will determine the issue as to when Megan should have been born had there not been a negligent delay.
    2. Whether any damage to Megan's brain and its consequences are attributable to the period of negligent delay in performing the caesarean section and, if so, the extent thereof.

Events of 22nd – 23rd August 2000

6. There are a number of sources of direct evidence as to the events of 22nd – 23rd August 2000 leading up to Megan's birth. Mrs Sarah Baynham made a detailed witness statement dated 19th March 2012, which she supplemented in evidence on 7th October 2014. Her mother, Margaret Smith, made a witness statement dated 19th March 2012 and also gave evidence on 7th October 2014. On behalf of the Defendant, Midwife Lindsey Durkin made a witness statement dated 17th June 2012 and gave evidence on 7th October 2014, and Dr Ekpo E Ekpo, the locum Obstetric Registrar at the hospital who carried out the examination of Sarah Baynham and performed the emergency caesarean section made a statement dated 14th March 2012 and gave evidence on 7th and 8th October 2014. Both had the benefit of contemporaneous medical records. All witnesses have the handicap of trying to recall events that occurred many years ago. Memories fade. I am quite satisfied that each witness has done his or her best to recall events as they remember them. Mrs Sarah Baynham has the additional difficulty of having been in severe pain and, as she said in her statement, lapsing in and out of consciousness in the time leading up to the caesarean section. Her knowledge and memory of events is, therefore, incomplete.

7. It was whilst watching television on the evening of Tuesday 22nd August 2000 that she started to notice pain in her stomach. She thought it was probably Braxton Hicks again and, following earlier advice she had been given, decided to have a bath. She cannot remember what the time was. According to Dr Ekpo's note, he was told by her that the pains started about 9 pm. There is no reason to doubt that timing. After her bath, she lay in bed and the Braxton Hicks/contraction type of pain continued. A short while later she "had extreme pain in her stomach". She had never experienced this type of pain in the past. In her oral evidence she described it as coming on with no warning; it was constant and excruciating. In her statement she said "whilst the pain was coming from my stomach, it was affecting my whole body. My tummy was tightening beyond a contraction all over and around my back etc." Adam was still in the living room; she had to crawl on all fours to get his attention. Once alerted, Adam telephoned her parents, who drove to their daughter's flat. She said the time interval between the onset of the pain and the telephone call was less than 5 minutes. Both Mrs Baynham and Mrs Smith agreed it would take approximately 5 minutes for them

to get to the flat; Mrs Baynham said it would then take less than 5 minutes for them to get back to the car; her mother initially said it would have been about 3-4 minutes, but later agreed it would have been about 5 minutes. The car journey time to the hospital, both agreed, was about 15 minutes. At the hospital, given the delivery suite being two floors above reception and the need for Mrs Baynham to be taken in a wheelchair, Mrs Baynham agreed that she saw the midwife approximately 5 minutes later. Midwife Lindsey Durkin has recorded in the hospital contemporaneous record that Mrs Baynham was admitted at 23.25 hours. The continuous CTG trace commenced at 23.22 hours.

8. Accordingly, on the essential timings given, the pains first began at about 21.00 hours and, on the agreed evidence of the Consultant Obstetricians and Gynaecologists, were probably the onset of the abruption, which extended with further separation at the time of the extreme pain, which occurred at about 22.50 hours or shortly thereafter. I am satisfied that was the case. It is agreed that there was probably no further separation after 22.50 hours.

9. Midwife Durkin, unsurprisingly, had no memory of the events at all. She relies upon her note in the Contemporaneous Record. That reads –

    23.25 Admitted. Distressed ++
    Abdo pain – abdomen tense attempted to ausculate FH. Unable hear.
    Muffle sounds – Sonicaid @ 85 bpm.
    Mat pulse 80-90
    ° pv loss. Dr summoned. IVI commenced.
    Bloods for FBC + G+S
    By M/W Barnett

10. In evidence, she explained that the CTG was extremely difficult to interpret. She called for medical assistance straight away, established intravenous access and sent for a full blood count and save of serum anticipating that a caesarean section was likely to be required.

11. She confirmed she was present when the Registrar, Dr Ekpo, examined Mrs Baynham. He, too, has no recollection of the case and relies on his notes in the Contemporaneous Record which he said, were made after the examination and possibly after the caesarean section had been performed. The entry immediately following Dr Ekpo's note is timed at 23.50. Dr Ekpo said a gap may have been left; if so, there was a gap of over three-quarters of a page and he filled it neatly and completely. The note reads –

    23.30 • G4 Para 2 +1 28/40 prev preterm x 2
    At 32 & 34 weeks
    • c/o • Period-like pains started about 21.00 hrs tonight
    • Now intermittent, labour pains occurring 1 in 5 mins
    • No urinary nor bowel symptoms
    • No SROM
    O/E • Seems to be in labour
    • Contracting 1 in 5 mins
    • Vital signs stable
    • Chest clear clinically

• Abdomen: SFH: 28 cm
FHR: 88/m
Re-checked w scan → seems rather slow
VE: Cx posterior, uneefaced, OS closed

Δ : Preterm labour
Fetal Bradycardia
- steroids
- SCBA informed
- D/W Mr Murphy, for CS in view of fetal bradycardia
- For penicillin G protocol. GBS carrier

12. Dr Ekpo concluded Mrs Baynham was in preterm labour. This was a misdiagnosis. He reached this conclusion on the basis of her history, being told the description of the pains, like labour pains, that had started at 21.00 hours occurring one in every five minutes and checking for himself by touching her abdomen, and the other information he was given and findings on examination, but because there was a fetal bradycardia, a slowing of the FHR, which in Megan's case should have been between 120/min and 160/min, whereas it was 88/min - he felt he needed to perform an emergency caesarean section. He discussed the case with the on-call Consultant Obstetrician, who agreed. Arrangements were commenced. The paediatrician and theatre team had been called by 23.55 hours. Mrs Baynham arrived in the operating theatre at 00.18 hours. The attempt to administer a local spinal anaesthetic, commenced at 00.20 hours, was ineffective so she was given a general anaesthetic at 00.32 hours and Megan was delivered at 00.40 hours and treated by the attending paediatricians. It was noted by Dr Ekpo that there was a 200ml retroplacental fresh blood clot. Mrs Baynham had suffered a concealed placental abruption.

<u>Issues and findings in relation to the delivery of Megan</u>

13. It is accepted on behalf of the hospital that there was an unacceptable delay between the decision to carry out a caesarean section (23.45 hours) and its being undertaken (00.40 hours). It is conceded it should have been effected within no longer than half an hour, so by 00.15 hours; accordingly, there was 25 minutes of negligent delay. It is the Claimant's case that the decision to perform a caesarean section should have been made within a minute of Dr Ekpo commencing his examination of Mrs Baynham at 23.30 hours, so there was a further negligent delay of almost 15 minutes. This is disputed by the Defendant. It is the Defendant's first position that, in the light of the Agreed Note of the Expert Meeting held between the Neonatologists and Paediatric Neurologist held on 23rd April 2013 question 7, this is not an issue that requires resolution. Question 7 was

> "What effect can be attributed to the alleged failure to deliver the Claimant before 24.00 hours on 22 August?"

The recorded answer was

> "See our answer to question 6. There is no difference in our opinion if the time of delivery had been advanced by just 15 minutes."

The answer to question 6 by the Claimant's experts was that if delivery had occurred by 00.15 hours, the Claimant would have had a less severe cognitive deficit and less

severe cerebral palsy by reason of the white matter damage having been less. Having heard the evidence of Professor Levene and Dr Harding in relation to this answer and their basic opinions as to the ongoing effects of the abruption, I do not consider it to have been intended to be nor should it be interpreted as acceptance of there being no relevance of any additional delay. Accordingly, there needs to be determination of the period of the negligent delay.

14. On this issue I heard evidence from Mr Robert Hammond, a Consultant Gynaecologist and formerly a Consultant Obstetrician and Gynaecologist, on behalf of the Claimant and from Dr Ekpo and Mr Derek Tuffnell, a Consultant Obstetrician and Gynaecologist, on behalf of the Defendant. Both the Consultants have great expertise and clinical experience.

15. Placental abruptions are rare. They are concealed (i.e. there is no vaginal discharge of blood) in about 30-35% of cases. Diagnosis is dependent upon a consideration of the history, presenting symptoms and the results of examination. Mr Hammond considers Dr Ekpo, having received as succinctly as possible the information from Midwife Durkin, who, it is agreed by all, acted in an exemplary fashion, and with a distressed patient with severe abdominal pain, should have summoned emergency assistance within a minute, then checked the fetal heart rate and mobilised staff for an emergency caesarean section to be carried out. Mr Hammond could and did not accept that the pain would have been anything other than continuous or that there were sensations of ongoing contractions. He did accept that if Dr Ekpo's findings were correctly recorded, he could not criticise his decision making.

16. Midwife Durkin's note makes no direct reference to the severity or continuity of the abdominal pain. There seems to be no obvious reason for Dr Ekpo to have recorded the history from Mrs Baynham as he did if that is not what she said. She recalls being asked questions and vaguely remembers answering but cannot say what answers she gave. Accordingly, I find that Dr Ekpo has probably recorded in his note what she did say. Mr Tuffnell's evidence was that the clinical features of any woman with placental abruption can be very variable and there can be contractions though they are usually very frequent, but not necessarily so. Mr Hammond's evidence was that the pain may change in intensity but is constant and that it is implausible that Dr Ekpo detected contractions. There was a spur in the CTG trace at 23.43 hours but no reliable conclusions can be drawn from it.

17. It is accepted that it was for Dr Ekpo, as the clinician, to make the diagnosis and the decision; failure to carry out this decision carefully would leave him open to justifiable criticism. In due course, he diagnosed preterm labour and bradycardia. The latter made this a medical emergency, requiring urgent delivery by caesarean section. He accepted that fetal bradycardia is an obstetric emergency. Whether he should have diagnosed placental abruption is a secondary issue, which it is not necessary for me to resolve. However, I accept the evidence of Mr Tuffnell that it should have been considered as the likely diagnosis at the conclusion of the examination but that it was not a conclusion to which he should have come within a minute or two and that Mr Hammond is using a retrospective analysis. It was appropriate and necessary for him to determine the FHR, and Mr Hammond accepted it was good care to use a Pinard stethoscope and good practice to check for any possible confusion with the maternal heart rate using an ultrasound scan, as Dr Ekpo

did, as well as consider the history and make an assessment on the information he was given and the results of his examination. Mr Tuffnell considers the reasonable time range to complete such tasks is 5-10 minutes. He put the maximum time at 15 minutes. In all the circumstances, given the findings, and applying the principles of the well established test of determining medical negligence according to practice accepted as proper by a responsible body of professional medical opinion (*Bolam v. Friern Hospital Management Committee* [1957] 2 All E R 118) I accept the evidence of Mr Tuffnell that the decision to carry out a caesarean section could and should not have been made within a minute and that, whatever the diagnosis should have been, it should have been made within 10 minutes, in other words by no later than 23.40 hours. Accordingly, Megan should have been delivered by 00.10 hours at the latest and I find there was negligent delay in her delivery of 30 minutes. It is upon this finding that I proceed to consider the issue of causation.

The consequences of the abruption

18. It is important to identify and emphasise what is not in dispute: -
    a. Both Mr Hammond and Mr Tuffnell agree that it is likely that the placental abruption started earlier in the evening with a significant extension when the pain increased, which I have found to be at around 22.50 hours. This is likely to have led to significant hypoxia, and that was clear at the point of admission because there was a fetal bradycardia. It is not suggested that there was any deterioration in the placental circulation after admission.
    b. It is agreed that Megan's FHR probably remained constant between admission and delivery at between 85 and 90. There is no actual record of the FHR after about 23.56 hours, something which was highlighted during the course of the evidence of the Neonatologists. It is recorded in the contemporaneous notes that the FHH c/c Scanner was heard with a scanner at 00.06 hours.

19. Mr Hammond considers that Megan was probably exposed to roughly the same degree of hypoxia throughout the period between admission and delivery and that "the progression of the hypoxic insult would have been broadly linear", a view considered to be "unhelpful" by all of the other experts, including Professor Levene and Dr Harding on behalf of the Claimant, in ascertaining when the fetal injury would have occurred and is one which, therefore, I reject. I am satisfied that the major part of the abruption having occurred at around 22.50 hours, it remained stable thereafter.

20. There is also agreement that: -
    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.

21. There is a fundamental dispute as to the effects of the period of hypoxia-ischaemia consequent upon the negligent delay in delivering Megan. It is the Claimant's case that the duration of the avoidable period of hypoxia-ischaemia
    i. Had an effect on the severity of the reperfusion injury and the extent of the resulting haemorrhagic venous infarction and thereby made a material contribution to Megan's cerebral palsy. It is asserted that the persistent ventriculomegaly to be seen on the neuro-imaging is not the result of the hydrocephalus but represents an enlargement of the lateral ventricles as a consequence of the loss of cerebral white matter due to PVL; and
    ii. Was responsible for an increase in the extent/severity of the bilateral PVL (white matter damage) and thereby increased the severity of her cerebral palsy and her neuropsychological impairments.

22. The Defendant's case is that the abruption was the priming event for the IVH, which was a reperfusion injury, and was responsible for all the consequential injury and that the delay in delivery had no material effect on the outcome. The Defendant contends that the extension of the period of hypoxia-ischaemia consequent upon the abruption cannot be shown, on a balance of probabilities, to have had any impact on the extent of the GMH-IVH or the PVL or, therefore, on the extent of the cerebral palsy or

cognitive deficits. It is to this issue of causation that the great majority of the evidence in this case has been directed. It is accepted by all the experts that this is a very complex case and there is no single feature which can be said to be determinative of the central issue.

The expert evidence on causation

23. 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.

24. I have also been provided with reports, including a signed Joint Note following a telephone conference on 3rd April 2013, and heard evidence from Consultant Neuroradiologists, Dr W St C Forbes, on behalf of the Claimant, and Dr N Stoodley, on behalf of the Defendant. Again, each is highly qualified and a very experienced expert. I deal first with the neuroradiological evidence.

25. Ultrasound scans of Megan's brain were taken on the 25th, 29th and 30th days of August 2000. It is agreed that the results of such scans are very dependant on the setting of the machine and the 'live' observations of the operator, which are recorded in the hospital notes. The few images that are available are helpful in that they reveal certain features but are not determinative of what was to be seen: ultrasound scans being poorly specific, can miss abnormalities and can show abnormalities that are not really there, according to Professor Levene. The Radiologist on 25th August noted

> "Bilateral Grade II IVHs, right larger than left. See photographs. Fresh blood in right temporal pole. Some dilatation of both temporal poles, right greater than left. Marked bilateral flaring with left (*it is accepted this is an error and it should be right*) basal ganglia appearing bright. Average ventricular width 10mm".

26. The same Radiologist (as is apparent from the handwriting) noted on 29th August that extension of the right-sided haemorrhage into the parenchyma consistent with a Grade IV IVH with some PVL on the right side and a Grade III IVH on the left with dilatation of the left lateral ventricle and enlargement of the temporal horn. Increased brightness of the basal ganglia was also noted.

27. In relation to the report of "bilateral flaring", although all of the expert witnesses confirm an increase in echodensity on the right, none considers there is clear evidence of increased echodensity or flaring on the left. Professor Levene expressed himself as 'neutral' but, in any event, he attached no significance to this aspect, asserting whether there was or was not increased echo-density/flaring is not of relevance; there obviously was white matter damage as a consequence of the hypoxic-ischaemic event. Dr Harding was "not sure" there was a bilateral flare but explained that if the white matter damage was diffuse you would not expect to see flares in the first few weeks, so, in a case such as Megan's flares would be unusual. Dr Rennie, on the other hand, whilst accepting that ultrasound scanning is not 100% reliable and poorly sensitive for the detection of white matter damage presumed to occur in the early stages, considered that the although the absence of evidence on the scan images is not a strong feature, it is a piece of evidence which, on the balance of probabilities, would have been present if there had been an insult to the brain sufficient to cause severe white matter damage that evolved into a loss of tissue.

28. Both Neuroradiologists agree that, based purely on the imaging findings, it is not possible to give an opinion as to whether the brain injury was caused by the effects of the hypoxia-ischemia and/or secondary to the extensive IVHs that Megan sustained. Dr Forbes, in his report of 12th June 2012, referred to there being evidence of a mild PVL. In his evidence on 9th October, he varied this to mild to moderate. He also referred to evidence of bilateral flaring to be seen in the first ultrasound scan but accepted that there was no marked bilateral flaring to be seen in that or later scans, an opinion with which Dr Stoodley agreed. He accepted he would expect to see some bilateral flaring in images if there had been a pre-delivery insult causing damage but said that in a significant number of cases, albeit the minority, no flaring is to be seen. He considered that the absence of flaring is neutral, an opinion shared by Professor Levene who said the ultrasound scans are not helpful in determining whether there was or was not PVL. There can be no doubt there was PVL; it is to be seen on later images. The question is whether it was a consequence of hypoxia-ischaemia or IVHs or both. Dr Forbes considers hypoxia-ischaemia to be a significant contributor to the brain injury. Dr Stoodley confirms there are competing causes for Megan's PVL and he cannot differentiate between them: all the abnormalities are explicable on the basis of being secondary to the IVHs. However, on the basis of the imaging appearances alone, the possibility that a period of hypoperfusion contributed to the PVL cannot be excluded.

29. 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.

30. It is to the evidence of the Paediatricians/Neonatologists one has to turn for answers. During the course of the evidence, intermittent reference was made to extracts from textbooks, including those of Professor Levene and Dr Rennie, and studies/articles. All passages sought to be relied upon have to be considered in their context. In relation to studies, none was directly addressing the particular circumstances obtaining in this case; reliance was sought to be placed on basic principles/physiology rather than specific findings. Accordingly, I only refer to any such material where I consider it of relevance or not to a particular issue.

31. The starting point is those matters upon which there is agreement set out in paragraph 20 of this Judgment. The Agreed Note of the Expert Meeting, recorded that

> "We agree that Megan sustained IVH with parenchymal venous infarction… This probably occurred between birth and the first cranial ultrasound scan on the 25th August 2000, and then "extended" by the 29th. We agree that this was due to "hypoxia – reperfusion" caused by the placental abruption, as the priming injury".

32. Professor Levene explained that the placental abruption severely compromised Megan prior to birth, causing bradycardia and severe metabolic acidosis at birth. At paragraph 7.4 of his original report of July 2012, he stated: -

> "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."

33. Later in that report he stated that if Megan had avoided the last [40] minutes of asphyxial insult prior to birth he felt that the size of the IVH would have been smaller and he felt that "on the balance of probabilities she would not have developed parenchymal extension which is the cause of her dense left-sided hemiplegia and therefore would have been much more independently mobile" and "she would have escaped severe intellectual impairment". In his letter of 19th February 2014 in response to CPR 35 Questions posed by the Defendant he stated

> "I am unable to determine the degree of saving of intellectual capacity by earlier delivery at 00.00 or 00.15. Her cognitive impairment is as a result of the total duration of the asphyxia insult prior to delivery and reducing the timing by 25-40 minutes represents an indivisible benefit in her cognitive impairment."

In a disclosed letter of same date to his Instructing Solicitor, he explained this opinion

> "… the white matter injury is dependent on the duration and severity of the hypoxic ischaemic event. The number of white cells that are irreversibly damaged is dependent in part on the duration of the asphyxial result and in turn the majority of Megan's cognitive impairment has occurred as a result of the white matter injury. Therefore if the duration

> of the prenatal asphyxia insult could have been reduced by 25-40 minutes then the damage to her white matter would have been less severe which would have resulted in preservation of more white matter and consequently some preservation of her cognitive ability. It is however, impossible to determine how much less cognitive disability would have sustained if she had been born 25-40 minutes earlier than she actually was."

34. In the Neurological Joint Statement, in response to questions 5-8, he developed a series of divisible scenarios to produce an outcome whereby the PVL mediated hypoxic- ischaemic damage is largely attributed to the period of delay prior to delivery.

35. In evidence, Professor Levene accepted that, from the moment of the abruption, Megan lost autoregulation of her cardiac output and was thereby rendered vulnerable: it was what happened thereafter that caused the ischaemic injury. By the time of her birth, she was significantly compromised and it could not be said when her brain became irreversibly damaged. He asserted the Defendant's experts' approach was selective and ignored the significant period of bradycardia, which must have led to low blood pressure, and that white matter does not sustain hypoxia-ischaemia very well at all. He said it was intuitive that if the time of the episode was reduced significantly – by 40 or 25 minutes – she would have suffered much less brain damage and there was an increasing acceleration towards the end of the period. He accepted there was a good deal of speculation; he said on both sides, but that his evidence was based on "firm principles" and the Defendant's case ignored the bradycardia and the acidosis.

36. In her report Dr Harding stated

> "The fetal response to hypoxia includes a fetal bradycardia. In this case, there is evidence of a prolonged fetal bradycardia prior to delivery. Although preterm infants are able to tolerate a longer exposure to an asphyxial insult than term infants, prolonged exposure will result in increasing acidosis, hypotension and hypoperfusion of the brain.
>
> The evidence shows that Megan was severely acidotic at birth. That degree of acidosis would have accrued over a period of time. I think that it is likely that the injury to Megan's developing white matter was compounded during the period from her mother's admission to labour ward (*sic*) to the time her circulation was restored after delivery.
>
> …
>
> It is possible that free radical damage from iron contributed to some of her periventricular white matter injury.
>
> …

It is my opinion that Megan's extensive white matter damage is, on the balance of probabilities, most likely to have occurred as a consequence of the prolonged hypoxic-ischaemic insult that resulted from the placental abruption.

…

If Megan had been delivered by 00:00 hrs on 23/8/2000, she would have been exposed to a hypoxic insult of 65 minutes rather than 105 minutes. She would not have completely avoided the priming event for GMH-IVH, but it is my professional opinion that had the hypoxic-ischaemic insult have been reduced by 40 minutes that there would have been a material reduction in the degree of white matter injury and Megan may not have developed the large right-sided parenchymal infarction. In my professional opinion, it is likely that if she had been delivered by midnight, there would have been a significant improvement in her motor function, cognitive function and ability to communicate, but I am unable to determine the precise contribution of the delay to her overall outcome."

37. Dr Rennie, in her report of September 2012, stated

"In my opinion, hypoxic ischaemia associated with a placental abruption causing fetal bradycardia and acidosis was the major factor behind Megan's preterm brain injury. The IVH/periventricular haemorrhagic infarction which is responsible for her damage probably occurred on the 25th of August… A post natal evolution as result of ischaemic reperfusion is quite well described, and in my opinion this is the most likely mechanism of damage in Megan's case. This postnatal occurrence is associated with ischaemia/reperfusion combined with loss of auto regulation, fluctuation and often there is a triggering event (as in this case with the reintubation).

Ischaemia reperfusion is not a function of time as suggested. A reduction in the damaging minutes would not have avoided the necessity to resuscitate Megan, nor would it have avoided the associated postnatal events associated with her RDS which would have occurred in any case… If Megan had been delivered at 0000 or 0015 she would still have been acidotic, she would still have been exposed to the initial phase of the abruption followed by a fetal bradycardia lasting at least 35 minutes (from 2325) and probably longer, and she would still have required resuscitation and ventilation. In my view the same sequence of events would have occurred in exactly the same way. A reduction of 25 or 40 minutes would not have made any difference to any of the factors in play here."

38. Dr Ferrie, in his report, concluded

> "In my opinion Megan's condition can be wholly explained on the basis of her sustaining bilateral IVHs in the newborn period.
>
> …
>
> On the basis of the evidence currently available I am of the opinion that the severity of the hypoxic ischaemic insult occurring in association with the placental abruption is likely to have been at its maximum shortly after the abruption occurred, that is prior to her mother's admission to hospital. It follows that even if Megan had been delivered earlier than she was… this would not have prevented her developing the brain damage responsible for her current condition."

39. In his evidence he, as did the other expert witnesses, identified the reasons for his opinion, to which I now turn.

Discussion – the expert evidence on causation

40. I have found that, had there not been negligence on the part of the hospital, Megan should/would have been born by 00.10 hours; there was an extension of the period during which she was being exposed to the hypoxic-ischaemic episode consequent upon the abruption of 30 minutes. The issue, therefore, that I have to resolve is whether that period had any material affect on Megan's ultimate condition. In order to do so, it is necessary to examine the competing arguments in the light of the agreed facts and available objective evidence. I preface this discussion with the comment that I cannot and will not rehearse all the fine detail of the respective arguments. Rather, I will concentrate on what I consider to be the core matters.

41. It is accepted that, although there is no direct evidence as to Megan's heart rate prior to her admission to hospital or from about 23.56 hours, it is likely it was between 80-90/min from admission to birth. There can be no measure of fetal blood pressure until after birth. The Claimant's experts point to the data within the Article by Gunn & Bennet "Fetal hypoxia insults and patterns of brain injury: Insights from animal models" published in *Clin Perinatal* 2009 September; 36(3):579-593. That study traced the results of the effects on lambs of differing gestation where there was complete occlusion of the umbilical cord. In the case of lambs of the closest equivalent to Megan in terms of gestation (0.6), they show a declining heart rate by almost 50% within a few minutes of the occlusion then a steady decline to about 28% of the pre-occlusion rate by 30 minutes. Over the same period, after an initial period of hypertension, hypotension soon developed and progressed steadily so that the mean arterial pressure was reduced to about 28% of its pre-occlusion level after 30 minutes. The important distinction in Megan's case is that although she was undergoing a period of hypoxia-ischaemia, there was not a total loss of blood flow and her heart rate was probably remaining steady. The complete occlusion of the umbilical cord of the lambs showed a drop in FHR with a drop in arterial pressure. It is, therefore,

inappropriate to transpose these findings unquestioningly to Megan's situation. Dr Harding accepted that her thesis was based on there being additional damaging hypoperfusion in the relevant period, which I have found to be 30 minutes, immediately prior to birth. She accepted that her case was more likely if Megan's heart rate fell but she could not say she did not come to harm with a stable heart rate. She emphasised, as did Professor Levene, that the longer the period of the insult the worse the outcome; the latter described it as 'intuitive' that if the time was reduced significantly – 25 or 40 minutes – there would have been much less brain damage. They pointed to and relied upon the paper of Kayani, Walkinshaw and Preston "Pregnancy outcome in severe placental abruption" *BJOG; an International Journal of obstetrics and Gynaecology* July 2003, Vol. 110, pp 679-683. The objective of the article was 'To determine the relationship between decision to delivery interval and perinatal outcome in severe placental abruption'. The conclusion was that "In this small study of severe placental abruption complicated by fetal bradycardia, a decision to delivery interval of 20 minutes or less was associated with substantially reduced neonatal morbidity and mortality". However, it is noteworthy that the study related to only 33 cases, of which only 7 were under 32 weeks gestation, so Professor Levene accepted no statistical significance could be attached to it, and that the authors stated "We are unable to demonstrate a relationship between the duration of bradycardia and outcome – which could be due to small numbers". Accordingly, whilst having regard to the paper, it is of very limited relevance to the circumstances obtaining in this case.

42. It is common ground that white matter damage is associated with IVHs. Kuban et al in the article 'White matter disorders of prematurity: Association with intraventricular haemorrhage and ventriculomegaly' published in The Journal of Paediatrics May 1999 p 539 state "The association of IVH with WMD with what is presumed to be periventricular haemorrhage infarction is purported to be strong. In contrast, periventricular leukomalacia is presumed to be most often caused by hypoxic-ischaemic insults despite a paucity of evidence to support that view" (p 540). Dr Harding , on page 23 of her report, reproduced a Figure under the narrative

> "It is postulated that haemorrhage into the germinal matrix impedes the flow of blood from the medullary veins (that drain the cerebral white matter) into the terminal vein. This impairment of blood flow leads to an area of venous infarction, which may be haemorrhagic."

Dr Rennie opined that the size and location of a venous infarction is a function of the part of the venous system that is obstructed and she did not understand the logic of it being time related. Dr Ferrie agreed with this position. Chapter 20 of Professor Levene's textbook, which addresses neonatal intracranial haemorrhage, in the section headed Intraparenchymal Haemorrhage, at pp 411-412, refers to unilateral intraparenchymal haemorrhage and studies relating thereto, but does not refer to a temporal link or identify anything of direct relevance to the features in this case. There is no clear documented support for the time related approach of the Claimant's expert witnesses. Such literature as there is relating to this issue does not support a time related connection between IVHs and consequential white matter damage – see A Spinello et al. 'Severity of abruptio placentae and neurodevelopmental outcome in low birth weight infants' published in Early Human development 35 (1993) pp 45-54 and Volpé 'Neurobiology of Periventricular Leukomalacia in the Premature Infant' published in Paediatric Research Vol. 50 No. 5 2001 p 553. Moreover, in his

evidence, Professor Levene accepted that he could not point to any literature that informs in relation to the size of an IVH being time related and that there was no data. His proposition was the duration of an insult was like a gas tap; the longer it is on the greater the reperfusion.

43. A not inconsiderable amount of questioning related to the timing of the reperfusion injury/IVH. This was not an area of controversy at the time of the Joint Meeting, as referred to above. The positions of Dr Rennie and Dr Ferrie were set out in their respective reports; in short, both considered it probable that the bleeding occurred as a result of reintubation on 25th August. The bleeding was observed to be "fresh blood" on the first ultrasound scan that afternoon. Professor Levene and Dr Harding did not identify a timing in their reports but in their evidence timed it at, or, in the case of Dr Harding, developing from, birth/resuscitation. The development of the IVH in subsequent imaging is consistent with the bleeding having occurred recently ("fresh blood"). Although it is not determinative of the basis for my conclusion in this case, on this issue, despite the criticisms made of Drs Rennie and Ferrie in relation to their evidence under cross-examination, I am satisfied that, consistent with such documented research evidence as there is within the Kluckow and Evans paper 'Low superior vena cava flow and intraventricular haemorrhage in preterm infants' Arch Si Child Fetal Neonatal Ed 2000;82 F188-F194 in relation to complicated high grade haemorrhages, this was a later IVH. In relation to the Kluckow and Evans paper, Professor Levene accepted that that paper addressed the issue of blood flows after birth and did not address a temporal relationship between events and blood flows prior to birth, and that he could not prove a temporal relationship from it.

44. A feature upon which Professor Levene and Dr Harding place considerable emphasis was the level of acidosis. Dr Harding described Megan as being "severely acidotic" and that this could only be because she had been significantly compromised and therefore produced metabolic acid. The pH and base excess levels were well outside the normal ranges. Dr Harding stated that the levels recorded indicated brain damage was already occurring. Dr Ferrie starkly disagreed and asserted that acidosis does not mean that brain damage is being or has been caused and there is no correlation between them; there will be some babies, he said, with much more severe acidosis than Megan had who have no brain damage. His view was that the whole condition of the baby at delivery has to be considered rather than one finding. Dr Rennie stated that base deficit and oxygen levels do not correlate with brain injury in the form of causing severe bilateral white matter damage. Professor Levene accepted acidosis in itself does not cause damage but it is a marker of the amount of damage; some babies, he said can cope remarkably well with acidosis and not be brain damaged. In answer to the proposition that if there had been a permanently damaging acidosis in Megan's case, it would have taken longer to clear, he responded "perhaps".

45. Dr Rennie referred to the full umbilical cord blood gas results, which reflected the situation at birth. Although the acidosis indicated the fetus was experiencing a reduction in input, the umbilical oxygen venous result, which was within the normal range but on the low side, indicated that there was sufficient oxygen to enable the fetus to pick up oxygen and carry it back in the umbilical venous blood at the time of delivery. Dr Harding's response was that Mrs Baynham was pre-oxygenated; Dr Rennie said such pre-oxygenation as she received would have had a very small impact on the umbilical venous blood. The issue of calcium bicarbonate level was referred to

by Dr Rennie, which indicated an oxygen debt had been built up but that the bicarbonate buffer had not been exhausted. Dr Ferrie expressed a similar view but earlier in his evidence expressed the qualifications that this was not an area in which he claimed specific expertise; he had not referred to it in his report because he had not considered it important at that time and he did not think that, of itself, the level of bicarbonate was going to assist the court in resolving this case. I attach no significance to the bicarbonate level.

46. However, both Dr Rennie and Dr Ferrie did attach significance to the absence of evidence of any renal impairment. It was agreed that Megan's renal function was reasonable for a pre-term baby ventilated for RDS. Dr Rennie knew of no reason why the kidneys should be saved in preference to the brain of a compromised baby. Professor Levene explained renal impairment is not necessarily a consequence of pre-natal injury; however, he would expect such impairment in more than 50% of cases. Accordingly the absence of any renal impairment in this case, although not, of course, determinative in itself, is a feature of relevance, being something that would be expected in a majority of cases of pre-natal injury.

47. It is accepted by both Dr Rennie and Dr Ferrie that it is possible that Megan suffered PVL as a result of the hypoxic-ischaemia, however, as well as relying on her condition at birth, the totality of the results, including the renal function, and the relative ease of resuscitation, they also point to the fact that all of Megan's injury is entirely explicable as being as a result of the GMH-IVH that was bilateral. Megan's condition is exactly what they would expect as a consequence of the complex Grade IV and Grade III IVHs. The release of free iron and free radicals as a consequence of the breakdown of the blood can be a cause of WMD, as is well documented in the literature. Dr Ferrie considered if Megan had suffered WMD as a result of a hypoxic antenatal insult then he would have expected relatively symmetrical cerebral palsy. The marked asymmetry was wholly typical of what he would expect as a consequence of IVH and not typical of a case of PVL.

48. Moreover, Dr Rennie and Dr Ferrie refer to the absence of encephalopathy (diffuse brain dysfunction) and microcephaly (head circumference under the 3rd centile) as evidence supporting their view that the damage and functional deficits are a consequence of the IVHs. Dr Ferrie considered the absence of encephalopathy as weak evidence, which was consistent with the evidence of Professor Levene, who said it would almost to be expected that in a baby of 29 weeks you would not expect to see signs of encephalopathy. Dr Ferrie attached greater weight to the absence of microcephaly, stating that, in his opinion, if the Claimant's case was correct, then Megan would now have be microcephalic.

49. It was accepted by all the expert witnesses that there were many possibilities in this case. I accept and respect the good faith and firmly held opinions of both sides and each has sought to identify features to which significance can be attached in support of their respective cases. That is an inevitability in a case such as this where there are many possibilities and no single determinative feature.

Conclusion

50. 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, 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.

51. The claim is, therefore, dismissed.

52. I pay tribute to Megan and her family and the obvious love and care they have for each other, recognising that this judgment will come as a great disappointment to them. Similarly, I gratefully acknowledge the enormous assistance that has been provided to me by all the medical expert witnesses and the legal teams in this difficult and complex case.