Case No: HQ17C04199

IN THE HIGH COURT OF JUSTICE
QUEEN'S BENCH DIVISION

Neutral Citation: [2019] EWHC 3542 (QB)

Royal Courts of Justice
Strand, London, WC2A 2LL

Date: Wednesday, 18 December 2019

Before:

THE HON. MRS JUSTICE LAMBERT DBE

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Between:

AB (By Her Litigation Friend DE)	**Claimant**
- and -	
East Lancashire Hospitals NHS Trust	**Defendant**

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Mr William Featherby QC and Mr Rachit Buch (instructed by **Addies Solicitors**) for the **Claimant**
Mr John Whitting QC (instructed by **Hempsons**) for the **Defendant**

Hearing dates: 02-06 and 10 December 2019

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JUDGMENT

Mrs Justice Lambert :

1. The Claimant is an eight-and-a-half-year-old girl who suffers from a right sided hemiplegic cerebral palsy associated with developmental delay affecting her motor and cognitive function. The cause of her condition is a perinatal arterial ischaemic stroke affecting the left basal ganglia region of her brain. She brings this action, by her mother and litigation friend, for damages for personal injury and consequential financial loss on the basis that, but for the negligence of the Defendant's medical staff in her mother's antenatal care, the stroke would have been avoided. She benefits from an order protecting her anonymity and that of her mother, I therefore refer to the Claimant's mother in this judgment by way of her cipher, "DE." The trial before me addresses liability only with quantum to be considered at a separate trial in the future if necessary.

2. Mr William Featherby QC and Mr Rachit Buch acted on behalf of the Claimant and Mr Whitting QC on behalf of the Defendant. I am grateful to them all for their help.

Chronology

3. The claim on breach of duty raises two main points for my determination: the first is whether a finding of a retroplacental haematoma at 16+ weeks gestation is a significant finding mandating senior obstetric review, consultant-led antenatal care and serial growth scans throughout the pregnancy; the second concerns the interpretation of cardiotocograph traces ("CTG traces") of the pre-term fetal heart as opposed to the term fetus. I set out below the uncontentious factual framework of the events giving rise to the claim drawn mainly from the hospital records. It covers the key events in DE's antenatal care which are relevant to breach of duty and the early stages of the Claimant's neonatal care where the timing of the Claimant's first abnormal neurological signs is relevant to her case on causation.

Antenatal Care

4. DE was aged 24 when she fell pregnant with the Claimant. It was her first pregnancy. On 10 September 2010 she was referred by her general practitioner for midwifery led care. Her antenatal care was uneventful until 22 November 2010 when, at 16+ weeks' gestation, she self-referred to the Early Pregnancy Assessment Unit ("the EPAU") at Burnley General Hospital with a complaint of feeling faint. This is the first of the two occasions upon which it is alleged that the Defendant's management was negligent.

5. The EPAU was a small unit at the hospital, separated by two floors from the main obstetric department. It was run by nursing (rather than midwifery) staff and in general there would be two staff nurses on duty together with an obstetrician. On the 22 November 2010, Ms Sharon Burridge, a band six nurse, and Dr Ben Dadebo a locum specialist registrar in obstetrics and gynaecology were on duty.

6. DE was seen first by Ms Burridge who recorded the history which she took from DE on a pro-forma document. So far as is relevant, she noted her observations (blood pressure, respiration and temperature), the problem which had led to the attendance at the EPAU (fainting) and further information including that DE smoked 5 cigarettes per day; that she was 16+ weeks pregnant; that there were no complaints of bleeding or pain or of clots or tissue being passed.

7. DE was then seen by Dr Ben Dadebo who worked from an adjacent room. He performed an ultrasound scan which confirmed the presence of a singleton pregnancy, a foetal heart and foetal movements. He also recorded seeing a "*small area of retroplacental hypoechogenicity at the fundus highly suggestive of a haematoma*." Under the section below his findings, he noted his impression which was of a "*single live fetus with retroplacental haematoma*." Ms Burridge then completed a discharge note which recorded: "*Felt faint, attended EAU. Single live fetus with retroplacental haematoma noted. Keep 20-week scan. No bleeding noted*".

8. On either the 23 or 24 November 2010 (the date remains in doubt but is of no importance) there was a discussion in the Unit involving Nurse Gillibrand, Nurse Singh and Dr Dwivedi, which led to the decision that, because of DE's rhesus negative blood status, she should be given anti D immunoglobulin which was then administered on 25 November 2010.

9. The pregnancy resumed its uneventful course. DE was seen for the purpose of an anomaly scan at 20 weeks' gestation on 17 December 2010 when, according to the record, all of the structures which were visualised appeared to be normal including the placenta.

10. DE once again self-referred to hospital, this time attending the birth unit (the labour ward) on 24 February 2011 when she was 30 weeks plus 2 days gestation. This is the second occasion upon which it is alleged that the antenatal care was negligent. DE's complaints on this occasion were of episodic right sided abdominal pain and pins and needles affecting her arms.

11. On arrival she was triaged by Midwife Halliwell at 12.40 who recorded: "*self-referral to birth unit with history of abdo[minal] pain r[ight] sided. At present not there. Has had numbness of the arms and legs on occasions. On admission, B[lood]P[ressure] 110/55, pulse 90, temp[erature] 36.8, [oxygen] sat[uration]s 99%. Urine nitrates and MSSU [mid-stream urine specimen] sent*". Fetal well-being was assessed by way of a cardiotocograph ("CTG"). The trace ran from 12.37 to 13.45 when it was stopped for the purpose of obstetric review. At 13.00 Midwife Halliwell noted a possible "*loss of contact*" on the CTG paper trace "*as baby very active*".

12. The Claimant's mother was then reviewed by Dr Vivian Lee, the on-call obstetric ST2. So far as is relevant her contemporaneous note reads:

    "*Presenting Complaint:*
    1. *Right sided abdominal pain since 23.00 last night*
    2. *Numbness and tingling in legs and arms/on and off for days.*

    Right sided stitch like pain 23.00 last night
    Lasted for minutes -> pain = resolved now
    Numbness and tingling while driving
    Normal fetal movements. No P[er] V[aginum] loss
    [No] weakness/dysphagia/dysphasia noted by family
    [No] nausea/headaches
    On examination alert and pain free"

13. Dr Lee performed an abdominal examination, noting that it was soft and non-tender with no palpable tightenings. She recorded a normal neurological examination and the absence of clinical evidence of a DVT. In the light of these findings, she requested a repeat CTG trace to be run (because of loss of contact) with a provisional plan to discharge DE and for her to be reassessed at the antenatal clinic in two weeks' time. In the margin of the note, she recorded: "*D/W SpR Johnson and agree.*"

14. The CTG trace was run again from 14.32 to 15.30 and the trace reviewed by Dr Lee. She recorded a note, timed at 15.00, that the repeat CTG had been reviewed and was reassuring and her plan that DE should be discharged home for reassessment in 2 weeks' time at the antenatal clinic. Both the accuracy of the timing of this note, and the timing of a discussion which Dr Lee had with the Specialist Registrar (Dr Johnson Amu) are contentious. Before leaving the unit, DE was once again reviewed by Midwife Halliwell who recorded the normal haemoglobin result and her advice to DE that she should see the midwife for antenatal care in 2 weeks but that she should ring the unit if she had any concerns.

15. DE was seen again at regular intervals at the antenatal clinic. The pregnancy appeared to be progressing well. The pregnancy was allowed to continue post term (her due date was 2 May 2011).

The Delivery

16. At 06.00 on 16 May 2011 (at 42 weeks' gestation) DE was admitted to hospital with contractions. CTG monitoring produced a trace which was suspicious with reduced beat to beat variability and some early decelerations. A fetal blood sample was taken which showed a mild metabolic acidosis and at 13.12 a decision was made that the Claimant should be delivered by emergency caesarean section.

17. The Claimant was delivered at 13.23 on 16 May 2011. The placenta was noted to be gritty and calcified but was not sent for histology. A large amount of meconium was present. The Claimant appeared flat with no spontaneous breaths and a slow heart rate. The Claimant was immediately transferred to the resuscitaire and was resuscitated effectively by suction (of meconium from the airway) and inflation breaths. Her APGAR scores were 5 at one minute, 6 at 5 minutes and 10 at 10 minutes. Her birthweight was 2535 g on the 0.4th centile for gestation based on the RCPCH growth chart for term girls. Her head circumference was on the 2nd centile. Her length was on the 50th centile. She was therefore very small for gestational age. She was admitted to the neonatal ward.

Neonatal Course

18. At 14.00, the Claimant was noted to be in respiratory distress. Sub-costal recession was observed.

19. At 16.40 at the ward round, the Claimant was noted to be tachypnoeic (breathing very rapidly).

20. On 17 May 2011 at 10.05 (at 21 hours of age) at the ward round, it was noted that the Claimant was pink, well perfused and alert with a good sucking reflex/rooting reflex, bilateral symmetrical Moro reflex was present. Mild hypertonia to both upper and lower limbs was noted.

21. Later on 17 May, at 20.20 (at around 31 hours of age), the Claimant had a desaturation down to 60% and was noted to have turned bluish. Tactile stimulation was given with no effect and so facial oxygen was administered. At 21.20 there was a further desaturation.

22. On 18 May 2011 at 01.15 (at around 36 hours of age) the Claimant had a further desaturation down to 67% with, what was described in the notes as "*mouthing*" which took several seconds to normalise. At 04.00, the Claimant had further profound desaturations associated with colour change (she became blue with a staring look).

23. At 06.00, at review, the following concerns were noted: "*has had two episodes of profound desaturations, hypertonia, deviation of eyes. Dropping to 40s to 60s associated with increased heart rate, mouthing, starey look, deviation of eyes to one side and hypertonic. Also noted to be cyanosed with one episode. Desaturations not related to feeds, 1st episode lasted for a few seconds, 2nd episode for approximately 5 minutes, not improved with tactile stimulation, needed facial oxygen. After the episodes, baby was noted to be hypertonic by nurses*".

24. At 07.00 the clinical impression was that the Claimant had probably had "*fit like episodes ?cause ?infection ?Metabolic*"

25. At 07.55 a cranial ultrasound was performed "*with difficult views*", but on subsequent review at the consultant cranial ultrasound meeting (on 24 May 2011) it was recorded that no abnormality could be seen.

26. Neuroimaging of the Claimant's brain (on 13 March 2012) at 10 months of age demonstrated a range of findings which were all considered to be secondary to destruction to the left basal ganglia region of the brain and associated white matter destruction.

The Issues:

27. The Claimant's pleaded case is set out in the Re-Re-Amended Particulars of Claim, the reason for the various iterations of the document being the sequential/delayed disclosure by the Defendant of documents, principally those relating to treatment at the EPAU.

28. The claim on breach of duty focusses upon two dates: 22 November 2010 and 24 February 2011. On both of these occasions it is alleged that there were findings which mandated senior obstetric review: on 22 November 2010, the finding on ultrasound scanning of a retroplacental haematoma and, on 24 February 2011, the Claimant's presentation (abdominal pain) in conjunction with abnormalities in the fetal heart trace and the earlier scan finding.

29. Central to the case on breach of duty is the assertion that a retroplacental haematoma "*can be a sign of placental abruption*" (the pleaded claim) or that a retroplacental haematoma is a placental abruption (the claim as it emerged at trial). An abruption is a separation of the placenta from the uterine wall and it is the Claimant's case that, at any gestation, it can cause a reduction in placental function and thereby lead to growth restriction due to the reduction in the surface area for gas and nutrient exchange between mother and fetus. Given this risk, the Claimant's case is that the finding of the haematoma, even at 16 + weeks, is highly significant and the only reasonable obstetric management on 22 November 2010 was to transfer DE to consultant care for consultant led antenatal care.

30. It is the Claimant's case that, having suffered one placental abruption on 22 November 2010, she went on to suffer a further abruption on 24 February 2011 and that this explains the abnormalities in the fetal heart trace on that occasion: the reduction in beat to beat variability, the fetal tachycardia, the absence of any accelerations and the presence of decelerations. Given the fact of the earlier abruption and those abnormalities on the trace, this was a second opportunity to refer DE for consultant-led antenatal care. The failure to do so was negligent.

31. There are a number of links to the Claimant's chain of factual causation. The first link is that any reasonably competent obstetric consultant would have, in either November 2010 or February 2011, instigated consultant led antenatal care and serial growth scans given the risk of growth retardation associated with the scan finding. At either 30 weeks (the pleaded claim) or

at or around 34 weeks (as the Claimant's expert accepted at trial) the fetus would have crossed below the 10th centile and would in consequence have been categorised as "small for gestational age" or "SGA." Further ultrasound scanning to monitor the Claimant's size and the volume of liquor and Doppler tests would (or should) have been undertaken. The pregnancy would not have been permitted to continue to term and labour would have been induced at or around 38 weeks.

32. Delivery at 38 weeks would have avoided the stroke. Although the Re-Re-Amended Particulars of Claim do not elaborate upon the cause of the stroke, at trial the Claimant's case was that the stroke was caused by a small fragment of blood clot or debris from the calcified and gritty placenta which crossed through into the fetal circulation and lodged in the left middle cerebral artery causing occlusion and death (or infarct) of the area of the brain served by that vessel. The Claimant's case is that the stroke occurred (or at least the embolisation of the placental debris had occurred) before the umbilical cord was cut.

33. There are, unusually, very few points of contact between the Claimant and Defendant.

34. The Defendant denies breach of duty and both factual and medical causation:

    a. It denies that the finding of the retroplacental clot in November 2010 was a significant finding. Such a finding is common in the early weeks of pregnancy and a small haematoma which is not associated with any vaginal bleeding does not indicate, let alone mandate, senior obstetric review or ongoing care or serial growth scans. Routine midwifery antenatal care, which would include a detailed anomaly scan at 20 weeks, would spot the continued presence or enlargement of the haematoma, was standard (and reasonable) practice. As might be expected, at 20 weeks the haematoma had resolved. Even if a consultant had reviewed the scan findings, he or she would not have done more than refer DE back to standard midwifery-led antenatal care.
    b. It denies that the CTG trace in February 2011 demonstrated abnormal features, bearing in mind that the trace was not of a term fetus. Whether viewed in isolation or in conjunction with the complaint of episodic pain, those features were not significant and did not indicate or mandate consultant care and serial growth scanning. Even if a consultant had reviewed the CTG and the relevant history, no further action would have been taken. In particular, he or she would not have organised serial growth scans.
    c. The Defence takes issue with the Claimant's case on the causation of the stroke; it avers that it is likely that the stroke was suffered in the neonatal period and not pre-delivery relying in part upon the Claimant's condition at delivery and the timing of the Claimant's abnormal neurology at around 31 hours of age.
    d. Perhaps the only relevant points of contact between the parties' respective cases is the Defendant's admission that, had the Claimant's intrauterine growth retardation been diagnosed, then early, preterm, delivery would have been indicated. It is also admitted that, if the medical cause of the stroke is proven to be embolisation of placental debris, then delivery at 38 weeks would probably have avoided the stroke. All other elements of the Claimant's case (on breach and causation) remain in issue.

The Factual Evidence:

35. I set out below the factual evidence given at trial which is relevant to the issues above. I heard evidence from DE and from all of the midwives and doctors who were involved in the relevant care in November 2010 and in February 2011.

36. Three points need to be made at this stage: first, save for DE who had a partial recollection of events in February 2011, none of the witnesses had any memory of the events in question and all were therefore reconstructing events on the basis of the records and usual practice. This is hardly surprising given the lapse of time since the events took place. Second, although I heard evidence from members of the midwifery team, following cross examination of the Claimant's expert in midwifery, Ms Abigail Osbourne, the claim against the midwives in negligence was withdrawn. It follows therefore that the midwifery factual evidence is now of only limited importance and, as Mr Featherby put it in his closing submissions, relevant only to the extent that it sheds light upon the management of Dr Dadebo and Dr Lee. Third, two witnesses, Ms Fiona Clarke and Ms Fiona Hamer, both consultant obstetricians employed by the Trust at the relevant times in November 2010 and February 2011, gave evidence concerning the action which, hypothetically, they would have taken had a referral been made to them in November 2010 (Ms Clarke) or in February 2011 (Ms Hamer). At the beginning of the trial, objection was taken by Mr Featherby to the admissibility of this evidence. It was agreed however that I would hear the evidence nonetheless and rule on its admissibility in this judgment. I deal with the point below at paragraphs (57-61).

The Litigation Friend DE

37. DE had no recollection of having attended the EPAU in November 2010. She could not remember ever having passed blood from her vagina at any stage in the pregnancy and she thought that if she had done so then this was something which she would have remembered. So far as she was concerned, the pregnancy was uneventful save for the occasion in February 2011 when she was taken to hospital by a work colleague with abdominal pain and pins and needles in her arms and legs. She told me that during the evening of 23 February 2011 she had started getting pain in the lower part of her abdomen, on the right side. The pain had not been so bad as to prevent her from getting off to sleep. However, the next day at work the pain continued but it was coming and going. On one occasion she stood up from her desk and had an odd and unpleasant sensation of pins and needles in her arms and legs which made her think that she was going to collapse. Her supervisor (whose agreed statement was part of the evidence) witnessed her expression of pain (wincing and wobbling as though about to fall) and her supervisor took her to hospital. DE agreed that the abdominal pain came and went and so she did not dispute that she may have told Midwife Halliwell that it was not present when she was seen by her at 12.40; nor did she dispute that she may have told Dr Lee that the pain was not there when she was seen at 14.00. However, her recollection was that during the second trace (that starting at 14.32 and running to 15.30) she had another episode of pain which she reported to Midwife Halliwell afterwards. She said she could not remember exactly what she said to Midwife Halliwell but she definitely mentioned it to her. She went home and by the next day she was back to her usual self. She agreed that the episodic pain had lasted for no more than 24 hours, "a dayish." Neither the pain nor the "*funny sensation*" as she described it came back during the pregnancy.

Nurse Burridge and Dr Dadebo

38. Two witnesses were called by the Defendant covering the events of November 2010: Ms Burridge and Dr Dadebo. The claim is now restricted to the management and decision-making of Dr Dadebo alone but given that Mr Featherby maintains that Ms Nurse Burridge's evidence has a bearing upon Dr Dadebo's role and his management, I mention it here. Nurse Burridge

told me that she would have taken her pro-forma history in to Dr Dadebo in the adjacent room within the EPAU and waited in the room whilst the scan took place. Although she had no recollection, she thought that following the scan Dr Dadebo would have spoken with DE to reassure her of his scan findings and advise on further management. She would have given DE a copy of the discharge letter which she had completed for inclusion in the handheld notes. The EPAU notes including Dr Dadebo's scan report would have been filed in DE's general notes. Nurse Burridge added that any clinical decision concerning management would have been made by Dr Dadebo and not by her.

39. Dr Dadebo explained that his reason for performing the scan, in the light of the complaint of fainting, was a concern that the pregnancy might be ectopic. The scan which he performed confirmed that the pregnancy was intrauterine but also showed a small area of hypoechogenicity which was highly suggestive of a retroplacental haematoma as he recorded in his scan report. He told me that in his experience this was a "*very very common finding in pregnancy*" and that typically the haematoma would resolve spontaneously. He said that in the absence of vaginal bleeding or pain, he thought that he would have reassured the Claimant's mother that provided that the placenta continued to supply the baby adequately then the pregnancy would continue but that if she had pain or bleeding then she should return. It was put to him that his function on 22 November 2010 had been that of a sonographer only and he made no clinical judgements at all about concerning management: Dr Dadebo accepted that his note was not as complete as it might have been, in that he had not personally recorded either his clinical thinking or his management plan, but denied that this was because all he had done was perform a scan without interpreting its significance. He observed that the note which had been made by Nurse Burridge that the Litigation Friend should keep the 20-week anomaly scan, was an accurate record of his plan.

40. Dr Dadebo did not accept that he should have made a referral to the consultant obstetrician. The relevant Trust Guidelines "*Booking and Referral Criteria*" (May 2010) set out the indications for referral for consultant care. They included (a) a bleeding disorder in mother or fetus (b) recurrent unexplained ante partum haemorrhage at a gestation greater than 12 weeks (c) placental abruption (d) vaginal bleeding and/or abdominal pain 16 – 24 weeks. Dr Dadebo told me that the presentation on 23 November 2010 did not fit within any of those categories. His interpretation of a bleeding disorder would be a condition such as thrombophilia or haemophilia and there was no suggestion that either the mother or baby were affected in this way. He told me that there was no ante partum haemorrhage and no placental abruption "*by definition*" nor was vaginal bleeding or abdominal pain a feature of her presentation. In his questioning Mr Featherby appeared to accept that none of the categories for consultant referral were met but looking at those categories or indications "*in the round*" there was a case for referral to a consultant. Dr Dadebo did not accept that this was an appropriate use of the Guidelines, nor did he accept that the finding of a retroplacental haematoma represented a "*potential catastrophic problem*"; nor that "*there was a risk here of placental abruption*."

41. For completeness, I record that I heard evidence from Ms Singh, Ms Gillibrand and Dr Dwivedi concerning the decision to administer anti D immunoglobulin on either the 23 or 24 November. The sum of the evidence is that there was a discussion (the precise impetus remains unclear) concerning DE's rhesus negative status involving all three women. Whether Dr Dwivedi was spoken to by telephone or in person is not, in my view, relevant. The only point of note is that, when she administered the anti D immunoglobulin on 25 November, Nurse Gillibrand made an entry in DE's handheld notes recording the fact that a small area of *"? haematoma*" had been noted on the scan.

<u>Ms Fiona Clarke</u>

42. Ms Clarke had been a consultant in obstetrics and gynaecology since 2000. She explained that, had DE been referred to her for an opinion, following the Dadebo scan, she would have reassured her and told her that the appropriate course was to continue with her routine antenatal care including the 20-week anomaly scan. This was because the finding of a retroplacental haematoma was, in the absence of vaginal bleeding, not dangerous. It was a common finding and the vast majority resolved.

Midwife Halliwell

43. I need only mention three aspects of Midwife Halliwell's evidence, other than the contents of her note. First she said that the initial CTG was instigated by her as part and parcel of a routine "work up" so that the doctor who was then to review DE would have all of the relevant information; second, had she been informed by DE of a further episode of pain during the second CTG trace, she would have noted it and brought it to the attention of Dr Lee; and third, she did not think that she would have told DE that the explanation for her pain was that the baby was pressing on a nerve. She might however have explained that the baby's position might have made it more uncomfortable for her.

Dr Lee

44. Dr Lee told me that it would be routine practice in the birth unit to obtain a CTG trace of the fetal heart as part of the initial midwifery assessment on arrival. She did not therefore instigate the first CTG. She reviewed the mother and performed her physical assessment (both abdominal and neurological) noting her findings in the records. Her abdominal examination was designed to find out whether the mother was contracting, to identify any fetal movement and to assess for abdominal tenderness. Neither examination gave her any concerns about the pregnancy. DE's handheld notes recorded the suspected presence of a haematoma but she would not have had access to the scan report itself which had been filed in the general notes. She would however have been aware of the normal 20-week scan.

45. She told me that she reviewed the first CTG trace but concluded that the trace was uninterpretable and not a suitable basis for clinical decision making. It was put to her that notwithstanding the loss of contact the first trace showed some accelerations and decelerations and a period of tachycardia. Her view however was that given the monitor may not have been attached properly then it could be recording maternal movement.

46. She considered that the second trace which started at 14.32 and ran until 15.30 was reassuring. She described it as such in a note which she made at the end of the paper trace by reference to the mnemonic "Dr C Bravado:"

    Dr = Define Risk – 30/40 abdo pain

    C = contractions – nil

    BR= baseline rate – 130 normal

    A = accelerations – present

    Va = variability – normal

    D = deceleration – variable shallow

    O= overall - reassuring

47. A number of features of the second trace were put to Dr Lee by Mr Featherby. She agreed that it showed a small number of shallow variable decelerations with quick recovery but on her interpretation the trace was otherwise healthy, it showed a number of accelerations, a normal baseline and normal variability.

48. She was taken to two sets of local guidelines: the Antenatal Triage Guidelines which included a care pathway for a mother presenting with abdominal pain at a gestational age of more than 20 weeks. The pathway indicated the need for registrar review in the event of any risks being identified on history or examination. Dr Lee did not accept that the mother fulfilled the criteria for the care pathway as, at the time of her hospital assessment, no pain was present and the CTG had been, as judged by her, normal. The second set of local guidelines was the relevant "Booking and Referral Criteria" which listed as an indication for referral for consultant opinion: "CTG request." It was suggested to her that, as she had requested a repeat trace, the guideline for referral was satisfied. She did not accept this. She explained that CTG monitoring was part of the routine assessment on arrival at the birth unit and all she had done was request that the monitoring be repeated due to loss of contact producing an uninterpretable trace.

Mr Johnson Amu

49. He told me that he did not review DE himself nor personally review the CTG trace. If he had been contacted by telephone to discuss the trace then he would have asked for a description of the trace. If he had been told that there were variable decelerations then he would probably have wanted to see the trace for himself.

Dr Hamer

50. She was the consultant obstetrician to whom DE would have been referred by Dr Lee/Dr Amu. Her evidence was that having reviewed the notes and the CTG trace for the purposes of the litigation, had DE been referred to her she would have discharged her for review to the antenatal clinic. She would not have instigated consultant led antenatal care. She would not have requested serial growth ultrasound scans. Her reasoning was that both CTG traces were essentially normal for this gestation and that pain in pregnancy was very common and nonspecific and if it had been brought to her attention it would not have triggered serial growth scans.

Expert evidence on Breach of Duty and Factual Causation:

Mr Gerald Mason.

51. The Claimant relied upon the expert evidence of Mr Mason, a specialist in obstetrics and feto-maternal medicine. He was a consultant in the NHS until 2012 when he took early retirement. He told me that he remained in clinical practice both within the UK and overseas. He gave evidence over the best part of a day.

52. Mr Mason's central thesis was that the presence of a retroplacental haematoma at 16 + weeks' gestation, without more, mandated consultant review and serial ultrasound growth scans during the third trimester of pregnancy. The rationale for this opinion was that a retroplacental haematoma is, definitional niceties apart, an antepartum haemorrhage and a placental abruption. He accepted that the haematoma or clot may resolve over time. But that did not, in his opinion, alter the fact that the presence of the clot would have damaged that portion of the placental surface over which the clot had formed and that that damage was irreparable, causing a reduction in the surface area for gas exchange between the fetus and her mother. Although the impact of the lesion would depend on the size of the damaged area and the underlying

functional reserve of the placenta, the existence of a retroplacental bleed was very significant and had the potential to interfere with fetal growth. He was therefore critical of Dr Dadebo who had not considered the finding to be significant and who had failed to refer the mother for consultant care. He was by implication also, critical of the hypothetical management of DE by Ms Clarke who would not have kept the mother under consultant review and would not have arranged for serial ultrasound growth scans. He did not accept that either Dr Dadebo or Ms Clarke should have relied upon the fact that, at 20 weeks, a detailed anomaly scan was to be undertaken. This might give false reassurance as the placenta is not generally visualised in any detail and the residual damage to the surface of the placenta caused by a resolved haematoma would not be seen.

53. Mr Mason was critical of the management by Dr Lee. He disputed her interpretation of the CTG traces. In his opinion, although the first CTG was affected by loss of contact, it should have been reviewed carefully by Dr Lee and, if it had been so, she should have appreciated that it demonstrated abnormal features. There were a number of decelerations associated with a possible rise in the fetal heart rate to 170 bpm which was, he told me, an unusual pattern to see in a 30 week gestation fetus. In his opinion, the second trace showed no accelerations as there were no sections where, to his eye, there was a rise in the fetal heart of 15 beats per minute sustained over 15 seconds. There were however decelerations, some reduced beat to beat variability with he thought either a change in the overall baseline or a series of decelerations before the trace was switched off. In his view, the trace in conjunction with the clinical presentation of pain mandated consultant management of the pregnancy and serial growth scans.

54. In Mr Mason's opinion, if serial ultrasound scans had been undertaken, then by around 34 weeks (and not 30 weeks as pleaded) the Claimant would have been shown to be small for gestational age. The cause for the growth restriction was damage to the placenta caused by the abruption in November 2010 and in his view by a second abruption which caused the mother's condition in February 2011. If her growth restriction had been diagnosed then the pregnancy would not have been permitted to continue beyond 38 weeks.

55. Mr Mason was cross examined with skill and care, and over a lengthy period. I set out at this stage only the key points of his evidence which emerged at this stage:
    a. He did not accept that it was inaccurate to classify or describe the haematoma, found at 16 + weeks, as a "placental abruption." Although generally a placental abruption is defined as a collection of blood which appears on scan (and/or is otherwise obvious clinically) after 24 weeks gestation, his view was that a retroplacental haematoma at 16 weeks gestation should be described as an abruption. He posed rhetorically, "*What is a placental abruption other than a retroplacental haematoma*?"
    b. Mr Mason's statement that DE had suffered an antepartum haemorrhage in November 2010 was also challenged by reference to the definition in the Royal College of Obstetricians and Gynaecologists ("RCOG") "Green Top Guideline No 63" dated November 2011, which defines an antepartum haemorrhage as a bleeding in the genital tract which occurs after 24 weeks. Mr Mason's opinion again was that a haematoma is a collection of blood and therefore an antepartum haemorrhage.
    c. Mr Mason accepted that there were no national guidelines which advised practitioners that the presence of a small retroplacental haematoma mandated senior management and serial growth scans. His view however was that all reasonably competent obstetricians would be aware from basic principles that a retroplacental haematoma is going to damage the placenta and therefore potentially put the growth of the fetus in jeopardy and take action accordingly.

d. As to the features of a CTG trace at 30 + 2 days' gestation, he was taken to the paper by *Afors and Chandraharan* in the Journal of Pregnancy 2011, which emanated from research done at St George's Hospital in London, where the authors noted that the characteristics of fetal heart tracings differ in the preterm fetus compared to a term fetus. In the opinion of the authors, before 30 weeks, the fetal baseline heart rate can be higher; the frequency and amplitude of accelerations can be reduced with the fetus exhibiting accelerations with a peak of only 10 beats per minute and shorter lasting; fetal heart rate decelerations in the absence of uterine contractions often occur and baseline variability may also be affected due to incomplete development of autonomic nervous system. At trial, Mr Mason had difficulty in accepting the validity of some of these observations, taking refuge in the fact that the paper dealt with trace features up to 30 weeks' gestation, whereas DE was 30 weeks plus 2 days gestation. I pause to observe that this was a surprising approach given his acceptance in his joint meeting with Mr Howe that "*in preterm fetuses, around 30 weeks gestation there are recognised differences compared with more mature foetuses. These include higher baseline rate, less baseline variability, lower frequency and amplitude accelerations*." When the point was pressed by Mr Whitting, Mr Mason appeared to accept that the only real feature which was abnormal was the absence of (as opposed to the lower amplitude or frequency) accelerations.
e. Mr Mason was also pressed on the appropriate or reasonable management of DE on 24 February 2011. He explained that the trace should have been continued, although he fairly observed that whether the trace would have been reassuring or not was a matter of speculation. He accepted that a growth scan taken at around 24 February 2011 would not have shown that the Claimant had fallen below the 10th centile. His view however (at least in the joint expert meeting) was that serial scans should have been continued given the risk of growth retardation.

<u>Dr David Howe</u>

56. The Defendant relied on the evidence of Mr David Howe, a consultant in fetomaternal medicine in Southampton. I set out only the key points in his evidence below.

57. Mr Howe considered that the management in November 2010 and February 2011 was reasonable:

a. It would not be normal practice in 2010 to invoke consultant led care after an episode of bleeding during the first half of pregnancy. The lesion seen on the ultrasound scan was small and not associated with vaginal bleeding. Such a finding would be common in the first half of pregnancies, affecting around a quarter of pregnancies.
b. There is no published guidance on management which supported the approach advised by Mr Mason. The 2011 RCOG "Green Top Guidance" dealt with antepartum haemorrhage as defined there as (and as generally understood to be) a bleed into the genital tract after fetus viability, that is, 24 weeks. By contrast, his view that no additional action was required was supported by a leading textbook dealing with high risk pregnancies edited by James and Steer. The text advised that vaginal bleeding at less than 20 weeks' gestation indicated the need for an acute referral to hospital as a sign of threatened miscarriage but no long term additional action. Nor did the contemporaneous RCOG guidance on small for gestational age pregnancies relevant to 2010 (published in 2002) offer the advice that serial scans were indicated after bleeding in the first half of pregnancy. There was therefore no published standard or guidance which supported the allegation of breach of duty.

c. The clinicians were entitled to place weight on the 20 week anomaly scan. Mr Howe's evidence was that the placenta can be visualised clearly at 20 weeks. The fetus would not be so big as to obscure it and, even if the sonographer was not aware of the previous finding, it would be part of the routine examination to look at the placenta because part of the value of the scan is to exclude a low lying placenta which covered the cervix and for that purpose the full extent of the placenta would need to be examined.
d. Mr Howe accepted that one of the risks of a placental separation was the likelihood of the mother going on to have further separation (which if it occurred would be classified as an abruption). He did not accept however that early pregnancy bleeding (such as experienced by DE) was associated with fetal growth restriction, hence the fact that serial growth scans are not standard practice.
e. Mr Howe's view was that neither the first nor second CTG trace demonstrated abnormal features given the gestational age of the fetus. To his eye there were shallow accelerations such as he would expect to find in a healthy 30 week gestation trace.
f. He supported the management on 24 February 2011, as he did the management on 22 November 2010. In his opinion, the hypothetical management by Ms Clarke and Ms Hamer would have been both standard practice and consistent with a reasonable standard of care.

Legal Principles

58. There is no material difference between the parties as to the relevant test which I must apply. I apply the "*Bolam*" test which derives from the direction given to the jury by McNair J in *Bolam v Friern Hospital Management Committee* [1957] 1 WLR 583 at 587 where the judge stated in connection with a medical doctor: "*he is not guilty of negligence if he has acted in accordance with a practice accepted as proper by a responsible body of medical men skilled in this particular art… Putting it the other way round, a man is not negligent, if he is acting in accordance with such a practice merely because there is a body of opinion that would take a contrary view*." In the present case therefore and re-formulating the Bolam test, the question is whether no reasonably competent obstetrician would have acted and exercised judgement in the way in which Dr Dadebo and Dr Lee did, in fact, by not referring DE for consultant review or that Ms Clarke or Ms Hamer would have done by, as they told me, reviewing DE and referring her back to routine antenatal care. I bear in mind that differences of opinion and practice exist and will always exist in the medical and other professions. A court may prefer one body of opinion to another, but that is no basis for a finding of negligence. See: *Maynard v West Midlands RHA* [1984] 1 WLR 634 at 638E.

59. I recognise that in making my assessment of the evidence, I should not delegate the task of deciding the issues in the case to the expert. The issues are for the court to decide taking into account all of the evidence. In making the assessment of whether to accept an expert's opinion the court should take into account a variety of factors including, but not limited to: whether the evidence is tendered in good faith; whether the expert is "responsible", "competent" and/or respectable; and whether the opinion is reasonable and logical. See: *Bolitho v City and Hackney HA* [1998] AC 232. In this context, the task of the court is to see beyond stylistic blemishes and "*to concentrate upon the pith and substance of the expert opinion and to then evaluate its content against the evidence as a whole and thereby assess its logic*." See: *C v Cumbria University Hospitals NHS Trust* [2014] EWHC 61.

Discussion/Conclusion: Breach of Duty

a) Admissibility of Evidence of Clarke and Hamer

60. I start by clearing the decks of the issue raised by Mr Featherby concerning the admissibility of the evidence of Ms Clarke and Ms Hamer. It must be said that, even at the point of his closing submissions, it remained unclear to me whether there was a challenge to the admissibility of the whole of that evidence, or only part of the evidence, or whether the impact of the objection is that I should place little or no weight on the evidence of those two witnesses.

61. The starting point (and, in my view, end point) to any challenge on the admissibility of what, as a matter of fact would have occurred if DE had been referred to a consultant obstetrician in either November 2010 or February 2011, is *Bolitho* [see above]. That case concerned an alleged negligent failure by a paediatrician to attend a child with breathing difficulties and to intubate. The paediatrician admitted a culpable failure to attend but denied that, had she done so, she would have intubated the Claimant. The case states that the approach to be taken by the court in these circumstances is to pose two questions: first, what would have been done had the clinician attended? Second, if she had failed to intubate, would that have been negligent? The first question involves a factual enquiry, to which the *Bolam* test is of no relevance. The *Bolam* test is however central to the second question. It follows that factual evidence dealing with what would, hypothetically, have happened in a particular set of circumstances is admissible evidence. To the extent that there is a dispute (as in this case) as to the reasonableness of that action, then I must assess it by reference to the *Bolam* test.

62. I have therefore no doubt that, insofar as the evidence of Ms Clarke and Ms Hamer describes what they would have done had DE been referred to them and why they would have taken their particular course, is admissible. Further, that evidence does no more than confront and deal with the factual allegations in the Re-Re-Amended Particulars of Claim at paragraph 24 which assert precisely what "*would have happened*" if a referral to a senior obstetrician had been made in November 2010 and February 2011. In these circumstances, it would be curious if for some reason the evidence of those doctors to whom it is said a referral would have been made were not admissible.

63. The witness statements provided by Ms Clarke and Ms Hamer are limited to what they would have done had DE been referred to them and their rationale. The statements were obviously carefully drafted with an eye to avoid trespassing into inadmissible opinion evidence. To the extent that in their oral evidence they did go further, then this was in response to the questions posed in cross examination by Mr Featherby. In any event though, I put out of my mind any part of their evidence in which they have commented more generally and with an expert eye on the issues raised in the case.

64. As to the weight to attach to their evidence, I accept that they remain employees at the Trust. That said, they struck me as very fair and undefensive witnesses and I record at this stage that I accept their evidence concerning their management had DE been referred to them and the reasons which they gave for that management.

65. However, I recognise the force of Mr Featherby's submission that my evaluation of the merits of the claim should not be a question of weight of numbers (of witnesses) and to that extent I should not be tempted to reject Mr Mason's evidence simply because there are three doctors (Clarke, Hamer and Howe) who would not have adopted the course which Mr Mason contends would have been the only reasonable course to take. I accept this point. My evaluation of Mr Mason evidence should not be and has not been influenced by the number of doctors which the Defendant has been able to marshal in support of its position.

b) Factual Conclusions

66. I turn first to my assessment of the factual witnesses and give my conclusions on the various factual issues which Mr Featherby raises for my determination.

67. In closing his case, Mr Featherby launched his, highly personalised, attack on Dr Dadebo, submitting that he was "*dissimulating*", *"unconvincing*" and "*disingenuous*" in his explanation that he performed a scan to exclude an ectopic pregnancy. Mr Featherby's point here is that Dr Dadebo should or would have known that an earlier scan had shown the pregnancy to be intrauterine. Setting aside the stumbling block that Mr Featherby did not put this to Dr Dadebo in cross examination, I reject this assessment of Dr Dadebo. Not only did he strike me as a fair and open witness who was prepared to make concessions (for example in connection with his note keeping) but Mr Featherby's submission only begs the, to my mind, unanswerable question of why Dr Dadebo would suggest the need to exclude an ectopic pregnancy if it were not the truth.

68. I do not accept that Dr Dadebo was, as Mr Featherby submits, acting purely as a sonographer or technician on 22 November 2010. I accept his evidence that he performed the scan, that he then interpreted it and using his clinical judgement and experience concluded that the finding was not of particular significance for the reasons which he gave. His evidence that he was the clinical decision maker accords with the evidence of Nurse Burridge and I reject the suggestion that Dr Dadebo was, for some reason, seeking to dovetail his evidence to fit with that of his nursing colleague. Mr Featherby's submission begs another (to my mind again, unanswerable) question: why would Dr Dadebo have performed the scan in the first place if he was not intending to take clinical responsibility for it and advise on his findings. The only conceivable basis upon which Mr Featherby could found his submission is the absence of a contemporaneous clinical note by Dr Dadebo setting out the management plan. However as both he and Nurse Burridge said, Nurse Burridge's note covers the management plan sufficiently.

69. There is no basis at all for Mr Featherby's submission that Dr Dadebo was not an honest witness and I reject the submission out of hand. I accept Dr Dadebo's evidence of what he did on 22 November 2010 and why he reached his conclusions.

70. Mr Featherby's closing submissions were also directed at the reliability of the account given by Dr Lee. Many of the points which he makes concern timings. Mr Featherby describes "*irreconcilable anomalies*" in her evidence. For example:
    a. the timing of her discussion with Dr Amu which Mr Featherby submits must have been (for various reasons) before the start of trace 2. However, her witness statement makes the point that doing the best she can to reconstruct the events of the afternoon, she too believed that she discussed her plan with Dr Amu before (and not after) the start of the second CTG trace. This statement was never put to her in cross examination.
    b. Mr Featherby also submits that the timing of the note at 15.00 is another irreconcilable anomaly, but it is not. Dr Lee explained that she thought that the timing on the note was wrong and that she only reviewed the trace after it had finished at 15.32. I accept this evidence: her "Dr Bravado" note is written on the paper trace, right at the end. If she had reviewed and discharged DE half an hour earlier, the location of this note would make no sense.

71. In summary therefore I also accept Dr Lee's evidence of what she did and her reasons. I did not find her to be defensive as Mr Featherby suggested in his closing. Like the other Trust witnesses, she struck me as someone who was anxious to do her best to give an honest and accurate reconstruction of her actions and thought processes many years before. I accept her

evidence in her statement that her conversation with Dr Amu preceded the second trace and note in this context her own admission that she should have discussed the second trace (as well as the first) with someone more senior. Nothing turns on this point though, as I accept that had a referral been made to Ms Hamer she would have discharged DE back to routine antenatal care.

72. I turn finally to Midwife Halliwell, and the issue which arises for my determination of whether DE reported a further episode of pain during the second trace to her. I do not accept that DE has trimmed her evidence to fit in with the records as submitted by Mr Whitting. However, I am satisfied that if such a complaint of pain had been made, then Midwife Halliwell would have recorded it. Midwife Halliwell had been interested and concerned to find out whether DE was in pain or not on her arrival and she noted the negative response to questioning on the point. If spontaneously DE had reported pain after the second trace, then it would be inconsistent with her earlier interest in the topic to have failed to record it and act upon it. I find that DE has simply mis-remembered this, given the interval of time since the events in question.

c) Conclusions on Expert Evidence

73. I deal first with the significance of a finding of retroplacental haemorrhage at 16 + weeks and Mr Mason's opinion that it should have led to consultant antenatal care and serial growth scans through pregnancy. In this context, I note the following points:

a. The Green Top Guideline No 63 on Antepartum Haemorrhage was published by the Royal College of Obstetricians and Gynaecologists in 2011 and developed primarily for clinicians working in obstetric units in the UK. The Guideline defines antepartum haemorrhage ("APH") as "*bleeding from or into the genital tract occurring from 24 weeks of pregnancy and prior to the birth of the baby*." It goes on to say that one of the most important causes of APH, although not the most common, is placental abruption as it is a leading cause of perinatal and maternal morbidity worldwide. Within the section addressing whether antenatal care should be altered following APH, the Guideline advises that: "*following APH from placental abruption or unexplained APH, the pregnancy should be reclassified as "high risk" and antenatal care should be consultant-led. Serial ultrasound for fetal growth should be performed*."

b. It is clear from a plain reading of the Guideline above that the authors are advising that in the event of antepartum haemorrhage (as defined in the Guideline) which is considered to be secondary to a placental abruption, then the appropriate management is consultant led antenatal care and serial growth scans, in other words, just that management which Mr Mason states was mandated in the presence of the finding of a small retroplacental haematoma at 16+ weeks gestation. The Guideline however confines the definition of an antepartum haemorrhage as a bleed in the genital tract occurring after 24 weeks. It does not state or even suggest that similar management to that advised in the event of a bleed after 24 weeks should follow a bleed before 24 weeks and at 16+ weeks' gestation. I am driven to conclude that if the Royal College had intended that the same management should be provided in the event of a bleed at 16+ weeks gestation, then the Guideline would say this. It does not do so. The question is why not?

c. Mr Howe provides the complete answer to the question. He explained to me that the management of bleeding in pregnancy differs according to the gestation. The risks associated with the condition, the requirements for investigation and the potential for clinicians to intervene are different at different gestations. At an earlier gestation the risk presented is of miscarriage. A bleed after 24 weeks however carries much more serious risks of additional and severe life-threatening complications: disseminated intravascular coagulation and major obstetric haemorrhage. The options for treatment also differ according to the gestation and, critically, after 24 weeks the fetus is viable so the clinician has the option to intervene to deliver the baby which would not be present before 24 weeks. As he put it simply, a retroplacental haematoma and an abruption are both similar in the sense that they involve a separation of the placenta from the uterine wall, but the implications of the separation are very different. For this reason, it is not appropriate to describe them as variations on a theme.

d. Mr Howe's evidence puts the guidance provided to clinicians in the Green Top publication in a logical context. Furthermore, as Mr Mason was driven to accept in cross examination, there are no national guidelines which advise that a retroplacental bleed at 16 weeks should be managed in the same way as a bleed in later pregnancy. He was unable to point to any literature which supported his view. By contrast, Mr Howe took me to a standard text book "*High Risk Pregnancy*" edited by James and Steer which was current at the time (published in 2006) which advised that the appropriate management of vaginal bleeding before 20 weeks was an acute referral to hospital (for risk of miscarriage) but, critically no long term additional action. Mr Howe also explained that even now and following national guidance published in 2016 intended to reduce the incidence of stillbirth, bleeding in early pregnancy is not one of the indications for serial scanning.

e. The Trust local guidelines do not assist the Claimant either. The Booking and Referral Criteria dated May 2010 refer within the criteria for referral for consultant care to "*recurrent unexplained antepartum haemorrhage at a gestation of more than 12 weeks*" Mr Featherby relies upon this statement as a pointer in the direction that antepartum haemorrhage is not confined to a bleed after 24 weeks. But I neither follow nor accept this point: the Guidelines qualify the phrase antepartum haemorrhage by reference to the gestation to which it is referring making it clear that the terms are not, as submitted, "interchangeable." The Defendant's 2009 Guidelines on "Major Obstetric Haemorrhage" specify that the causes of bleeding from the genital tract both before and after 24 weeks are the same. But this is not Mr Howe's point. His point is that the risks of the condition before and after 24 weeks are different: hence the different investigations and the different management options.

f. Finally, on this topic, I deal with two submissions which feature in Mr Featherby's written closing submissions.

24 "The first is that Mr Howe's expert opinion that serial ultrasound scanning was not mandatory was not *Bolitho* logical and rational. Underpinning the submission, is the assertion that "*serial scanning is non-invasive, not inconvenient and inexpensive*," points which were never explored in evidence. The absence of evidence on the points is determinative. I add that whilst I might accept that scanning is non-invasive, I am not prepared to accept that serial scanning is not inconvenient and not expensive. I have heard evidence, which I accept, that findings of retroplacental haematoma are reasonably common in the second trimester. At face value, serial ultrasound scans for all of these

women throughout their pregnancies would it seems to me be worrying and inconvenient for them; and inconvenient and expensive to administer and implement.

25 The second submission focusses upon what DE should have been told by Dr Dadebo concerning the scan finding. When pressed, Mr Featherby accepted that he was not and could not embark upon a case which was based in some way upon informed consent. This has never been how the case was formulated. The point therefore takes him nowhere. I add however that Dr Dadebo gave evidence as to what he believed he would have said to DE which would have included the finding which he had made and his advice that she should keep her antenatal appointments including her 20 week scan. I accept this evidence and, for the reasons which I set out below, accept that it was reasonable for him to have limited his advice to DE in this way."

74. In addition to the points which I have raised above, there were some very curious aspects to Mr Mason's evidence which I cannot overlook.

75. Mr Whitting's closing submissions emphasise the evolution of the Claimant's case from the letter of claim through to the latest iteration of the claim in the Re-Re-Amended Particulars of Claim, the implication being that the changes in the case mirror changes in Mr Mason's thinking. I can't accept this submission at face value. It seems to me that, in this case as in any other, there may be the potential for subtleties in the case being overlooked or lost in translation. However, that aside, Mr Whitting has some good forensic points arising from Mr Mason's report of February 2019 and his evidence at trial.

76. The first concerns Mr Mason's statement at the beginning of his examination in chief that his opinion had been strengthened very considerably by disclosure (late) of the EPAU records which included the scan report which confirmed the location of the haematoma as retroplacental. Mr Mason's problem here however is that, as it emerged during cross examination, his thesis that the haematoma was equivalent to an antepartum haemorrhage and placental abruption, had always been founded upon the assumption that the location was retroplacental. I accept that the confirmation of his assumption by the report of the scan would have been reassuring but not that as a consequence his opinion had been strengthened in the way he described. Also, that he had only assumed that the bleed was retroplacental was not a fact which he made clear in his report of 2019. This would have been an important point to mention given that he told me that an alternative contender for the location of the bleed would have been beneath the chorion membrane. Such a bleed was far more common than a bleed behind the placenta. If the bleed had been sub-chorionic then the management implications would have been different as a sub-chorionic bleed would be unlikely to have any long term impact on placental function. He should have made clear that his opinion was founded on an assumption. He did not do so.

77. This raises another linked issue. As I have already said, Mr Mason informed me that a sub-chorionic bleed as far more common than a retroplacental bleed. He was asked by Mr Whitting therefore why, when he wrote the report, he had assumed that the location of the bleed was behind the placenta. Initially he answered by stating that the placenta was normally sited and that this made a retroplacental bleed more likely. Having accepted in further questioning that both a retroplacental bleed and a subchorionic bleed are associated with a normally sited placenta, he offered the further explanation that his assumption had been based upon the outcome of the pregnancy. A new point which, as Mr Whitting observed, did not feature in his report.

78. The further point which arises from the 2019 report is based upon Mr Mason's statement in that report that DE had suffered a significant antepartum haemorrhage. In reaching that conclusion, he appears to rely upon three facts: that the scan had demonstrated a haemorrhage; that a blood sample had been sent to the transfusion service for analysis and a labour summary which recorded the fact that DE had suffered from vaginal bleeding during pregnancy. In fact, the scan report described only a small area of haematoma, the blood analysis was likely to have been in connection with the administration of anti D immunoglobulin and (this was not disputed) the reference in the labour summary to DE having had a vaginal bleed was a mistake. Two points arise. First, the basis for Mr Mason's reported conclusion that DE had suffered an APH was wrong. Second, and of more significance, as Mr Whitting explored in his cross examination, there was really no need for Mr Mason to find support for his conclusion that DE had suffered an antepartum haemorrhage if, as he maintained at trial, the fact of the retroplacental haematoma was synonymous with an antepartum haemorrhage. All he need state in the report is that there had been a haematoma.

79. I do not accept Mr Whitting's "punchline" that Mr Mason was making the case up as he went along or, as Mr Whitting suggested, that Mr Mason had deviated so far from his duties as an independent expert such that I could conclude he had taken on the role of advocate in the case. However, I accept that there were a number of aspects of his evidence which were very unsatisfactory and that, overall, the impression was not favourable. However, in rejecting (as I do) Mr Mason's thesis that a small retroplacental clot at 16 + weeks gestation mandated a referral to a consultant, consultant led antenatal care and serial growth scans I have focussed on the "*pith and substance*" of his opinion (see Green J in *C*), rather than matters of impression or style. I accept Mr Howe's evidence that a retroplacental clot at 16 + weeks' gestation is in the absence of vaginal bleeding a noteworthy but clinically innocuous finding and that it required no special or enhanced antenatal care. His opinion is consistent with national teaching at the time (and now). I find that Mr Mason's opinion that (as it was put in closing) the terms are "clearly interchangeable" and that to suggest otherwise is to take a "pedantic approach" is wrong. In terms of the associated risks, the range of investigations and the management options, the terms are quite different. I therefore accept that a reasonable body of obstetricians in November 2010 would have noted the finding but advised the continuation of routine antenatal care: Dr Dadebo did not act in breach of his duty of care to DE nor would Ms Clarke have acted in breach of duty had she seen DE and referred her back to routine antenatal care.

80. This deals with management in November 2010. I turn then to the management in February 2011. Given Mr Mason's ultimate acceptance that the trace features must be considered in the light of the fact that the trace is of a preterm fetus, I find I can take this relatively shortly. As Mr Whitting submits, Mr Mason's analysis of the trace boils down to one feature which he considers to be abnormal: the absence of accelerations. All of the other features which were exhaustively examined in cross examination of Dr Lee and in Mr Mason's own evidence were by the conclusion of the evidence agreed to be common features of a trace of a preterm fetus. In respect of this outstanding issue for my determination, I note two things: first that the *Afors* paper states that in a preterm fetus it is normal to find fewer and lower amplitude accelerations; second Mr Howe did not accept that there were no accelerations. He described and pointed out to me a number of low amplitude accelerations such as he would expect to see in a trace at this gestation. His opinion was that overall, both CTGs demonstrated a heart rate pattern typical of a healthy baby at this gestation, with shifts between low and high amplitude variability consistent with a baby in a sleep and awake phase, shallow short lived variable decelerations and a series of low amplitude accelerations. I was able to see for myself the accelerations which he referred to. I accept this evidence. Even if I am wrong in this, I accept Mr Whitting's point that Mr Mason accepted that the absence of accelerations is considered to be an equivocal

feature of CTG interpretation and that there is no NICE or RCOG guidance which supports Mr Mason's statement that the absence of accelerations at this gestation would require further action.

81. I also mention DE's clinical presentation on 24 February 2011, although again I can take this shortly given that Mr Mason accepted that, viewed in isolation, it did not indicate enhanced antenatal care. Both Mr Howe and Mr Mason accepted in their joint discussion that it is very common for pregnant women to be seen with pain, including abdominal pain which subsequently resolves spontaneously and for which no specific diagnosis is made. They observe that in any large hospital maternity unit, this would be an "*almost daily occurrence*" and the numbness of the legs and arms is most likely to have been due to hyperventilation caused by anxiety or pain or both. Given this agreement, my finding that the CTG was normal and my conclusion above that the scan finding did not indicate the need for consultant review, the point falls away.

82. Having considered each of the key allegations, I find that the Claimant has not made out her case on breach of duty on either of the occasions: November 2010 or February 2011. The management of the clinicians at the Trust was reasonable. The management which Ms Clarke and Ms Hamer have said that they would have instigated would also have been reasonable. This finding is dispositive of the claim.

83. I therefore need only tie up Mr Mason's loose ends on factual causation. I bear in mind his evidence that, had the CTG trace been continued on 24 February 2011 (as he told me in his opinion should), it remained a matter of pure speculation as to what it would have shown. He could not say, one way or the other, whether it would have been reassuring or not. I also bear in mind his agreement that a growth scan at 30 weeks would not have shown that the Claimant was suffering from growth retardation. In order for the Claimant to make good her case therefore she would have to be able to satisfy me that notwithstanding these matters, the only reasonable management would have been to continue repeat ultrasound scans to check on growth until 34 weeks (or thereabouts). The only basis upon which such a course could be justified would be if I were to have found that the presence of a retroplacental clot at 16 weeks was a clinically very significant finding. For the reasons which I have given, I am against the Claimant on this issue. Finally, there remains an issue between Mr Mason and Mr Howe concerning the cause of the Claimant's growth retardation and (a) whether it was linked in any way to the finding on scan in November 2010 and (b) whether there was a further bleed in February 2011. Given the agreement that, whatever the cause of the growth retardation, had it been diagnosed after 34 weeks it would have led to an early delivery, it does not seem to me to be an issue which I need to decide. Nothing turns on it.

84. Notwithstanding my conclusion on breach of duty, I do however address the Claimant's case on medical causation given its importance to the parties.

The Timing and Cause of the Claimant's Stroke

85. The Claimant relied upon the evidence of Dr Kevin Ives, a consultant neonatologist at the John Radcliffe Hospital in Oxford and Dr Gayatri Vadlamani, a consultant paediatric neurologist at Leeds Teaching Hospital NHS Trust. The Defendant relied upon the evidence of Dr Anthony Emmerson a consultant neonatologist, now retired.

86. The issue for me at this stage is whether the cause of the stroke was embolisation of placental debris into the fetal arterial circulation. As explained by Dr Ives, the Claimant's case is that the source of the embolus was likely to have been placental clot or debris associated with the gritty and calcified placenta at birth. The embolus would have travelled from the placenta via the placental vein, into the right atrium where it would pass through the patent foramen ovale into the arterial circulation and then along the carotid artery before lodging in the middle cerebral artery. If I find, on balance, that this is the likely mechanism for the stroke, then it is agreed between the parties that earlier delivery would have avoided the stroke as (a) the embolisation would have had to have occurred before delivery (or at least before the umbilicus was cut) and (b) delivery at 38 weeks would have avoided the embolisation (not least because the placenta would not have been likely to have been so degenerate at that gestation). The issue for me is therefore relatively speaking, a narrow one.

87. I agree with Mr Whitting that the issue is largely unlocked by my conclusions on two matters: first the current state of knowledge on the cause or causes of PAIS and second, given the delay in onset of neurological signs, whether the lodgement of the embolus in the middle cerebral artery might only lead to neurological signs some hours after.

88. I deal first with what is known within the profession concerning the aetiology of PAIS. I find that I can deal with this shortly.

89. Dr Ives accepted that the cause or pathogenesis of PAIS is poorly understood within the scientific and medical world. He accepted that within the literature on the subject there were a large range of conditions which are variously said to be either causes of stroke, or associated with stroke, or risk factors for stroke or possible causes of stroke. In the meta-analysis undertaken by *Li and Others* (published in European Journal of Neurology in 2017) intending to determine the significance of clinical factors and exposures to the risk of PAIS, a list of around 30 conditions were listed as risk factors for PAIS, including primiparity, gender, abnormal CTG tracing, emergency caesarean section and IUGR. In Janet Rennie's textbook "*Neonatal Cerebral Investigation*" in the section addressing causes of stroke she also lists a large number of factors which have been "*identified as increasing the risk of perinatal arterial stroke, including placental disorders."* In Cheong and Cowan's paper "*Neonatal Arterial Ischaemic Stroke: Obstetric Issues*" published in Seminars in Fetal and Neonatal Medicine in 2009, the authors recorded that the pathophysiology of PAIS is thought to be related to clot formation and "*there are risk factors unique to the pregnant woman and fetus which predispose to a hypercoagulable state. Pregnancy is a hypercoagulable state and a period of high risk for maternal stroke. Newborns are vulnerable to stroke in their own right, due to various having a high haemoglobin concentration, relatively slow serum activity levels of protein S and protein C*." In the same publication, also in 2009, In "*Epidemiology and Classification of Perinatal Stroke*", the author John Kylan Lynch observed that "*over the last several years, our knowledge of perinatal stroke has substantially improved but much work remains*." He noted that factors associated with stroke in the perinatal period include "*maternal disorders, blood disorders, cardiac disorders, infection and other miscellaneous disorders*." Within the text he listed a large number of maternal and infant disorders which he described as having been "*reported with*" perinatal stroke.

90. Dr Ives' opinion is that one of the factors predisposing to PAIS is a degenerate placenta. However he accepted that nowhere in the literature was there any study which "*states categorically that there's strong evidence for placental thrombi*" as a cause of stroke. Having reviewed the evidence in cross examination, it seems that the high point of the causal link, possible causal link or association between placental emboli and stroke is to be found in two papers: "*Neonatal Stroke*" by Mary Rutherford and Frances Cowan in British Journal of

Medicine 2011 where the authors comment that "*Neonatal AIS is presumed in most cases although seldom proven to result from emboli from the placenta passing through the patent foramen ovale where the branching of the left carotid artery offers the easiest anatomical path*" (my emphasis). The second is Volpe's most recent textbook "*Stroke in the Newborn*" published in 2017 "*the placenta has been recognised increasingly as a potential source of emboli and cerebral infarction. The principal placental lesions have involved fetal vessels and have been associated with intrauterine infection with chorionamnionitis and maternal and perhaps fetal coagulopathies*" (again, my emphasis). However another (and later) text published in The Lancet by Dunbar and Kirton in 2018 states that "*the pathophysiology of neonatal arterial ischaemic stroke remains incompletely understood in most cases*" and "*the potential role of the placenta in neonatal arterial ischaemic stroke merits consideration. Strong indirect evidence supports placental thromboembolism as a leading cause of neonatal arterial ischaemic stroke..More direct evidence comes from a case control study that reported perinatal stroke to be associated with any category of placental pathology as well as amniotic fluid inflammation*."

91. I have no doubt that Dr Ives has a firm and genuine belief that the placenta was the source of the embolus and the cause of the stroke. However, contrary to Mr Featherby's closing submissions, the literature to which I have been referred falls far short of demonstrating "a well-established causal link" as he puts it between placental deficiency and stroke in term babies. Dr Ives himself did not even suggest that the literature painted such a picture. He agreed with the proposition which was put to him that "*it is a widely held hypothesis that these lesions occur as thromboembolic phenomena from debris originating in the placenta*" adding that he strongly supported that hypothesis. However, as Mr Whitting submitted, a belief, however strong, of an association or possible causal link between placental pathology and PAIS which has not been scientifically demonstrated is not proof of causation in general and certainly not in the particular case. Likewise, a widely held hypothesis, or presumption, is not proof on the balance of probabilities of cause, or in the legal context, of causation.

92. Nor are there any particular factors in the Claimant's case which might lead me to the conclusion on the balance of probabilities that the stroke was caused by placental embolisation. There was general agreement that the Claimant's presentation at delivery does not help me: it was consistent with an emergency caesarean section and meconium aspiration rather than any significant cerebral event. Although the placenta was described as being gritty and calcified, there was no histology of the specimen nor any other finding which confirmed the placenta as being the source of the embolus.

93. Dr Vadlamani offered little by way of an additional relevant perspective on the topic. Her opinion was based upon her assessment of the various "neonatal causes" for PAIS. Having excluded causes such as polycythemia or an intravascular line or a cardiac disorder, she concluded that the presence of an abnormal placenta, placental failure and other features associated with the labour pointed in the direction of a prenatal cause for the stroke. However, her opinion does not (in any material and additional way) grapple with the difficulties which were explored with Dr Ives: namely that the causes of PAIS remain only imperfectly understood; the range of possible causes or associations or risk factors is long and the causal link between an abnormal placenta and stroke has not been established and remains an hypothesis only. It seems to me therefore that I need say no more about Dr Vadlamani's evidence on the point.

94. It follows that I find against the Claimant on medical causation as well as breach of duty. I accept that, on the review of the literature it remains possible that the stroke was due to a placental embolus. But equally on the scientific evidence it could have been due to any one of a huge range of other factors (including, primiparity, an abnormal CTG, the normal

hypercoagulability of the newborn) none of which arise from the alleged negligence of the Defendant. It seems to me that on an application of the well-known principle in *Wilsher v Essex Area Health Authority* [1988] AC 1074, the claim must fail on causation.

95. I also have in mind that there is a further stumbling block in the Claimant's causation pathway; the interval of time, of either 21 hours or 31 hours, before the Claimant demonstrated any abnormal neurological signs and the fact that the cranial ultrasound scan at around 42 hours of age was apparently normal.

96. It is Dr Ives' opinion that the mild hypertonia which was recorded at 21 hours was the first sign of the stroke; and Dr Emmerson's opinion that a mild hypertonia is not unusual in the neonatal period and it was not until 31 hours when the Claimant had a desaturation and turned blue that she showed evidence of the brain infarction. This is not a matter which I need to resolve. Either way there was a gap of some hours between the postulated brain damage and the brain damage becoming clinically obvious. Dr Emmerson's point is a simple one which is that there is no scientifically plausible explanation for a significant gap in time between the occurrence of the brain damage and the baby becoming unwell.

97. Both Dr Ives and Dr Vadlamani sought to address this point: Dr Ives by reference to literature which appeared to him to suggest that there may be a gap of as long as 3 days before the brain damage manifests itself and Dr Vadlamani by reference to a new and unheralded point which was derived from a section in *Volpe* concerning the neuropathology of PAIS. Dr Ives' difficulty is that the literature upon which he relied to substantiate the possibility of a time interval between insult and clinical manifestation does not, I find, make clear whether the authors were referring to strokes in the neonatal period (in which case there was no gap) or strokes which had occurred before birth and the presentation had been delayed. It does not therefore help me one way or the other. Dr Vadlamani had a similar problem because, as Mr Whitting pointed out, it was not clear from the section to which she referred how the authors had timed the insult relative to the neurological manifestation and whether the authors were describing the neuropathology in order to explain a gap between insult and clinical manifestation or not. She was not, as a paediatric neurologist able to take the point any further.

98. As to the negative finding on the cranial ultrasound at 42 hours of age (and 6 hours after the frank seizures). Dr Ives' and Dr Vadlamani's reminded me that the sonographer recorded that the ultrasound only showed "*difficult views*." However, this point falls away as the scans were subsequently reviewed by the consultant team and were confirmed to be normal. Dr Emmerson's view was that, although it may take a few hours for oedema to develop in the brain following a stroke, a postulated time interval of 42 hours is simply too long a period for no changes in the brain to appear on scanning if the stroke had occurred before the umbilical cord was cut. Although apparently neither Dr Ives nor Dr Vadlamani were prepared to accept this, neither recorded their view in their reports or in the joint note (even though it was an issue raised by Dr Emmerson in the joint meeting).

99. I am not able to square this particular circle. If I were to have to decide the difference between the parties on possibility of there being a time interval between insult and clinical or ultrasound findings, it seems to me that I would probably need evidence from a neuropathologist and a neuroradiologist. I don't decide the point and, as such, it does not count against the Claimant. My conclusion on medical causation is based upon what is known in the literature concerning the link between placental thrombus and PAIS and its application to the facts of this case.

100. This claim fails on both breach of duty and causation. I recognise that this conclusion will be a source of great disappointment to DE who, I appreciate, will have pinned her hope of an

easier life upon the outcome of this litigation. However, I find that the Claimant's disabilities have not been caused by any fault in her antenatal care and I dismiss the claim.