



Neutral Citation Number: [2025] EWHC 3345 (KB)

Case No.: KB 2023 001568

**IN THE HIGH COURT OF JUSTICE**  
**KING'S BENCH DIVISION**  
**ROYAL COURTS OF JUSTICE**

Date: 18<sup>th</sup> December 2025

**Before:**

**MR JUSTICE RITCHIE**

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**BETWEEN**

**OAJ**

**BY HIS MOTHER AND LITIGATION FRIEND CFT**

**Claimant**

**- and -**

**DORSET COUNTY HOSPITALS NHS FOUNDATION TRUST**

**Defendant**

**John de Bono KC** (instructed by **Lester Aldridge**) for the **Claimant**  
**Andrew Kennedy KC** (instructed by **Browne Jacobson**) for the **Defendant**

Hearing dates: 2, 3, 4, 5, 9 December 2025

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**APPROVED JUDGMENT**

This judgment will be handed down by the Judge remotely by circulation to the parties' representatives by email and released to The National Archives. The date and time for hand-down is deemed to be at 14:00 pm on 18.12.2025

**Mr Justice Ritchie:**

**The Parties**

1. The Claimant is now aged 14 and suffered very severe brain damage before or around his birth at the Defendant's Dorchester Hospital on 1.9.2011. His mother is his litigation friend. I made an anonymity order at the start of the trial because he is so vulnerable. This is a terribly tragic case and has been a prolonged heartache for his mother.

**Bundles**

2. For the hearing I was provided with 2 lever arch trial bundles, a core bundle of medical notes and two skeleton arguments. During the trial some additional documents were added to the trial bundle and the relevant CTG traces were copied larger, to help us all read them.

**Summary**

3. This was the trial of a preliminary issue relating to breach and causation. The relevant events happened between 23.18 pm on 31 August 2011 and 10.19 am on 1.9.2011, a period of 11 hours. All timings below relate to the night of 31.8.2011 and the morning of 1.9.2011. For reasons which were unclear at the time, the Claimant suffered Acute Profound Hypoxic Ischaemia in his mother's uterus and perhaps Chronic Partial Hypoxic Ischaemia as well, causing severe cerebral palsy (brain damage). The Claimant's case is that this injury should have been avoided by an earlier midwife referral to obstetricians, continuous CTG monitoring and earlier caesarean section, because his mother had complained of heavy bleeding at home when her membranes ruptured. The Defendant's case is that the standard of medical care given by the hospital staff was reasonable and, even if there was any breach, none caused the injury which would have arisen in any event.

**Terminology and acronyms**

4. I shall shorten some words using acronyms thus:

**C:** the Claimant.

**M:** the Claimant's mother.

**SRM:** Spontaneous Rupture of Membranes (the amniotic sack around the baby ruptures naturally and liquor is released through the cervix, into the vagina).

**APH:** Antenatal Placental Haemorrhage (a bleed from the placenta before birth).

**PA:** Placental Abruption (the coming away of a part of or all of the placenta from the uterus, causing bleeding).

**BSL:** Blood Stained Liquor (a mixture of amniotic fluid and a smallish amount of blood).

**FHR:** Fetal heart rate.

**CTG:** a machine which produces a trace which provides information on the heartbeat of the mother and the fetus.

**CTG Acceleration:** when the fetal heart rate goes up quite a lot and for a sufficient time to come within the definition (a good sign).

**CTG Deceleration:** when the fetal heart rate goes down quite a lot and for a sufficient time to come within the definition (a bad sign).

**CTG Baseline:** the mid-point of the fetal heart rate in beats per minute (the normal rate for a fetus is 110-160 bpm).

**CTG Baseline variability:** By how much the fetal heart rate goes up above and down below the baseline (normal is a variation of 5 bpm or more, up to 15 bpm).

**CTG Bradycardia:** very low fetal heart rate (under 100 bpm or under 80 bpm – a very bad sign).

**CTG terminology when M is in labour:** normal; suspicious; pathological.

**CTG terminology before M is in labour:** normal or abnormal.

**Augmentation:** induction of labour using drugs (in this case, either Prostin or Syntocinon).

**IOL:** Induction of Labour.

**CS:** Caesarean section (the operation to deliver the baby by surgery). There were two categories of CS: category I (a crash CS to carry out the delivery as soon as possible and in any event within 30 minutes); category II CS (with urgency but more safety precautions and in any event aiming to deliver within 75 minutes).

**APHI:** Acute Profound Hypoxic Ischaemia (the complete cessation of blood flow into the fetus).

**CPHI:** Chronic Partial Hypoxic Ischaemia (intermittent cessation of blood flow with intermittent resumption of blood flow, often associated with contractions).

### **The Issues**

5. There are two main issues: whether there was a breach of the duty of care owed to C by the clinicians and whether any such breach caused the brain injuries. The sub issues are as follows:

- 5.1 Did the midwife clinicians breach their duty of care to C at night?
- 5.2 Did Mr Siddig breach his duty of care to C in the morning?
- 5.3 What type of hypoxic injury did C suffer: APHI, CPHI or both?
- 5.4 When did C suffer the hypoxic injury? At nighttime or in the daytime?
- 5.5 What caused the hypoxia?
- 5.6 Did any breach cause or contribute to the brain injuries?

The parties provided a longer list of 18 issues. I do not consider that I need to list them here. The factual issues will be dealt with below.

### **The applications**

6. I granted permission for the Claimant to amend his particulars of claim at the start of the trial to regularise them with the final expert evidence which emerged quite late. This was unopposed.

**Pleadings and chronology of the action**

7. The claim was issued in June 2023. The amended particulars of claim (PoC) summarised the asserted chronology of events, including an allegation that on admission, midwife Coliandris failed to note M's complaint of a lack of fetal movement. There was no allegation made that midwife Coliandris failed verbally to take a history from M after admission. I raise that here because, as will become apparent below, that was one of the criticisms of the midwife put at length in cross examination. M alleged that she did not go to the toilet that night until between 02.40 and 05.15 am. She then did and reported to a midwife that she had seen more blood in the toilet. It was pleaded that no record of this report was made and no action was taken. (In fact, a record was made of M telling midwife Roger that she had been to the toilet and had seen fresh blood in the toilet at 08.35 am). C asserted negligence as follows:
- (1) The working diagnosis on admission was PA which mandated obstetric review, so midwife Coliandris was negligent in failing to do so.
  - (2) The Defendant was negligent in failing to offer augmentation (induction) soon after admission and it was a reasonable alternative treatment. If that had been offered it would have been accepted and continuous CTG monitoring would have been done with augmentation.
  - (3) Continuous CTG monitoring should have been done soon after admission in any event.
  - (4) After M reported going to the toilet and seeing fresh blood in the night, midwife Coliandris was negligent in failing to assess M and attach the CTG for continuous monitoring.
  - (5) At 08.48 am Mr Siddig was negligent in diagnosing a local source for the bleed. At that time, the 20 minute CTG trace was suspicious and recent fresh bleeding had occurred with an unfavourable cervix, so a category I CS should have been carried out.
  - (6) At 09.24 am Mr Siddig was negligent. The 40 minute CTG trace was suspicious. That, combined with the further visualised 100 ml of fresh blood and the unfavourable cervix indicated an evolving PA and mandated a category I CS due to the risk of PA.
  - (7) It was negligent to discontinue the CTG at 9.40 am.
  - (8) The 51 minutes of delay between the decision to do a CS and the delivery at 10.19 am was negligently long.
8. The Claimant pleaded causation thus:
- (1) The brain injury was caused by either APhi or CPhi. (It was not pleaded that it was caused by both.)
  - (2) The timing of the brain injury was either:
    - (a) nighttime (00.51 – 08.30 am); or
    - (b) daytime (09.37 – 10.19 am) at or near the start of that period.
  - (3) There was evidence of fetal compromise lasting until delivery.
- If it was a nighttime injury:

- (4) Had M been reviewed by an obstetrician soon after admission it is probable that the working diagnosis would have been PA and the plan would have been to accelerate labour (augmentation) and put continuous CTG in place.
- (5) Alternatively, M should have been offered immediate augmentation as a reasonable alternative treatment and M would have chosen that and hence obtained continuous CTG monitoring.
- (6) If there had been continuous CTG monitoring after 00.50 am that would have disclosed the emerging brain damage on the trace and led to a category I CS and earlier delivery. The failure to provide continuous monitoring caused or materially contributed to the brain injury.

If it was a daytime injury

- (7) If the decision for a category I CS had been made at 08.48 or 09.24 am the delivery would have been completed by no later than 09.54 am and would have avoided part or all of the brain injury, so the breach made a material contribution to the injury.
- (8) Had CTG monitoring been continued after 09.40 am, fetal distress would have become apparent, bradycardia would have arisen and a faster CS would have been achieved, preventing some of the brain injury.

9. In the defence, the Defendant recited their asserted chronology of events and denied breach of duty on admission. It was the Defendant's case that midwife Coliandris examined M after admission and put on a CTG trace and examined that. All the results for M and C were normal and reassuring. There was no pain, no continuing fresh bleeding, no tenderness to palpation of the abdomen, no tense or "woody" abdomen, no fetal distress and M's vital signs were normal. There was no evidence of a significant bleed or abnormality, just BSL. PA was not the working diagnosis, so medical review was not warranted. It was not standard in 2011 to offer induction until after 24 hours had passed. Conservative management was standard (so waiting 24 hours before induction). Continuous CTG monitoring was not warranted. The Defendant denied that M told the midwife between 02.40 and 05.15 am that she had seen fresh blood in the toilet. At 05.15 am the notes recorded "*no further bleed*". It was pleaded that at 08.48 am there had been "*no further bleed since admission*" (I mention here that this pleading was factually wrong because it overlooked the record made on 08.35 am of "*pad seen pink liquor/fresh small amount of fresh blood on pad. M reports losing more fresh blood down toilet*"). The Defendant went on to assert that no CS was mandated at 08.48 am because there was no pain, the abdomen was not tense and only 20 minutes of CTG had been seen (40 minutes were needed, see the 2007 Guidelines). For 09.24 am the Defendant pleaded that it was reasonable for Mr Siddig to decide to do a category II CS because there was a normal FHR baseline, no FHR bradycardia, only two shallow decelerations, reasonable variability and no clinical indication of a serious threat to M or C, so a responsible body of obstetricians would have chosen a category II CS. Stopping the CTG for the transfer to theatre was unavoidable in 2011. Monitoring in theatre was not routine in a general hospital in 2011. They generally used a Sonicaid at

that time. The CS was done in 51 minutes, which was within the guideline period of 75 minutes.

10. On causation the Defendant pleaded that the injury probably arose at nighttime, not in the daytime. This was because of: (1) C's condition at birth, which was not one evidencing severe hypoxia (the pH and base excess of the cord gasses disclosed this). C would have needed more than 10 minutes of restored blood flow to recover so well before birth. (2) Thus, if the injury occurred at night, any later breach by the consultant in the day was causatively irrelevant. (3) If induction was mandated soon after admission then Prostin would have been offered. No continuous CTG monitoring would have been provided with Prostin, only 40 minutes of CTG to check fetal wellbeing before and after giving it.
11. A request was made by C for further and better particulars of the defence and these were provided in February 2024. D explained that watershed brain damage would not be expected from APHI and the deep grey matter damage was more widespread than expected from APHI. On 13.11.2025 D served a notice to admit that the injury occurred during the night. No such admission was made.

### **The eye witnesses**

12. I heard or read the evidence from the following witnesses.

The Claimant's witnesses:

12.1 M.

12.2 No evidence was called from the Claimant's father who was present during the relevant 11 hour period.

The Defendant's witnesses:

12.3 Ms Jean Coliandris, treating midwife.

12.4 Ms Jane Hall, treating midwife coordinator (read).

12.5 Mr Siddig, treating consultant obstetrician.

12.6 Dr Fatima Shah, treating registrar in obstetrics (read).

### **Expert witnesses**

13. I heard or read evidence from the following experts.

The Claimant's instructed experts:

13.1 Ms Jean McConville, midwifery.

13.2 Mr Gerald Mason, consultant in obstetrics and gynaecology.

13.3 Dr Marcus Likeman, consultant neuro-radiologist (read).

13.4 Dr Gary Hartnoll, consultant neonatologist (read).

13.5 Dr Shakti Agrawal, consultant paediatric neurologist.

The Defendant's instructed experts:

13.6 Ms Susan Brydon, midwifery.

13.7 Professor Neil Thornton, consultant in obstetrics and gynaecology.

13.8 Dr Neil Stoodley, consultant neuro-radiologist (read).

13.9 Ms Janet Rennie, consultant neonatologist.

13.10 Dr Neil Thomas, consultant paediatric neurologist.

### **Historic witness evidence**

14. The events occurred 14 years ago. The evidence of the Defendant's witnesses was mainly about what they considered was a fair and reasonable interpretation of their own notes (and those made by other clinicians) and what they "would have" done as normal practice back then, not what they could actually recall of the events. Understandably, none of the treating clinicians could recall the events. Similarly, the evidence of M was generally that she could not recall the details of the events, however she did assert that she could recall the two bleeds which she suffered, one before going to hospital and the other in hospital. I acknowledge that it is inherent in many traumatic birth cases that some events become more illuminated in the minds of parents due to the trauma. I also take into account for all the eye witnesses that memory, being a human talent or function, is not perfect. On the contrary it may be degraded by time, enhanced by emotion, refined by repetitious discussion or lost or buried in part or in whole. In addition, it may be innocently reconstructed in part or in whole by discussion, events, emotion (for instance anger or sorrow) and time.

### **The chronology of events based on the medical notes and the Court's factual findings**

15. It will aid understanding of the chronology of events if I set out the contents of the medical notes first. I have expanded many of the acronyms or shortenings, to make the wording clearer. I have also added some of M's evidence to keep all of the alleged events in the chronology. I have also made some inferences and incorporated some of midwife Coliandris' and Ms Hall's evidence. The chronology below includes some, but not all, of my findings of fact made on the balance of probabilities. I shall explain later on why I have made these findings.
16. **Pregnancy.** M was born on 27.4.1982 and was aged 29 at the time of the events. This was her first pregnancy. She had suffered Chlamydia in the past but none of the experts stated that was relevant to any of the events. Her antenatal course was normal. The only irregularity was that in March 2011 clinicians noted a cervical erosion with some blood after peeing and a speculum examination showed a posterior cervical ectropion (ulcer) which bled on contact. She was noted as very anxious. The experts inferred that placenta previa was not seen at the 20-week scan. That means the placenta was not below the baby. Instead, it was positioned normally. M had taken iron tablets. When the pregnancy had lasted 40 weeks and 5 days the events below occurred.
17. **At home.** At night, on 31.8.2011, M was in bed and felt a pop and a tightening of her vagina and lower back. She went to her toilet and felt a "gush". She looked in the toilet and saw bright red blood. She described it in her witness statement as "*really heavy bleeding*". She called the hospital and spoke to midwife Grant-Jones, who noted at 23.18 pm (probably an inaccurate timing), M's recorded complaint was "*heavy bleeding*". M self-described as being very worried that she had lost her baby and was

extremely upset and probably shouted. She was advised to ring an ambulance and come in ASAP. A telephone note was made of this call. The ambulance service was called at 23.15 pm (which timing I find was accurate). The ambulance arrived quickly at 23.22 pm.

18. **Ambulance records.** The paramedics recorded M's history of events thus: *"lying in bed, felt a pop in vagina and lower back went to loo. Passed "some bright red watery blood"*. No clots, no pain. Checked on route, *very little blood*, very anxious, stating she cannot feel the baby move. That note partially undermined the accuracy of M's telephone account of heavy bleeding and her witness statement account of really heavy bleeding.
19. **Admission records.** The telephone call information was passed by midwife Grant-Jones to the co-ordinating midwife who allocated the emergency to midwife Coliandris. She then prepared a room for M and an emergency team was put on standby for a suspected PA, which is a major danger for mother and baby. M arrived by ambulance and midwife Coliandris spoke to the paramedics and made admission notes at 00.10 am on 1.9.2011. The special factors noted were "APH?" and "cervical erosion". The reason for admission was "APH/SROM." Blood stained liquor seen on the pad. No pain. Examination: Temperature: 36.8. Blood pressure: 118/82. M's pulse: 83. Baby's presentation: cephalic, OP, 4/5 FHR: 140 bpm. This reading was obtained using a Sonicaid and a Pinnard (both hand-held devices). She recorded that the Ambulance paramedics reported stable observations. She watched as M moved herself from the ambulance bed onto the ward bed. M looked well and reported no pain. Midwife Coliandris examined M's abdomen and found it soft and not tender. M was not actively bleeding. Midwife Coliandris inspected the pad and saw BSL on it (not fresh blood). Midwife Coliandris had therefore found that all of M's vital signs were normal and the baby had a normal FHR and was lying in a normal position. She had tested for clinical signs of PA and found none. I find that the examination was not done in silence. Midwife Coliandris spoke to M throughout. She decided to put a CTG on M's abdomen to monitor the FHR and M's heart rate and to observe contractions and fetal movement. At the start of the trace the FHR was 142 bpm. Midwife Coliandris' plan was for the CTG to run for 40 minutes (as required by the guidance at the time) and then to carry out a speculum of her vagina and to "review". The Claimant asserted that by "review" she meant review only by an obstetrician. Midwife Coliandris asserted that it was a review by herself. In my judgment "review" was probably wider than either assertion. It was review, first by the midwife and then, if thought necessary, by an obstetrician. Midwife Coliandris reassured M and gave her refreshments. She sent away or stood down the emergency team because she had not found any abnormal signs for M or C.
20. M's evidence was that midwife Coliandris was dismissive of her condition and her counsel cross examined on the basis that midwife Coliandris failed to take any history from M and failed to note any such a history from M. Midwife Coliandris asserted her normal practice would have been to take a maternal history and make a note of it but



she accepted that there was no express note that she had done so. However, her admission note covered all of the relevant information about the home bleed. M made no express complaint either in her witness statement or in her pleading that no maternal history was taken on admission. I find as a fact that the midwife accurately noted the events at home as: ?APH, and had read of the telephone note of “heavy bleeding” at home and took into account M’s complaints, which in my judgment she was likely to have verbalised to midwife Coliandris on or after admission, about her fears for the baby as a result of the heavy bleed at home and lack of movement of the fetus. I consider that the midwife did read the telephone note and the ambulance records which noted M’s concern about lack of fetal movement. I infer that she was aware of this complaint. She did not make a separate patient’s history note because all the relevant content was already in the admission notes, clinical notes, midwife Grant-Jone’s note and the ambulance notes. I reject the unpleaded allegation that the midwife failed to take a maternal history.

21. 40 minutes later, at 00.50 am, midwife Coliandris executed her plan. She made further clinical notes of her examination of M. She had described the risk as “APH” and the reason for admission as “APH/SROM. I find that she understood the difference and was determining into which category M fell. She noted irregular contractions, 2 every 10 minutes. She noted from the CTG that the FHR baseline was 140 bpm and showed good variability of >5bpm, with accelerations present and no decelerations. Overall, rightly, she concluded the CTG trace for the FHR was normal. She then carried out a speculum examination of the vagina which confirmed that M had achieved a SROM and there was BSL in the vagina. Her plan then was to remove the CTG, which had run the 40 minute course and let M rest. I find that by “BSL” midwife Coliandris meant the not uncommon BSL which arises with SROM. She was able to distinguish this from a significant amount of fresh blood mixed with amniotic fluid, which she would have described differently. The recorded plan was that if further blood loss occurred or labour started (an increase in contractions) then M should tell the midwife and the CTG would be placed back on and further review would take place.
22. **Clinical records in the night.** M slept on and off, in the presence of her husband, C’s father. Neither of them called the midwives. **At 02.40 am** midwife Coliandris reviewed M. She used handheld devices to check on C’s FHR which was noted as 140-150 bpm (normal) with no decelerations. She noted *no further blood loss*, but some BSL seen. Contractions remained at 2:10 and mild. M was trying to sleep. She noted C’s father was present and could use the call bell if they needed assistance.
23. **At 05.15 am**, midwife Coliandris again checked on M and C. M had slept for short periods. Small amounts of BSL were noted on her pad. *No further bleeding* was reported. M had felt fetal movements and was not in pain but did feel the tightening of her contractions. The midwife used hand-held devices to check the FHR which was 140-150 bmp (normal). M was left to sleep some more.

24. **Disputed toilet trip.** In her pleading M asserted that between 02.40 am and 05.15 am, when it was *dark*, she went to the toilet and saw more blood, she told the midwife and was advised not to flush next time and no record was made in the notes. In her witness statement M stated this toilet trip occurred between 00.50 am and 05.15 am. In her live evidence M asserted that she made the toilet trip as the light was coming up. For reasons which I shall explain later, I find that this toilet trip did not occur at this time, it occurred later, probably between 08.10 and 08.30 am (when the CTG was reattached). I reject the assertion that M told midwife Coliandris about this. I accept that she did tell midwife Rogers, who noted it at 08.35 am.
25. **Clinical records, morning handover.** The night shift changed to the day shift around 7.00 – 08.00 am. Midwife co-ordinator Jane Hall came on shift and midwife Rogers took over M’s care. At 07.50 am midwife Rogers spoke to M and she reported that her contractions had stopped. M’s pulse was recorded at 80 bpm, temperature 36.4 and BSL was reported as still dribbling out. The midwife asked M to keep the next pad for viewing before changing. During this discussion I consider it likely and infer that M moved her body around, so changed her position. This may be relevant to the cord compression theory discussed later. The notes record that midwife Rogers had a discussion with co-ordinator Jane Hall due to the BSL and the plan they decided on was augmentation. To effect that M’s consent was required so, at 08.10 am, midwife Rogers had that discussion with M. She chose Prostin instead of Syntocinon (Oxytocin) and the plan was that if the cervix did not efface or dilate then to start the Syntocinon later. It was also part of the plan to discuss it with the obstetric registrar. Co-ordinator Hall asserted in her witness statement that she wanted a medical review and induction. As a result of this plan, at 08.30 am the CTG was reattached to M’s abdomen. After that M could not go to the toilet. The immediate FHR baseline reading was 105-108 bpm. M’s pulse was 72 bpm. The FHR was abnormally low. I shall analyse the potential meaning of the FHR trace below when I come to the expert evidence.
26. **At 08.35 am** midwife Rogers examined M and inspected the CTG trace. The baseline had risen to 118 bpm with “no further decelerations”. M had kept her pad and the midwife saw a small amount of pink liquor/fresh blood on it and was told by M that she had been to the toilet and lost more fresh blood down it. In my judgment this explains M’s recollection of the toilet in the night.
27. **At 08.48 am** Mr Siddig, the consultant in charge, came to M. He gave evidence that he was just doing the ward round, but co-ordinator Hall asserted in her witness statement and her Datix report, that she specifically took him to M due to her concerns. I accept Ms Hall’s evidence on that. The registrar, Dr Shah, was there too. The notes were perfunctory. Mr Siddig had others write the notes for him. I shall in any event call them his notes. He noted BSL and diagnosed a local cause for the BSL as “likely”. The CTG was characterised as “suspicious”. There were contractions 1-2 in 10; the baseline was 135 bpm; variability was described as down (using an arrow); no accelerations were seen and no decelerations. His plan was to continue the CTG for 40 minutes and then

to start IOL after vaginal examination. I point out here that Mr Siddig made no reference to the fresh blood entry written a mere 13 minutes before this examination and he did not carry out any examination himself at that time. All experts agreed that the relevant local source (the cervical ectropion) would not have produced significant fresh bleeding, so he was wrong about the source.

28. There were two further examinations by midwives before Mr Siddig returned. The first at 09.05 am involved abdominal palpation of the fundus and determined the baby's lie. A speculum examination was done showing liquor ++ and *fresh blood per vagina*. The midwife was unable to visualise the cervix. Prostin was put in. At 09.15 am the CTG remained suspicious. The baseline had risen to 155 bpm but variability had reduced to under 5 bpm and there were still no accelerations. The plan was for co-ordinator Hall and the registrar to review, which occurred at 09.20 am.
29. **At 09.24 am** Mr Siddig was brought back. Again, the notes were written by others. The writing is poor. On speculum examination he saw 100 ml of fresh blood (50 ml is categorised as a major APH by obstetricians). The CTG was interpreted as showing reduced variability, baseline slowly rising, no accelerations and two shallow decelerations. His impression was: "*Placental abruption*". He decided at 09.28 am that M needed a CS, so she was consented and prepared. The Unit's CS record shows that Mr Siddig did not choose category I, which was listed as: "immediate threat to life of woman or fetus", he instead chose category II which was described as: "maternal or fetal compromise, not immediately life threatening".
30. **At 09.35 am** the anaesthetist was in the room and M was being prepared for theatre. At just before 09.40 am the CTG was stopped and soon thereafter M was moved to theatre. At "10.- am" the notes say "knife to skin". If that is right then the CS operation took 19 minutes – which may be a rather long time. None of the experts mentioned this or criticised the length of the operation, so there is no evidence for me to draw any conclusions about that. Dr Shah did the CS. She noted no abruption and only two small clots on opening the uterus.
31. **At birth.** C's APGAR scores were: 5 @ 1 minute; 7 @ 5 minutes and 6 @ 10 minutes. He weighed 3.7 kilogrammes. Regular respiration was established at 2 minutes. Initially baby C was given to M and all looked OK but after 10 minutes the damage which C had suffered started to become more apparent, so C was taken to the SCBU where later he suffered epilepsy. MRI scanning took place 7 days later. This disclosed very severe brain damage due to APHI and perhaps CPHI. C was eventually transferred to Southampton hospital for treatment due to the severity of his brain injuries. He was not given brain cooling.

### **The eye witness evidence**

32. **The Claimant's mother's evidence** in chief, in her witness statement dated 4.10.2024 was written 13 years after the event. I was not provided with any evidence of an early

complaint by M to the hospital. She had gone to A&E in March 2011 when she had suffered a bleed during her pregnancy and cervical erosion had been diagnosed. Her description in the witness statement of the bleed at home on 31.8.2011 was contradictory. It included two versions. She wrote that she saw “*some bright red blood*” in the toilet but also wrote that it looked like “*really heavy bleeding*” which was not watery. Both of these cannot be correct, unless her use of language is very broad. This led her to call the hospital, at which time M states that she was very upset so that she was screaming. As for the paramedics’ notes, she contradicted what they wrote: that she had told them she had seen “*some bright red watery blood*”. For reasons which I shall explain a bit more, lower down, on the factual issue of the volume of blood which M saw in her own toilet, I prefer the record in the paramedics notes to M’s recollection of the events. She was clearly very anxious indeed, because she was worried that her baby had died. Paramedics are professionals. If M had told them she had suffered a really heavy bleed I consider that they would have recorded that. On admission M asserted that there was no examination of her. Yet the notes state that at 01.10 am there was an examination and the results of that examination were clearly recorded. On this issue I prefer to accept the clinical notes. I consider that they are likely to be more reliable than M’s memory and I do not believe that midwife Coliandris made those notes up. There is no dispute about the contents of the notes made at 00.10 am as to the examination findings. M accepts that: she was well, had no pain and no tenderness to palpation. She felt that the midwife minimised her fears, but standing down the emergency team was not criticised by any of the experts. M asserts that, had she been offered induction soon after admission, she would have accepted it. M made no mention in her witness statement of the visit by midwife Coliandris at 02.40 am. M wrote that at some point between 00.50 am and 05.15 am, having not got out of bed before then, she went to the toilet, saw more blood and flushed it away. She then spoke to the midwife and was asked not to flush it away if that occurred again so the midwife could see it. I do not consider that this evidence is credible when compared to the notes which recorded, at 05.15 am, that there was *no further bleeding*. I consider that it is more likely that this bleed in the toilet and the report to the midwife occurred before 08.35 am, when it was recorded by midwife Rogers in the notes. Also, in her live evidence, M asserted that this occurred around sun-up, yet in the pleading she asserted it happened in the dark before 05.15 am. No party gave evidence of the sun-up time on 1.9.2011 but the timing contradictions in M’s memory on this push me towards the morning being the more likely time. M challenged the record in the note at 05.15 am that there was no further bleeding, but I prefer the record in the notes made at the time to M’s memory, so many years after the event.

33. M’s next piece of disputed evidence related to her criticism that Mr Siddig did not speak to her at all on his ward round at 08.48 am. She asserts that he only spoke to the clinicians in front of her and left her in the dark, then when he left, the midwife was the one who discussed induction with her. M asserts that had Mr Siddig told her that the trace was suspicious and offered CS she would have chosen that, as she did about half an hour later when he suggested CS at 09.28 am.

34. In cross examination many of M's answers were to the effect that she could not recall the detail. She remembered going to the loo when there was "dusky light" outside. She did not deny the examination on admission, she just could not recall it. M did not deny a conversation with the midwife about how the bleed started, she just could not recall one, but M also stated she beat herself up for not telling the midwife about the bleed at home. As for the decision to induce labour, M's recollection was she was not asked and yet she did consent to induction. As to the conversation, if any, with Mr Siddig during his ward round, M could not recall him talking to her. She did recall him talking about her to his colleagues in front of her but M did not recall his plan to continue the CTG and induce labour at that time.
35. **Nichola Coliandris**, the nighttime midwife, gave her evidence in chief in her witness statement dated 26.7.2024, 13 years after the events. She could not recall any of the events herself. She interpreted her notes and spoke of her normal practice. On admission M transferred herself to the ward bed and midwife Coliandris examined her and noted the examination results. She noted the special factors as "APH?" and "cervical erosion". She explained that her plan was to CTG, examine and review and that meant review by herself. Having found all the signs for M were normal, after the CTG of C's heart rate was also normal and after the speculum showed no fresh bleeding, midwife Coliandris decided to admit M and advised her that if she bled any more or started labour, to inform the clinical staff. She pointed out that fetal movements were shown as present on the CTG trace as black rectangular blocks. M was noted as getting up for the toilet at 00.50 am. There was no evidence of PA and cervical erosion could cause bleeding. In her clinical judgment there was no need to refer M to the obstetricians at that time. The reviews at 02.40 and 05.15 am were also normal. There was no more fresh blood loss, no pain, there was fetal movement and a good FHR.
36. Under sustained and professional cross examination, midwife Coliandris did not change her evidence. She was a grade 6 midwife. She accepted that bleeding which is significant is potentially dangerous and a sign of PA. She was expecting an emergency admission with maternal collapse and the need for an emergency response. On M's arrival the facts were quite different. She was aware that women may be unable accurately to estimate the volume of blood loss. She had dealt with PA before and was aware of the risk of worsening. She would have called for obstetric review if any of her observations indicated the need. She accepted that her notes did not state she had taken a history from M but she believed that she had done so because the whole relevant history was recorded on the admission sheet. She accepted that the key issue was how much blood M had lost at home, but on what the paramedics recorded, her own examinations and the CTG, she did not consider M to have suffered a PA. She accepted she did not note M's concern about no fetal movements on admission but that was in the ambulance records and she had the CTG graph showing fetal movements within 10 minutes of arrival. Also, her normal practice was to ask. The key to her evidence was that she examined M, did a speculum, read the history, looked at the CTG and if there

had been anything abnormal she would have asked for medical review. But there was nothing abnormal, despite M's statement to midwife Grant-Jones. She did not accept the assertion put to her that she was ignoring M's self-report as irrelevant. Midwife Coliandris stated that the guidelines were that she was to do an assessment and if anything was abnormal she should call for obstetric review. M's report of bleeding made the case for assessment, it did not make the case for automatic review. She accepted that she herself had not identified the source of the bleeding, but some bleeding was not uncommon with SROM. For M, she only ever saw BSL not fresh bleeding. She inspected the pads at 02.20 and 05.15 am. There was no further bleeding. If M had reported further bleeding she would have noted it.

37. **Midwife co-ordinator Ms Hall** gave a witness statement in September 2024. She was not called. She went to work at 07.00 am on 1.9.2011 and the doctors came on shift at 08.30 am. She allocated midwife Rogers to M and was keen to have M reviewed for consideration of induction. After Rogers reviewed M at 07.50 am she spoke to Ms Hall about the BSL. Ms Hall considered augmentation to be a reasonable plan and asked for a discussion with M and then with the registrar. M agreed Prostin augmentation. When Mr Siddig saw M the CTG had been running for just under 20 minutes. It was difficult to interpret. Mr Siddig decided to continue the CTG and examine M later and assess the cervix and then consider starting induction. Ms Hall explained this to M. Soon thereafter she asked the registrar to review the trace and at 09.20 am Dr Shah decided that a CS was required. Mr Siddig saw M at 09.24 am and quickly chose a category II CS.
38. Ms Hall also completed an incident report (Datix) on 13.9.2011 in which she wrote, contemporaneously, that she had specifically taken Mr Siddig to M at 08.45 am because of her concern about the BSL and the CTG. She and Dr Shah had decided that CS was needed at 09.20 am before Mr Siddig returned and then he decided it was a category II case.
39. **Dr Fatima Shah** provided a witness statement dated August 2024. She did not give live evidence. She was the registrar who saw M in the morning and did the CS. She accompanied Mr Siddig on his ward round. She made the notes. When called to review the CTG at 09.20 am she decided that CS was needed but a consultant had to confirm that decision. There was no need for a category I CS because there was no heavy bleeding (I find that she was wrong about the bleeding). The CTG machine was taken into theatre because that was trust practice and she did not know why it had not been reconnected. She did the CS and noted no abruption. She gave no explanation for the 19 minute length of the operation.
40. **Mr Siddig** was employed by the trust when he wrote his witness statement in September 2024 and still is now. He is the lead consultant in obstetrics at the Dorset County hospital in Dorchester. He wrote that he obtained "DM" in 1977 and "MD" in 1985. He had no recollection of events. He summarised his notes of the ward round at

08.48 am and asserted that there were no confirmed signs of active bleeding, so he did not consider that APH was present. Stopping there, both in 2011 and in his witness statement he had completely overlooked the entry in the clinical notes timed at 08.35 am describing M seeing fresh bleeding in the toilet and the midwife seeing fresh blood on a pad. He asserted that M had not mentioned any fresh blood since her arrival. That assertion was also factually incorrect because the note made at 08.35 am. Despite the CTG being “suspicious” and displaying what he stated was “reduced variability” of the baseline FHR, he decided to wait for more CTG trace, to do a later examination and then to review and start induction. He was called back by Ms Hall and Dr Shah at 09.24 am, when he did a speculum and noted 100 ml of fresh blood. He reviewed the CTG, which showed no accelerations and two shallow decelerations and decided it was not “pathological” but only “suspicious”. He applied the 2007 NICE Guidelines. There was no bradycardia, there were no prolonged decelerations, according to him there was “no active bleeding”, no abdominal pain and she had a soft abdomen, so his impression was possible mild PA and there was no immediate threat to the life of M or C.

41. In cross examination, Mr Siddig admitted that he had provided a verbal witness statement to the trust in 2011, the notes of which had not been disclosed (if there were any). He denied that co-ordinator Hall took him over to M especially before his ward round at 08.48 am because the notes showed it was not an acute case. He repeated that there was no active bleeding at that time. When asked about the note made 13 minutes before his round which recorded active bleeding he stated that he had not seen that note and was not aware of the contents. I found that answer particularly unimpressive. He asserted that had he seen active bleeding it would have been recorded but he did not carry out any examination of M’s vagina or pad on that ward round. He could not explain why he did not examine M for fresh bleeding at that time. As for being called back to M at 09.24 am he stated that he did examine then and found 100 ml of fresh blood so suspected PA. He accepted that in neither of his sets of notes was there any record of him taking a history from M. He asserted that he would have done so and “sometimes they do not take a note”. As to his decision at 08.48 am that the cause of the bleed was a local one (cervical erosion) he relied on the “normal CTG”. (I find that it was abnormal both at 08.48 and 09.24 am, as Mr Siddig later admitted). He did not check for cervical erosion himself before me made that diagnosis. Then he made a rather unimpressive attempt to widen the definition of “local cause” to cover APH, placenta previa and PA. When cross examined on the daytime CTG at 08.48 am he accepted it was very unusual to have a baseline starting at 105 bpm and steadily rising over 30-40 minutes. He then asserted that it was normal and became quite confused about what he was saying. Eventually Mr Siddig accepted that he had used the wrong terminology (that for a woman in labour, when M was not in labour). He then asserted that the CTG was abnormal (the correct terminology for a woman before labour). As for the note made of his ward round at 08.48 am, which stated the variability was “reduced”, he asserted that he had not written the note and that was *not* what he thought. He asserted that M was in early labour but then asserted that M was contracting at 1:2. He was challenged on that because the notes recorded 1:10, so he withdrew that

statement. It was put to Mr Siddig that a CS was required at 08.48 am because: (1) M had suffered heavy bleeding at home and a fresh bleeding at 08.35 am and (2) the CTG trace for C was abnormal. He rejected this, considering that the CTG was not pathological with no prolonged decelerations and a normal baseline and so his plan for induction was correct and he wanted to wait for a longer CTG trace.

42. When asked about 09.24 am Mr Siddig again became confused. He asserted that he did not examine M then, but in fact he did a speculum then. He maintained that the bleed found at 09.24 was not an acute bleed and M and C were not in danger from an acute event. He advised that the cause could have been a leak behind the placenta or mild abruption. Mr Saddig admitted that he was not happy that once the CTG was removed at 09.40 am it was never put back on in theatre. With the CTG abnormality, he accepted that monitoring should have been continued or should have been reconnected ASAP. He explained it would take 20 minutes to transfer M to the theatre. He did not ask any theatre staff to reconnect M to the CTG. At the end of his evidence he could not really explain why he had ignored M's report of fresh blood down the toilet at 08.35 am.

### **Assessment of eye witnesses**

43. I consider that all of the factual witnesses were doing their best to assist this Court. With the events so long ago the evidence from memory is likely to have been degraded and somewhat coloured by the passage of time and emotions. M's evidence was honestly given, but I found the detail of it a little contradictory with the contemporaneous notes. Where it departs from the medical records I prefer the records.
44. As for the evidence from the clinicians, I was impressed by midwife Coliandris, who I consider was a straightforward and honest witness doing her best to assist me without guile or defensiveness. I was concerned by Mr Siddig's evidence. He was muddled, confused and ill-prepared. He was unaware, when he wrote his witness statement and at trial, of one key element of the contents of the core bundle of medical records: the fresh bleeding entry timed at 08.35 am. His answers in cross examination were contradictory and confusing and he tried to redefine certain words in an unimpressive way. His grasp of the facts was weak and on occasions wrong. I regret to say that I do not consider that I can generally rely on his evidence.

### **Guidance**

45. The relevant guidance provided to the Court by the experts is set out below. No Guidance from the Royal College of Nursing was provided. The Dorset County Hospital NHSFT Maternity Service Guideline for IOL was issued in 2009. It stated that IOL should be offered to all women at term +10 days. M was at term + 5 days. Women with high risk or complicated pregnancies should not be offered induction at the weekend unless there are compelling clinical reasons. 1.9.201 was not a weekend day. After using the Bishop's score assessment of the cervix (which was done for M) Prostaglandin was recommended as the best agent for cervical ripening. CTG for 20 minutes before such use and 40 minutes after was recommended. Prelabour rupture of



membranes was listed as a special situation occurring in 6-19% of pregnancies. The stated risks were: infection and prolapsed cord. Most women were recorded as going into labour within 24 hours of rupture. After that, the women should be given the choice of IOL or more conservative management. Continuous CTG was only recommended with the use of Oxytocin.

46. The Dorset Hospital Preterm and Term Labour Rupture of Membranes Guideline was issued in April 2011. In that the hospital advised that prelabour rupture of membranes (at 37 weeks or more) arose in 8-10% of all pregnancies. Infection was one of the most important aetiologies resulting. The requisite initial assessments were set out. Advice was given that: with good cephalic engagement of the baby and no additional risks of infection or complications, such as meconium stained liquor, and no indication for CS, conservative management for 24 hours or release home was appropriate.
47. The Dorset Hospital Guideline on CS was dated January 2010. Emergency CS was to be done when the fetus or mother were in a life threatening situation. There were 4 categories of CS listed. Category 1 was for immediate threat to life. Category 2 was for maternal or fetal compromise which was not immediately life threatening. 3 and 4 are less urgent. CS1 should occur within 30 minutes of the decision. It stated: *“for any emergency CS, continuous fetal monitoring must be maintained in theatre until the operation begins, so the monitor should be moved to theatre with the woman.”* The majority of CS were done under regional anaesthetic (spinal).
48. The NICE Guidelines on Intrapartum Care were dated September 2007. Prelabour rupture of membranes was covered at paragraph 1.10. The advice was to use speculum examination to confirm and to warn the mother of the risk of SROM, including infection. The advice stated that IOL becomes appropriate after 24 hours from the SROM. FHR should be assessed at the initial contact and then every 24 hours following rupture whilst the mother is not in labour. CTG trace classification in labour was set out. The 4 factors needed to be normal (reassuring) were: Baseline (110-160 bpm); Variability (equal to or over 5 bpm); no decelerations and present accelerations. The other definitions were: non-reassuring and abnormal. Antepartum haemorrhage was listed as a risk factor for postpartum haemorrhage.
49. The NICE Guidance on Inducing Labour was issued in July 2008. This stated that most women go into labour spontaneously by week 42. Women with prelabour rupture of membranes at over 37 weeks should be offered IOL with Prostin (PGE) or expectant management. IOL is advised as appropriate after 24 hours. One cycle of Prostin takes 6 hours or 24 hours depending on the type. Continuous CTG was recommended “when contractions begin” but then stopped if normal and reduced to intermittent auscultation. The woman may return home after Prostin is given with clear advice to return when contractions begin. On cord prolapse, the guidance stated that to reduce the likelihood of cord prolapse at the time of amniotomy (breaking the membranes) assessment should

be done of the baby's position and palpation for the umbilical cord should be done and a check should be made for a low-lying placenta.

50. The RCOG Green-top Guideline no. 63 on Antepartum Haemorrhage was issued just after the events, in November 2011. It advised that APH is bleeding from or into the genital tract after 24 weeks of pregnancy. The most important causes are placenta previa (low lying) and placental abruption, but those are not the most common. Other causes are "local ones: (bleeding from the vulva, vagina or cervix). The Guidance states that it is not uncommon to fail to identify the cause for APH. APH complicates 3-5% of pregnancies and is a leading cause of mortality. The RCOG categorised APH as follows:

"There are no consistent definitions of the severity of APH. It is recognised that the amount of blood lost is often underestimated and that the amount of blood coming from the introitus may not represent the total blood lost (for example in a concealed placental abruption). It is important therefore, when estimating the blood loss, to assess for signs of clinical shock. The presence of fetal compromise or fetal demise is an important indicator of volume depletion. For the purposes of this guideline, the following definitions have been used:

Spotting – staining, streaking or blood spotting noted on underwear or sanitary protection

Minor haemorrhage – blood loss less than 50 ml that has settled

Major haemorrhage – blood loss of 50–1000 ml, with no signs of clinical shock

Massive haemorrhage – blood loss greater than 1000 ml and/or signs of clinical shock.

Recurrent APH is the term used when there are episodes of APH on more than one occasion."

The RCOG Guidance recommended that women presenting with APH should be managed in a maternity unit and a multidisciplinary team, including obstetric staff, should provide clinical assessment. It did not say how quickly that MDT assessment should take place. The Guidance then went through the type of assessment by clinicians for women presenting with APH (at para 7). This included taking a history, assessing the presence of pain, palpation of the abdomen for a tense or woody feel which indicates abruption, speculum examination, assessing vaginal bleeding and the mother's vital signs and the fetal signs with CTG. The guidance focussed on massive APH and maternal collapse and resuscitation. The Guidance noted that diagnosis of placental abruption was a clinical diagnosis and there *was no reliable diagnostic test*. The Guidance stated that women presenting with spotting, with no placenta previa, no further bleeding and with reassuring clinical assessment, can go home. All women with

APH heavier than spotting and with ongoing bleeding should stay in at least until the bleeding has stopped.

## The expert evidence

### Midwifery

51. **Jean McConville** reported in December 2024. She is a well qualified expert and her credentials were not challenged. She retired from NHS work in 2015. She laid her conclusions out in an odd way, referring to each particular of negligence from the PoC one by one and then giving her advice on each. Usually, the pleadings follow the expert's report and the conclusions therein. Her main conclusion was, (1) that midwife Coliandris should have arranged medical review soon after admission and failing to do so was a breach of duty. This was because M had complained of heavy bleeding at home which gave rise to a risk that she was suffering an APH, so that should have been the working diagnosis. This, she advised, matched midwife Coliandris' initial plan: to examine, CTG and then "review", which Ms McConville considered meant review by a registrar. This expert advised that, because midwife Coliandris gave no other working diagnosis than "APH?", despite saying in her witness statement that she did not consider that there was any evidence of APH, she should have asked for medical review. In the conclusion, at the end of the report, Ms McConville stated that the midwife should have referred M for medical review and assessment shortly after admission because "*she had been admitted with continuing fresh bloodstained liquor, and the midwife queried APH.*". I should say straight away that there was no evidence in the medical records that midwife Coliandris saw any continuing fresh blood. What the paramedics and she saw was "very little blood" on M's pad. Ms McConville also advised, (2) that midwife Rogers was in breach at 08.35 am for failing to ask for medical review after noting the fresh blood on M's pad and hearing her report of fresh blood in the toilet. I struggle to understand why she advised that because medical review took place 13 minutes later at the request of the midwife coordinator Ms Hall to whom midwife Rogers had spoken.
52. Ms McConville relied on r.6 of the *Midwifery Rules 2004* which, she asserted, advised that if there is anything out of the normal – call a Doctor. Rule 6 of the *Nursing and Midwifery Council Rules 2004* actually provided:

"(3) In an emergency, or where a deviation from the norm which is outside her current sphere of practice becomes apparent in a woman or baby during the antenatal, intranatal or postnatal periods, a practising midwife shall call such qualified health professional as may reasonably be expected to have the necessary skills and experience to assist her in the provision of care."

That is not quite the same as the summary provided by Ms McConville. In evidence the Defendant's midwifery expert and Ms Coliandris considered that assessment of SRM and associated BSL bleeding on admission was within the role (sphere of practice) of a midwife.

53. Midwife McConville asserted that “*the midwifery texts, both state, that in any case of APH the midwife must seek a medical opinion*” (p23). She relied on advice in *Fraser & Myles on Midwives* 2009, which set out the risks of APH after “severe vaginal bleeding”. Also, on the advice in *Mayes Midwifery* 2004, at p777, which she asserted stated that vaginal bleeding is always “abnormal”; and at chapter 54 (no copy provided), which she asserted advised that conservative treatment for vaginal bleeding is not appropriate after 38 weeks of pregnancy. I have reviewed the text books attached to the report. I regret to say that not all of the parts of the text summarised in the report are provided in copy format so that I could read them in their context. This is not good practice by Ms McConville. *Myles Midwives* 15<sup>th</sup> ed (2009), at pages 336-337, define APH and stress that severe APH increases the risk of fetal and maternal morbidity and mortality. The editors state that when local causes are excluded, “vaginal bleeding in late pregnancy is due to placental separation from the placenta previa or a placental abruption” referring to table 20.1. In fact, that table set out some 2006 figures extracted from another textbook, stating that 31% arise from placenta previa, 22% from “abruption” and 47% from “unclassified bleeding” (most of which are caused by “marginal” and some of which are caused by “the show”). As to the initial appraisal of women with APH at admission, the text advises support and taking a good history, then making a rapid decision on urgency *and any need for medical review*. When taking a history, the circumstances of the start of the bleed should be ascertained; the colour of the blood; the degree of maternal shock; the consistency of the abdomen; the lie of the baby. The advice on the assessment of the mother’s physical condition covered: palor, breathlessness, emotional state, pulse, respiratory rate, blood pressure, temperature. The midwife was advised to assess the amount of blood loss; examine soiled articles; do abdominal palpation; carry out observation of contractions; ask questions about pain; but not to do a VE initially. Also, the health of the fetus should be examined by taking the FHR and asking about movement. What the book did not advise was referral of all mothers with any suspected APH whatever the examination findings or whatever the mother’s reported volume of bleed. The pages provided from *Mayes’ Midwifery* 13<sup>th</sup> ed (2004) (p777) relate to management of massive obstetric haemorrhage, not minor, moderate or major. The pages are not put in context, so do not help. The page from *Mayes’ Midwifery* (2010) 14<sup>th</sup> ed related to treatment in hospital on the delivery suite. No copies of any other pages were provided.
54. In the joint report with Ms Brydon, Ms McConville stuck to her advice that, because APH was the working diagnosis and because it is unpredictable, medical review was mandatory after admission due to the BSL seen on the pad from the ambulance and the maternal history of the heavy bleed. The origin of the bleed needed to be identified. She conceded that using CTG was the correct way to measure of fetal wellbeing, not the maternal report of not being able to feel movement in the ambulance. She agreed that, on admission, the midwife needed to do the checks, all of which she did. She advised that the obstetrician would decide what to do after the review.

55. In cross examination Ms McConville accepted that once the amniotic liquor is made blood stained it stays so. She stated that it becomes lighter over time. As to medical review after admission, she accepted that it would not have been urgent for M to be reviewed. The fetus was stable and M was stable. Despite what she said in her report, she accepted that it was not for the midwife to decide the source (where the bleeding was coming from), that was for the Doctor. She asserted that midwife Coliandris assessed that the bleeding came from the ectropion and asserted that she was not entitled to decide that M was wrong in her account of the volume of her bleeding. It was put to her that the joint obstetric report contained the agreed opinion that obstetricians did not expect midwives to send all mothers with APH for review, only significant ones. Ms McConville agreed, but then qualified that agreement asserting the midwife Coliandris had “decided” that the bleeding was from a source external to the uterus and had not believed M. Ms McConville admitted that BSL, alongside SROM and the show, were not uncommon. Ms McConville accepted that it was difficult for mothers to estimate the volumes of blood in amniotic liquor at home when looking in a toilet bowl. She also accepted that M’s bleed at home history was not consistent with the words she had said to the paramedics. She accepted that it was only if the midwife saw a larger amount of blood that she would need to do something about it. BSL does not usually contain a large amount of blood. She accepted there was no active bleeding on admission and there was none thereafter until 08.35 am, just BSL on pads. She accepted that none of the midwifery textbooks required the midwife to seek the source of the bleeding at the time of the SROM. She agreed that examination and observations could rule out massive APH but not minor or major APH, which could be symptomatically silent. At the end of cross examination Ms McConville accepted, without caveat, that not all APH cases mandated review by a Doctor. If the presentation was SROM and BSL, no review was mandated. If there was heavy bleeding, with an anxious mother and an ambulance delivery to hospital, then medical review was mandated, even with stable observations for mother and baby. That was based mainly on M’s report of the volume of bleeding at home. Ms McConville did not criticise the 13 minutes taken by the midwives to get Mr Siddig over to M after 08.35 am and the reported bleed at that time. She did continue her criticism of the midwives in failing to reattach the CTG in theatre.
56. **Ms Brydon**, was instructed by the Defendant. She edited *Myles on Midwifery* between 2004 and 2019, whilst in practice. In her evidence in chief in her report she advised that the assessment on admission was carried out to a reasonable standard. Medical review would only have been required if there was continuing, active bleeding, which there was not. In particular, the evidence in the ambulance notes was consistent with SROM not PA. M was not in labour, so it was reasonable to await labour starting. BSL accompanying SROM was not uncommon. M’s initial phone call indicated heavy bleeding so it was right to call her in urgently, but the ambulance records did not support heavy bleeding. If there had been heavy bleeding, the ambulance crew would have expected a mother in shock, collapsed, with blood in the vagina and streaking down her legs. The pad was checked in the ambulance and there was very little blood. The midwife then looked at the pad and identified only BSL. M was moved onto the bed

and no fresh bleeding emerged. Her vital signs were temporarily elevated, consistent with anxiety, not heavy bleeding. This settled in 20 minutes. There was no evidence of maternal collapse. The paramedics and midwife were more likely to be correct in their interpretation of the volume of blood in the liquor than the anxious mother looking into a toilet. It was reasonable to stand down the emergency staff and start the CTG. There was a normal CTG trace after admission, there was no active bleeding and so no continuous monitoring was required. The examinations did not reveal a tense abdomen or pain or reluctance to move. The FHR was normal. There were no abnormal signs. As for the asserted toilet bleed in the night she advised that, if this occurred, medical review was required. If it was merely BSL, then advice to retain the pads was enough.

57. In the joint report Ms Brydon stuck to her opinions for the same reasons. She stressed that midwife Coliandris knew of the suspected diagnosis of APH (she wrote it down) and searched for signs but what she found did not support APH. If any of the findings had been abnormal then medical review would have been needed, but none were. If there had been a heavy bleed from the uterus, then fresh blood would have been visible during the speculum examination. Instead, all that was found was SROM and BSL. M's account to the ambulance paramedics supported SROM and BSL not APH. Rightly, the midwife wrote as a special reason for admission: "APH?" but it was not made out. Ms Brydon advised that continuous monitoring was not usually done overnight without evidence of bleeding and this only arose at 08.45 am (actually 08.35 am). Medical review was mandated then.
58. In cross examination Ms Brydon disseminated in some of her answers and had to be pulled back to the question a few times. When challenged on the lack of a note made by midwife Coliandris of M's own account of her bleed at home, she stated that she would have expected questions to be asked of M during the examination. She did not accept that the midwife had to seek information about the volume of blood and seriousness of the bleed at home, she asserted that women could not estimate those. Reports of heavy bleeding were common, but midwives had to make their own assessment. Midwife Coliandris knew very well what the emergency was about, an emergency team had been gathered because of a report of a heavy bleed. The concern over APH was clear. She would not accept that it was substandard to fail to ask M about events at home because midwife Coliandris had all the information she needed from the records of the call to midwife Grant-Jones and the ambulance records. The latter disclosed no fresh bleeding, despite M being moved onto the ambulance bed and later off it. The pad only showed BSL. The history was more consistent with SROM and BSL resulting therefrom. Ms Brydon did agree that failing to ask about fetal movement would be substandard. Ms Brydon was taken to a paragraph in her report in which she had written that the history taken from M was consistent with SROM. It was put to her that there was no written history taken from M. She apologised for that misstatement in her report. This forensically well-prepared cross examination was effective, but was on an unpleaded complaint. Ms Brydon explained that it was clear from the admission records that midwife Coliandris was aware that M had asserted a heavy bleed at home

and had written that “?APH cervical erosions” were the special factors on admission. The heart of Ms Brydon’s evidence was her statement that the history from M of a heavy bleed was not enough to warrant medical review, it was the midwife’s job to assess M and C and the assessment was reassuring and consistent with SROM and BSL not APH, so medical review was not mandated. She advised that midwives are gatekeepers for onward referral to obstetricians.

59. Ms Brydon asserted that Ms McConville initially believed that M had suffered a haemorrhage, but after tests and examinations she considered that there was no evidence of haemorrhage. Counsel took Ms Brydon to the RCOG Greentop Guidance (No 63) which defined the categories of Antepartum Haemorrhage. This described the causes of APH as including: placenta previa, PA and local causes. It stated that it was not uncommon for clinicians to fail to identify the cause so it would be described as “unexplained”. The categorisation are set out above in my Guidance summary. Ms Brydon accepted those obstetric definitions but maintained her advice that the midwife had to assess M and C. M was well, the baby was shown to be well on the CTG trace. There was only BSL on the pad and no fresh blood. Speculum disclosed no fresh bleeding. There was no blood on M’s bed, in her vagina or on her legs. The clinical signs did not support the complaint of APH. There was nothing to report to the obstetricians. Ms Brydon assured the Court that if midwife Coliandris had done something wrong she would have criticised her, but she did not. She accepted that the midwife did not identify the source of the bleeding. She asserted that in her experience it was very unusual to have no tenderness after a PA. She ended her evidence in cross examination accepting that after the further bleed was noticed at 08.35 am a medical review was needed and that took place. She agreed that the CTG was abnormal from 08.30 am and explained that after the CTG was disconnected around 09.40 am it would have been difficult to put it back on when the spinal anaesthetic was being carried out.

## The Law

### Standard of care

60. When considering breach of duty and the standard of care in this case in relation to both the midwives’ clinical care and the obstetricians’ I have applied the principles set out in *Bolam v Friern Hospital* [1957] 1 WLR 582, by McNair J, addressing a jury thus:

“... I must tell you what in law we mean by “negligence.” In the ordinary case which does not involve any special skill, negligence in law means a failure to do some act which a reasonable man in the circumstances would do, or the doing of some act which a reasonable man in the circumstances would not do; and if that failure or the doing of that act results in injury, then there is a cause of action. How do you test whether this act or failure is negligent? In an ordinary case it is generally said you judge it by the action of the man in the street. He is the ordinary man. In one case it has been said you judge it by the conduct of the man on the top of a

Clapham omnibus. He is the ordinary man. But where you get a situation which involves the use of some special skill or competence, then the test as to whether there has been negligence or not is not the test of the man on the top of a Clapham omnibus, because he has not got this special skill. The test is the standard of the ordinary skilled man exercising and professing to have that special skill. A man need not possess the highest expert skill; it is well established law that it is sufficient if he exercises the ordinary skill of an ordinary competent man exercising that particular art.”

And

“A man need not possess the highest expert skill; it is well established law that it is sufficient if he exercises the ordinary skill of an ordinary competent man exercising that particular art ... in the case of a medical man, negligence means failure to act in accordance with the standards of reasonably competent medical men at the time.”

61. I also have taken into account the ruling of Lord Browne-Wilkinson in *Bolitho v City and Hackney HA* [1998] AC 232:

“In the *Bolam* case itself, McNair J. [1957] 1 W.L.R. 583, 587 stated that the defendant had to have acted in accordance with the practice accepted as proper by a ‘responsible body of medical men.’ Later, at p. 588, he referred to ‘a standard of practice recognised as proper by a competent reasonable body of opinion.’ Again, in the passage which I have cited from *Maynard’s case* [1984] 1 W.L.R. 634, 639, Lord Scarman refers to a ‘respectable’ body of professional opinion. The use of these adjectives - responsible, reasonable and respectable - all show that the court has to be satisfied that the exponents of the body of opinion relied upon can demonstrate that such opinion has a logical basis. In particular in cases involving, as they so often do, the weighing of risks against benefits, the judge before accepting a body of opinion as being responsible, reasonable or respectable, will need to be satisfied that, in forming their views, the experts have directed their minds to the question of comparative risks and benefits and have reached a defensible conclusion on the matter.”

### **Assessment of the midwifery standard of care**

#### **Further findings of fact**

62. On the balance of probabilities, taking the eye-witness evidence and all the expert evidence into account, I find that M had a spontaneous rupture of membranes at home on 31.8.2011 and this came with some fresh blood. I cannot determine where the blood



came from. M saw the blood stained liquor in the toilet when the blood was fresh and bright and it really frightened her. She called the hospital and made her report of heavy bleeding. When the ambulance arrived the paramedics asked her what had happened and she explained using the words “some bright red watery blood” about what she had seen in the toilet. When the paramedics checked the pad on route there was very little blood. When midwife Coliandris checked her pad the same applied and she described it correctly as blood stained liquor (BSL). When midwife Coliandris carried out a speculum at 00.50 am she again saw BSL, not fresh blood. I find as a fact, on the evidence before me, that once amniotic fluid is blood stained, after SROM, when it trickles or leaks out, it usually remains so, albeit the colour changes over time.

### **Breach**

63. I was greatly assisted by both midwifery experts who were of high professionalism and focus. Their opinions were also logical, from their points of view. However, Ms McConville mistakenly wrote that there was continuing fresh bloodstained liquor on admission. I find that there was no “fresh” BSL, only BSL. In her report, she considered that the maternal report of heavy bleeding at home was enough to mandate medical referral despite: (1) what M had said to the paramedics (“some bright red watery blood”); and (2) all the examinations and assessments of M and C being reassuringly normal; and (3) the difficulty for any mother in estimating blood volume in liquor in a toilet after SROM. I consider that Ms McConville stretched the meaning of the extracts from the text books which she relied upon a little further than they actually went. Not one of the texts stated that medical referral was mandated where SROM and BSL have arisen, against a background in which all of the examinations, signs and the CTG are normal, just because a mother had reported a heavy bleed at home. Ms Brydon advised that the reason for doing all the examinations and assessments was to determine whether M or C was at risk of having suffered an APH at home and the results did not support APH. Instead, the results supported SROM (which was confirmed on speculum examination) and BSL, which was common with SROM. I take into account that estimating the volume of blood in a pint glass or a half pint glass full of amniotic fluid would be difficult enough. Then, if one pours that down a toilet bowl, with more water at the bottom, it seems to me that to expect accurate volume of blood estimation by a very scared mother would be fanciful in many cases. Nor do I accept that it is the midwife’s task to determine the source of the bleed. She may be able to do so, but she may not. Overall, once Ms McConville had shifted her opinion in cross examination, I consider that the difference in opinions between the experts represented a reasonable range of opinions for midwives in practice in 2011. Thus, where Ms Brydon advises that midwife Coliandris was not negligent for failing to refer M on to a registrar after admission, I consider that a reasonable body of midwives would have acted as midwife Coliandris did, so she was not in breach. I should make clear that in my judgment, Ms Brydon’s opinion at least represents that of a small but reasonable body of the profession and more probably represents the standard of care of the majority in 2011, because it was more logical and better matched the advice in *Myles Midwives* 15<sup>th</sup> ed (2009) at pages 336-337. Having carried out every proper assessment and examination,

having noted that all the signs came back normal for both M and C, in the context of mere BSL on the maternal pad and taking into account the great difficulty for mothers accurately to assess the amount of blood loss in a toilet after SROM, furthermore taking into account the paramedics' note of what M told them (some bright red watery blood), referral to a registrar at 00.50 am was not mandated and might, quite rightly, have led to a raised obstetric registrar's eyebrows. Certainly, Professor Thornton would not have expected to have been called.

64. As for the actions of the morning midwives, midwife Rogers was involved in the call for obstetric review via her coordinator, Ms Hall, very soon after 08.35 am and that is what the midwifery experts both agree she should have done, so I consider that she was not in breach of her duty of care. Therefore, I find no breaches of duty by midwives Coliandris, Rogers or Hall up to that point. As for the asserted failure to reattach the CTG trace at theatre I will deal with that below under obstetric care but accept that any breach I may find involved the midwives.

### **Obstetric experts**

65. **Mr Gerald Mason** was instructed by the Claimant. In his report dated December 2024, he opined that there was no evidence of abruption or placental separation at delivery so that was not the cause of the bleeding before birth. He postulated that the bleed came from the edge of the placenta - a marginal bleed or minor PA. This, he advised, was not the cause of the hypoxia. So, another cause had to be postulated. C was well grown in utero and there was no evidence of placental failure. He advised that the risk of cord compression increases after SROM and that changing the maternal position could relieve it. Without fetal monitoring cord compression could have arisen unnoticed. He therefore concluded that there had been a period of cord compression before 08.30 am causing APHI, from which the fetus was recovering when the CTG was put on at 08.30 am. On breach, he advised that, given the maternal history, the midwife should have referred M to an obstetrician after admission. This was on the basis set out in the RCOG Green-top Guidance of November 2011 which defined the categories of APH. M had described a heavy bleed which would indicate a "major APH" (a bleed of between 50 and 1000 ml of blood, with no signs of clinical shock) and he relied on the Green-top advice requiring MDT assessment for APH. Stopping there, a few points arise. Firstly that was RCOG advice not RCN advice. Secondly, in my judgment, there is a difference between "no signs of clinical shock" and no adverse signs at all for either M or C after assessment. Thirdly, there were two midwifery experts instructed in the case, so he was trespassing outside his own area of expertise. He also criticised midwife Coliandris for failing to identify the source of the bleeding which he said was a breach of duty (although he accepted he was stepping outside his field of expertise in so advising). He advised that examination by a Doctor would have shown BSL coming through the cervix. The working diagnosis would have been PA and the only option would have been to accelerate labour with full monitoring. Even if induction was not chosen it should have been offered. On causation, he advised that CTG monitoring all night would have identified fetal distress and led to CS avoiding hypoxia. In relation to the

daytime care, he advised that the CTG was abnormal. There was a slowly increasing baseline from 105 bpm to 160 bpm and a spikey appearance with no accelerations but normal variability. After 40 minutes the trace showed reduced variability and decelerations and no accelerations. The working diagnosis, with the fresh bleed and unfavourable cervix, was APH which required a category I CS.

66. **In the joint report** with Professor Thornton, Mr Mason stated that the ambulance records indicated a minimal amount of bleeding but if the Court accepted M's description to midwife Grant-Jones in her phone call, that would have been a major APH of over 50 ml. In response, Professor Thornton stated that it would have been difficult for M to assess the volume of blood at home. Watery blood suggests bleeding associated with the SROM. There were no clots and that indicated bleeding which was not excessive and was under 50 mls. The lack of pain indicated no PA. There was minimal liquor on the pad in the ambulance, so there were no findings from the ambulance records to indicate APH. They both agreed that the report of lack of fetal movement would be not a concern. The paramedics had noted her concerns and both experts advised that the CTG trace indicated fetal movement soon after arrival. On arrival at hospital Mr Mason set out four possible causes for the bleeding: low placenta (ultrasound had discounted that); a local cause; PA or bleeding from the cord. The normal FHR indicated it was not a cord bleed, so APH was the most likely diagnosis. He advised that some blood loss in labour is not unusual, but it should not persist. He advised that, each time that BSL was noted that showed that the bleeding persisted and so was significant. Professor Thornton stated that BSL was of little consequence, was common and was not associated with adverse consequences (relying on a paper by *Gluck* covering 21,000 births). He advised that BSL is different from fresh blood leaks. Mr Mason stuck to his view that medical review was mandated after admission if the midwife could not identify the source. He relied on the RCOG Green-top Guidance. Professor Thornton advised that medical review was not mandated and there was no reason, after the reassuring assessments of both M and C, for the midwife to call a Doctor, but deferred to the midwifery experts. If there had been significant blood loss (>50ml) then obstetric review was appropriate, in accordance with the local guidance. He advised that the cause of the bleed could not have been determined by the midwife by speculum examination. The examination and the CTG excluded abruption, the uterus was soft and not tender, there was no significant bleed and M's blood pressure and pulse were normal, as was the fetal CTG. Mr Mason agreed that PA was a clinical diagnosis but advised that it can occur without pain or tenderness and the absence did not exclude PA and that any PA could evolve. They agreed that the source of the bleed was not identified. In retrospect, Mr Mason advised that it came from the edge of the placenta. Professor Thornton could not determine where it came from. Mr Mason went on to advise that, after admission, if it was established that the bleeding came from the uterus, the options were: ongoing monitoring and immediate induction, because it came from the placental edge and could progress. Although Mr Mason accepted that M was not in labour, he relied on the RCOG APH Guidance on labour, which required continuous monitoring of women in labour with vaginal bleeding. Stopping there, I do not see how

that advice helped me, because M was not in labour. Professor Thornton advised that M did not have signs of APH, so the options were to send her home or admit her for conservative observation. There was no indication for continuous monitoring at that time. In any event, immediate induction, in 2011, meant induction within 24 hours.

67. In his verbal evidence in chief Mr Mason accepted that he had shifted his position from his report. He stated that he and Professor Thornton agreed that after SROM, with only BSL, and reassuring signs, there was no requirement for the midwife to refer M to a Doctor, but significant blood loss would require such. Significant blood loss meant more than an egg cup full. He described the daytime trace as very unusual, he had never seen one like it before. He advised that the fact that the baseline recovered from 105 bpm to 160 bpm over 40 minutes suggested fetal recovery after an insult. In cross examination, he pointed out the two shallow decelerations later on the daytime trace and considered that they were probably associated with contractions. He confirmed his opinion that the daytime trace showed a recovering fetus. As for fetal movements on the daytime trace, M was interposed to give further evidence during Mr Mason's cross examination but she could not confirm that she had used the button to record fetal movement. Returning to Mr Mason, he was prepared to alter para 43 of his report to state that induction, if chosen after admission, would be within 24 hours and would not have been immediate. He accepted that if the registrar had been called and had chosen induction with Prostin, then continuous CTG would not have been applied. It would only have endured for 40 minutes and then, if the trace was normal, it would have been detached until contractions started. He accepted that midwife Coliandris did all the relevant tests and examinations at admission. He accepted that there was nothing more that a registrar could have done. He accepted that all the results pointed away from PA. He advised that the examinations did not exclude PA. He agreed that, on speculum examination, if all that was seen was BSL or "pink" BSL, that would not concern him, however if the midwife saw "frank blood", that would be a cause for concern. He accepted that, after birth, Dr Shah looked for evidence of abruption but found none. In any event he advised that abruption is not reversible, so the brain injury was not caused by abruption. He advised that, unusually in this case, there were two pathologies at work. Because SROM raises the risk of both PA and/or cord compression, in this case it was the latter which caused the brain injury not the former. He advised that at 08.48 am the recent fresh bleeding and the unusual CTG mandated CS, due to the risk of PA. He was asked why he advised that a category I CS was mandated when there was no immediate threat to M or C's health. He relied on the risk to their health from multiple bleeds, however he accepted that a category II CS would have been an acceptable choice so long as continuous monitoring was ensured or reconnection to the CTG in theatre was effected. He stated in re-examination that the 100 ml seen by Mr Siddig could not have come from a local cause.
68. **Professor Thornton's** evidence in chief, from his report dated January 2025, included his main opinion that the obstetric management was reasonable. He strayed into the midwifery experts' area passing comments on the admission and midwifery

management. He asserted that M's telephone report of heavy bleeding at home did not match the ambulance records of what she told them had happened at home. He advised, from the obstetric perspective, that there was no reason to ask for medical review based on BSL with SROM, no pain, no tenderness to palpation, a soft abdomen and a reassuring CTG for M and C, alongside no frank bleeding in the ambulance and on admission. He advised that abruption caused pain and tenderness, frequent contractions and fetal compromise. Maternal blood loss causes increased maternal heart rate and reduced blood pressure. None of these signs was present. He asserted that he would not expect to be called by a midwife to see a mother with SROM and BSL, when all the signs were normal. He advised that APH was not the working diagnosis at 00.50 am, because there was no evidence of PA. M was not in labour. It was not standard practice to do continuous CTG in those circumstances. He relied on the hospital protocol and national guidelines, which gave guidance that a mother who is not in Labour, with minimal bleeding, could be sent home, if all of the signs on CTG and examination were normal. Even if induction had been chosen, that would not have occurred at night and the local guidelines recommended starting with Prostaglandin (Prostin) and 40 minutes of CTG, not continuous CTG. As for the toilet bleed in the night assertion, he advised that if it occurred then a CTG, an examination and transfer to the delivery unit for induction would be required. As to the "ward round" by Mr Siddig at 08.48 am, he advised that the CTG was not an indication for urgent early CS and because there had been *no frank bleeding* since admission there was no indication for a CS. That statement was a fundamental defect in Professor Thornton's report. He completely overlooked the immediately preceding clinical note at 08.35 am of *fresh blood* on M's pad and her report of *fresh blood* in the toilet. This was highlighted in cross examination, to which I will come lower down. He focussed on Mr Siddig's examination at 09.24 am. Professor Thornton restated that, before Mr Siddig examined M at that, time there was "*no evidence of fresh bleeding*". That was likewise factually incorrect. He agreed with Mr Siddig that the CTG was "suspicious", although he pointed out that Mr Siddig had used the wrong terminology. There were shallow decelerations. When Mr Siddig inspected the vagina and saw 100 mls of blood Professor Thornton agreed that the working diagnosis was APH and a rapid CS was required. He advised that a category II was appropriate. He did not think that the CTG indicated the need for a category I CS, because the baseline was normal, the variability was reasonable and there was no bradycardia. He stated that neither it, nor the maternal signs, indicated an emergency or a threat to life. He advised that delivery within 51 minutes was reasonable. He deferred to the midwifery experts on whether CTG should have been reconnected. I found that advice odd. Once the consultant had decided on a CS due to fresh bleeding and a suspicious trace, knowing that the CTG would be disconnected for transfer, knowing that the local hospital policy was to take the CTG into the theatre, I do not understand why he took the view that the obstetricians should divest themselves of responsibility for ensuring the necessary FHR monitoring. He expressly stated that he did not criticise the lack of resumption of CTG monitoring. On causation, Professor Thornton deferred to other experts but passed his opinion anyway. He advised that the cause of the brain injuries was APHI or CPHI and the injury probably did not arise immediately before

birth because of C's reasonably good condition at birth. He advised that the chances of recovery from serious brain injury in the few minutes before delivery were "vanishingly small". Oddly, at ps 21- 22 in his report, in answer to PoC allegation "r", about what continuous CTG during the night would have shown, he wrote that "*there is no reason to believe that the CTG would have become pathological.*" I reject that opinion as contradictory with his opinion that the severe brain damage occurred during the night (by which I mean before 08.30 am). In answer to PoC allegation "u" he wrote: "*it is logical to assume that the pattern developed sometime between discontinuing the admission CTG and commencing it at 08.30 hours. Given that there was no definite indication of fetal compromise at 08.30 hours and thereafter, on balance, an earlier CTG would not have led to immediate delivery by*" CS. I reject that opinion as well. On the basis of his own evidence Professor Thornton advised that the serious brain injury arose at night. He rather grudgingly accepted, at the bottom of page 22 of his report, that "*there may have been evidence of fetal compromise*" if CTG had been continuous. Near the end of his report he advised that there was "no indication" for delivery at 08.48 am and stated that bleeding commenced at 09.24 am. But he was ignoring the *fresh blood* entry at 08.35 am. Finally, he did not accept that the cause of the serious brain injuries was cord compression causing APHI.

69. In the joint report with Mr Mason, in the early pages Professor Thornton stuck to his opinions. He stated that, in relation to admission with SROM and BSL, the RCOG Guidelines of 2001 had been superseded by the NICE Guidelines of 2007, which, (at p206), advised offering immediate induction, which meant within 24 hours. However, if there had been a significant APH, his "personal practice" (which he explained, in cross examination, meant the standard of professional practice in 2011) would have been to expedite delivery and undertake continuous CTG. Commenting on the night CTG trace, he stated that the normal variability of the FHR baseline on that trace showed the C's sympathetic and para-sympathetic systems were in-tact. As for the daytime CTG, he stated that, although the variability was normal, it was spikey and decreasing and there were no accelerations. This was unusual but not pathological. In retrospect, that spikey variability may have represented damage to the sympathetic and parasympathetic systems and he advised that the brain damage to C arose between the nighttime and daytime CTGs. Having rejected cord compression for a daytime injury in his report, in the joint statement he partly changed his mind. He agreed with Mr Mason that they could not say what the precise mechanism of injury was, but if it was cord compression it would have had to have been for long enough. C's condition at birth was an indication that there was no ongoing severe hypoxia or acidemia. Thus, it must have been a reversible compression. As for other possible causes, these included maternal positioning (vena cava compression), but all were unusual and abruption was certainly not the cause, because it is not reversible. As for the timing of the insult, they agreed that an APHI would not have arisen after 09.37 am because C's condition at birth was too good. It would have been much worse and there would have been a more marked arterial/venous cord gases differential. Therefore, night injury was more likely. Professor Thornton also changed his opinion on whether a CTG overnight would have

disclosed fetal compromise. Mr Mason advised that it would have and would have led to a fast CS. Professor Thornton accepted that there would have been CTG changes leading to expedited delivery. I ask myself why he did not say that in his report. Mr Mason advised that the day trace showed a recovering fetus after injury. Professor Thornton asserted that was an over interpretation. Mr Mason advised that if induction had been offered after admission, continuous monitoring would have been imposed. Professor Thornton advised that would not have occurred with Prostin, only with Oxytocin. Both experts criticised Mr Siddig's diagnosis made at 08.48 am, of a local source for the bleeding. The most unimpressive part of Professor Thornton's joint report was his advice on the events at 08.48 am. Despite Mr Mason pointing out to him that there had been "fresh bleeding" he ignored that and wrote: "*There was no indication to undertake a caesarean at 08.48 .... There were no symptoms and signs of evolving abruption... it was only at 09.05 hours when the PG was inserted that there was vaginal bleeding*". I do not know whether these experts actually had a discussion or just exchanged draft written comments, but this disconnect shows how one expert can mislead himself by overlooking a key entry in the clinical notes, despite it being referred to by the other expert. Professor Thornton accepted that, by 09.25 am, the decision to do a CS was necessary due to the visualised blood. As for the need for continuous CTG after that decision, Mr Mason advised it was necessary but Professor Thornton completely avoided answering the question of whether it should have been reconnected. Both experts added helpful notes at the end of the joint report. They agreed that, with significant and obvious APH, M should have been given continuous CTG or immediate induction (not in 24 hours). With SROM and mere BSL there was no reason for her to be reviewed by an obstetrician or to have continuous CTG or immediate induction. They also agreed that the injury occurred between 00.50 and 08.30 am. They disagreed on the longevity of leaking BSL after SROM. Mr Mason considered the staining to be short term. Professor Thornton advised BSL staining remained long term.

70. In his live evidence in chief Professor Thornton advised he did not consider that M had any abruption but warned that there had been no placental histology. He pointed out something about the maternal heart rate, which was not visible on the copy of the daytime trace provided to the Court, but was shown in Janet Rennie's enhanced copy in her report. In cross examination he advised that a mother would usually have a total of 5 litres of blood. A major haemorrhage would be a bleed of 50ml or more, up to 1000 ml. If a mother lost 1 litre of blood that would produce an increased pulse and reduced blood pressure. She would look pale and be shocked. Her pulse would increase. He was questioned on midwife Coliandris' alleged failure to take a note of the home bleed history from M after admission and accepted no note had been made of any bleed volume estimation. When challenged on his opinion about 08.48 am, he accepted that he had overlooked the entry at 08.35 am stating *fresh blood* on M's pad and flushed away in the toilet. He accepted that her account was consistent with a significant fresh bleed in hospital. I was unimpressed by his answers about the decisions made by Mr Siddig at 08.48 am. He criticised Mr Siddig for diagnosing a local cause for bleeding without examination, but would not accept that the 08.35 am note of fresh blood on the

pad and down the toilet was “significant” enough to mandate immediate examination and a CS. He accepted that the daytime CTG was very unusual due to the spikey baseline variability and the baseline rising steadily from 105 to 160 bpm and the decelerations. He advised that, at theatre, restarting the CTG trace would only be relevant before the spinal anaesthetic was inserted. As for the daytime CTG being evidence of previous serious brain injury, he advised that he had looked for literature on baseline variability after serious brain injury. He advised that if the brain centres controlling the parasympathetic and sympathetic systems had been damaged then variability would be affected. He accepted that the spikey baselines variability was potentially evidence of injury but did not know. He accepted that if C was suffering cord compression he would expect to see major FHR changes on the CTG (something he had partially rejected in his report). He was unable to determine the cause of the injury. It could have been cord compression or compression of M’s vena cava. In further questioning on the maternal heart rate he postulated reasons for it going up at admission and up around the time of the CS decision.

**Assessment of the obstetric standard of care**

71. I consider that both of the obstetric experts were very experienced and were doing their best to assist the Court.
72. Mr Mason went outside his field of expertise when advising the Court that midwife Coliandris breached her duty of care because she should have referred M to obstetricians and should have identified the source of the bleeding, soon after admission. That was for the midwifery experts. In any event, Mr Mason altered his opinion on referral to an obstetrician from a blanket mandation, due to M’s self-report over the telephone, to a requirement only if there was evidence of significant blood loss as opposed to SROM with BSL. Professor Thornton made a serious error in his report and in the joint report when he overlooked the fresh blood seen by the midwife at 08.35 am and the report by M of fresh blood in the toilet in the same note. He also strayed outside his field to comment on midwifery practice. In relation to the allegations of breach relating to midwifery practice, I prefer to rely on the midwifery evidence and I have done so above.
73. However, I do take into account the obstetric evidence in relation to the causation issue about whether, had an obstetrician been called after admission, anything other than conservative management would have been advised. On that issue, I consider that the evidence of Professor Thornton about the period after admission is probably what would have happened. On the balance of probabilities, I find that, had an obstetrician been called at say 00.50 am, he/she would have preferred the ambulance records of what M had said to the paramedics instead of her report over her phone, made in high anxiety. He/she would have seen minimal BSL on the pads and in the vagina. I take into account and find that, had an obstetrician been called: the CTG trace would have been the same; the maternal signs would have been reassuringly normal; M would not have been in labour; the fetal signs would have been normal; the examination results



for palpation of M's abdomen would have been pain free and reassuring, pointing away from APH and towards SROM and BSL. Thus, in my judgment it is unlikely that induction would have been offered by the obstetrician. Even if induction had been offered, I find that "immediate induction" in 2011 meant: within 24 hours. I consider it unlikely that Syntocinon would have been offered first, or accepted by M. The Dorset Hospital Guidance recommended Prostin which would have been chosen and started. It is unlikely that would have been started soon after midnight. It is more likely that would have awaited the day shift. Even if it had been started at 00.50 am Prostin required only 40 - 60 minutes of contemporaneous CTG. In the unlikely event that induction had been started at around 00.50 am, it would probably have shown nothing sinister, because the FHR was 140-150 bpm at 02.40 am.

74. As for the obstetric management at 08.48 am, I accept the evidence of both expert obstetricians that Mr Siddig was in breach of his duty of care when diagnosing a local cause for the bleeding. He did not even try to visualise the suspect ectopic. I also find that he negligently overlooked the note, made a mere 13 minutes before his ward round, setting out that fresh blood was seen on M's pad and M's report that she saw fresh blood in the toilet. I find that Mr Siddig should have examined M at that time and would probably have seen fresh blood in her vagina. That, combined with the odd trace, starting with a low baseline of 105 bpm (normal 110-160 bpm), the spikey variability and the lack of any accelerations, should have led to a decision for a CS. I accept Mr Mason's opinion that, at that time, the working diagnosis should have been a suspected PA. As a result, I find that a CS was mandated. Professor Thornton never really addressed the whole of the facts at this timepoint and carelessly ignored the fresh bleed report at 08.35 am so I do not find his opinion helpful on this timepoint.
75. The next issue on obstetric management is whether Mr Siddig should have decided on a category I or a category II CS at 08.48 am. I accept the local guidance on the hospital CS form was that a category I was reserved for cases where there is an identified immediate threat to the life of M or C and I add the expert's evidence that it also included a threat of serious injury to either. The CTG did not show that level of threat at 08.48 am. There were no decelerations. There was adequate baseline variability (5 bmp or above). There was no fetal bradycardia. The baseline was at 138 bpm. The maternal CTG and signs did not indicate heavy bleeding. Although Mr Mason advised that it should have been a category I at that time, in cross examination about the later time of 9.24 am, he accepted that it would have been reasonable to choose category II with continuous FHR monitoring. That seems to me to apply equally to the earlier time. So, I hold that it would have been adequate practice for a category II CS to have been chosen. CTG would have continued for a few minutes as M was prepared to theatre and then disconnected. This finding applies equally to the allegations of breach made in relation to 09.24 am.
76. The next issue related to midwives reconnecting the CTG to monitor the FHR after transfer to theatre. I find, on the expert evidence from Mr Mason (Professor Thornton

ducked this issue), and following the Dorset Hospital Guidance, that although the CTG had to be removed to transport M to theatre, it should have been reconnected ASAP at theatre or regular manual FHR measurements should have been done. I consider that the failure by midwives to measure the FHR at all after 09.40 am by CTG or by manual devices, was negligence by both fields of the clinicians. The clinicians reasonably believed that there was an emerging risk of PA which required urgent CS. That risk needed to be monitored. If the placenta did abrupt more seriously, both the mother and C would have been put at serious risk of death or injury in a far shorter period of time than 39 minutes.

### Causation

77. **Doctor Likeman and Doctor Stoodley, the neuro-radiologists**, examined the MRI scan of C's brain taken 7 days after the birth and the CT taken four months after. They concluded that the severe brain injuries C had suffered included damage to his: Posterior Putamina; Ventro Lateral Thalami; Peri-rolandic White Matter; Cortex; Cerebral White Matter; Grey Matter; Basal Ganglia and Thalami. They agreed that the pattern showed APHI. They differed on whether the pattern disclosed CPHI as well. Doctor Stoodley thought it showed evidence of CPHI at the watershed regions whereas Doctor Likeman opined that the damage was all explained by and attributable to APHI. As to duration of the APHI, Doctor Likeman estimated between 15 and 25 minutes but probably at the very upper end, near 25 minutes. Doctor Stoodley agreed that it would be nearer to 25 minutes if it was all caused by APHI but was unable to estimate the duration if it was CPHI. In his original report, Doctor Likeman stated that there was no definitive evidence of a CPHI injury. Doctor Stoodley stated in his report that it would have been better to have an MRI at age 2. Both experts advised that APHI becomes damaging generally after 10 minutes and that after over 25 minutes the fetus is unlikely to survive. Doctor Stoodley considered the injuries were too extensive to be purely APHI. They both deferred to other experts on the timing of the HI events.
78. **Doctors Janet Rennie and Gary Hartnoll were the neonatologists** instructed to report on causation and injury. In their joint report they agreed that C's condition at birth was slightly depressed. His acidosis readings were probably recorded the wrong way around so were likely to have been: Arterial: 7.01 venous: 7.08. I should mention that "arterial" for a fetus means the blood vessels coming out of the fetus and "venous" means the blood vessels going into the fetus from the mother's placenta. The difference was 0.07. In his report, Doctor Hartnoll had noted that C was handed to his mother for 10 minutes after birth which indicated his condition did not look too bad at that time, but it then deteriorated. He postulated that C's condition at birth indicated earlier injury, not one arising just before birth. He theorised that the FHR was recovering at the start of the daytime CTG. He postulated that M had probably changed her body position thereby unknowingly allowing resumption of blood supply. He considered that daytime APHI was possible but unlikely. Nighttime APHI was more likely. He asserted that if the FHR had been monitored between 05.15 and 08.30 am, the bradycardia would have been evident. Doctor Hartnoll was not called. Doctor Rennie reported (in January 2025)

that, at birth, there were no significant signs of acidosis and there was no bradycardia. So, she theorised, a recovery must have taken place and the duration of that recovery would have been longer than the 3-10 minutes pleaded in the PoC under the Agrawal, daytime injury theory. The relevance of that is as follows:

- If the injury occurred in the daytime it must have started after the end of the daytime CTG: at around 09.40 am.
- The duration of the APhi will have been near to 25 minutes, so at the earliest it will have ended at 10.05.
- C was born at 10.19 so the recovery period was 14 minutes at most.

Doctor Rennie stated that C's base deficit after birth was 10.7. She only had Doctor Stoodley's radiology report when she reported, so accepted a mixed cause for the brain injuries: APhi and CPhi. She advised that the blood results of M and C ruled out cord bleed as a cause. She postulated that, because M was not in labour, cord compression was less likely. She considered the rate at which C's body and M's placenta might have been able to clear acidosis. She researched and found studies by *Frasch 2009* and *DeHaan 1997*. She summarised them (slightly inaccurately) as showing that recovery in the live sheep tests, from a cord gasses pH of less than 7, took 2 hours and return to the normal baseline took 24 hours. She stated that base excess recovery occurred at an average of 0.09 mmol per minute. In fact, when one looks at the graphs in the *Frasch* paper, (BP 1087) the pH recovery (the lower right hand side graph) showed recovery from pH 6.85-6.95 to around pH 7.05 in about 30 minutes. So, to that extent, it did support her theories, but she overstated the conclusions. The graph, after 2 hours, is of no help because the scale is completely wrong. Overall, Doctor Rennie opined that C's perinatal history was "much more suggestive" of prolonged CPhi, and on balance the injury occurred between 05.15 and 08.30 am.

79. In her live evidence in chief Doctor Rennie relied on Doctor Thomas' estimate of the pH of C's cord gases at its nadir being 6.6-6.8 and the baseline deficit rising to 16 or above. In cross examination, she stressed her special interest in brain injuries. She accepted that this is a complex and difficult case. She summarised that she preferred to advise that it was a night insult not a daytime insult, which she thought would have been "extremely improbable" due to the recovery time needed to reach C's birth condition. She would have expected a large cord pH reading difference of 0.3 to 0.4 between the artery and the vein if the hypoxia had been recent. It was only 0.07. She postulated that in HI cases the fetus excretes acidotic blood and the placenta buffers and cleans it (if the cord is patent). But the clearance rate is limited. She accepted that a flat CTG can be seen in fetuses with already severely damaged brains before birth. She stated there was no precedent for this case. Usually, severely damaged fetuses die before birth. She agreed that it was possible that brain stem damage from the night injury caused disfunction of the sympathetic and parasympathetic systems affecting the variability of the daytime CTG trace. She tended to the view that the night injury would have occurred closer to 08.00 am than to 05.15 am. Doctor Rennie accepted that the

sheep experiments used very small samples. *De Haan* used 21 and 7 were controls. *Frasch* used only 10 and one died.

80. The **paediatric neurology** evidence was provided by **Doctors Shakti Agrawal and Neil Thomas**. Doctor Agrawal postulated in his report that the damage occurred in the daytime. He concluded that the insult ended 3-10 minutes before delivery and then recovery occurred. He considered that the length of the APHI was probably around 25 minutes. He postulated that the injury started at around 09.44 am. He favoured this theory because he had never come across severe brain injury and then good recovery in utero, whereas he said that he had “many times” come across events matching his theory. He asserted that his theory was more “plausible”. He postulated:

- 09.40 am CTG off.
- 09.44 am APHI starts.
- 10.09 am APHI stops.
- 10 minutes of recovery until birth at 10.19 am.

He listed the possible causes of APHI, all of which did not apply in this case, save for cord occlusion.

81. In cross examination by video (he was in the USA) he agreed that his theory was predicated on C suffering mainly APHI, with CPHI being only minor and in the background. He said that it was very difficult to distinguish between the two for severe injuries. It was more possible to differentiate between them with moderate injuries. He accepted that if the APHI had run up to birth then C would have been profoundly more sick. He would have had a FHR below 100 bpm; would have needed significant resuscitation; his blood gasses would have been more profoundly acidotic; he would have need brain cooling and his APGAR scores would have been much worse. Doctor Agrawal accepted that C deteriorated about 10 minutes after his birth. He would not accept counsel’s efforts to get him to agree that C’s blood gasses would have lowered to pH 6.6 or 6.8 at the end of the insult period. He accepted only a pH below 7.0. He explained his theory thus: he relied on Professor Thornton asserting that the daytime CTG was technically normal. There was, he said, “no indication that the baby was in trouble” at 08.30 am. He said that it was very difficult to reconcile that normal CTG as being from a fetus with a catastrophic brain injury. The severity of injury to C would have caused a complete severance of the sympathetic and parasympathetic nervous systems, resulting from damage to the Thalamus and Basal Ganglia. This would produce a complete loss of variability on the CTG trace. The rise from 105 bpm to 160 bpm indicated that the sympathetic nervous system was intact. He interpreted the trace as it neared 09.40 am, with two decelerations and the variability reducing, as approaching the time of injury. He opined that it was demonstrating sluggish physiology, not damaged physiology. Counsel pointed out that none of this explanation was in his report or the joint report. Counsel put to Doctor Agrawal that interpreting the trace was a matter for the obstetricians, not for him and all the obstetricians and neonatologists had advised the time of injury was before 08.30 am. Doctor Agrawal

stated that his hypothesis was based on the obstetric evidence that the APHI was caused by cord occlusion, then relief through position change. He then described the process of damage to brain cells from APHI. There are 3 stages: (1) primary HI event damage; (2) a latent period; then (3) a secondary energy failure, causing more damage. C was born in the latent period and then decompensated about 2 hours later, so that C had seizures and was sent to SCBU. This followed the 3 phase model. He accepted that cooling the brain could reduce the secondary energy failure damage within 4-5 hours of the birth and so prevent some further damage. He described the secondary damage phase as “devastating” and arising at around 4 hours. He was asked at the end of his evidence whether he could apportion the percentage of brain damage in phase 1 from that in phases 2 and 3 but he could not do so, which rather undermined his assertion that, if all the brain damage had been caused by APHI before 08.30 am, the CTG trace would have flat lined. He also admitted that he had never seen his theory occur in clinical practice, only in his medico-legal cases.

82. In his report, **Doctor Neil Thomas** described the two main types of damaging HI as: (1) APHI, lasting 10-30 minutes, damaging the Basal Ganglia and Thalami and normally producing dyskinetic or dystonic CP, often with preserved cognition; (2) CPHI, lasting over an hour, normally causing spastic quadriplegia and learning difficulties. Relying on Professor Thornton’s evidence, he opined that the injury occurred in the night and relying on Doctor Stoodley’s evidence, he opined that the cause was mixed APHI and CPHI. He then went on to state that it was unlikely that the injury occurred through cord compression (which was a matter outside his expertise). I found his report to be little more than a bucket carrying opinions provided by other experts.
83. In the joint neurology report, Doctors Agrawal and Thomas agreed that C’s cord arterial pH was 7.01 and the original base deficit was -11.3. They agreed that his condition at birth was poor but not catastrophic. They disagreed on when the hypoxic event occurred. Doctor Agrawal favoured just before birth and Doctor Thomas favoured between 00.50 and 08.30 am. Although Doctor Thomas never examined C, both agreed that C would have been more severely acidotic at some time earlier. They agreed that C suffers from dyskinetic cerebral palsy, with GMFCS level 5 disability, spasticity and dystonia. He is entirely dependent on others and cannot log roll or lift his head. He is PEG fed and has multiple learning difficulties As to the cause (APHI or CPHI) Doctor Thomas deferred to the radiologists on that. Doctor Agrawal stated that the neurology did not support a predominantly watershed injury but if it occurred it was before the APHI. Doctor Thomas’ answer was vague (he said it was difficult) and he relied on other experts. Doctor Agrawal postulated an acute profound cessation of blood flow as the cause, such as from cord compression, although he allowed for some preceding CPHI. Doctor Thomas did not disagree. As to timing, Doctor Agrawal stuck to his opinion that it occurred after 09.40 am and Doctor Thomas asserted it was more likely before 08.30 am because of the CTG trace and the recovery time required.

84. In cross examination Doctor Thomas accepted he postulated his answers based on Doctor Stoodley's evidence of two causes for the HI and only the Defendant's experts' reports. He accepted that, had he seen Doctor Lightman's report, that would have affected his opinion. In my judgment, it would have been good practice for both sets of lawyers to have ensured that their experts had the other side's reports before the joint discussions took place. He accepted that it was possible that the injury occurred during the day but more likely that it occurred at night. He deferred to Doctor Rennie on recovery rates but considered the papers she relied upon as poor. He explained that blood gas pH is a negative representation of Hydrogen ions in the blood. The base excess measures the metabolic component of the acid base. The main factors contributing to acidosis are the CO<sub>2</sub>, the lactate and other bases. A normal pH is 7.35-7.45. A fall to 7 represents significant acidosis. A difference of 0.1 in pH would be significant, but a difference of 0.01 would not be. Doctor Thomas advised that the post event disabilities indicated that the insult arose within 12 hours before the birth. He advised that a CTG FHR baseline of less than 100 bpm would be consistent with APLI. He advised from his clinical experience that to recover from a severe APLI pH back up to a pH of 7.01 would take 2-3 hours, not 10 minutes. He would have expected more recovery if the APLI had occurred just after 02.40 am, so he favoured the injury arising later in the nighttime window before the 08.30 am CTG. In re-examination, Doctor Thomas advised that with C's severe APLI his pH will have reduced to 6.6-6.8.

### **Analysis of causation**

85. Attractively argued though Doctor Agrawal's theory was, there were various fault lines within it. Firstly, the sheep experiments did not support the level of recovery in C's levels of acidosis, from a very severe brain injury, to his condition at birth, in a mere 3-10 minutes (or indeed the 14 minutes maximum which I calculated above). I do place a little weight on those because they are the only review papers available and they were not criticised for their methodology, only for having small samples. Secondly, the other experts did not accept that recovery in that very short space of time was likely from their clinical experience. Thirdly, the 08.30 am CTG started with a baseline at 105 bpm, which is below normal, leading slowly up to 140-160 bpm. I accept Mr Mason's evidence that this might indicate a post injury recovery process. Fourthly, the Agrawal hypothesis rests on cord compression and so on M staying still for 25 minutes causing that compression. But, on his theory, this stillness was happening at a time whilst she was being prepared for theatre, then transported from her room to theatre, then being consented and prepared for the spinal anaesthetic. The entry in midwife Rogers' notes on the start of the operation was: "knife to skin 10-", in which case she must have had the spinal before then and been bent over for the needle insertion. That would have involved quite substantial body movement, potentially interrupting any cord compression. Whereas the nighttime compression would probably have occurred when M was asleep. Fifthly, I consider that Janet Rennie's approach was thoughtful and analytical. I prefer her evidence to that of Doctor Agrawal on the timing of the hypoxic insult. I also prefer the evidence of Mr Mason and Doctor Thomas on the timing of the insult.

86. For the above reasons, I find that C suffered an APHI, lasting 20-25 minutes, before 08.30 am and probably at the later end of the period 05.15 – 07.50 am. It probably ended at or just before 07.50 am, because midwife Rogers went to see M then, took her vital signs and BSL was seen dribbling, so M may well have moved her body during that time. There might have been some preceding CPHI, but on balance I prefer the expert evidence stating that there was a unitary cause: APHI. A lot of brain injury was caused during the APHI insult and I consider it likely that C's arterial cord gas pH probably reduced to 6.6-6.8 as Doctor Thomas postulated. After the APHI there would then have been a period of re-oxygenation and resuscitation for C in utero until birth, lasting perhaps 2-3 hours. Then, C was born with improved but still acidotic cord pH readings. Roughly 4-6 hours after the end of the APHI insult (perhaps very roughly between 11.50 am and 13.50 pm) the usual secondary period of cellular damage probably arose.
87. None of the experts, other than Doctor Agrawal, whose evidence on timing I have rejected, advised that there was any or any continuing hypoxia after 08.30 am if the main injury had occurred earlier. Instead, they advised the C was being resuscitated in utero. I find that there was no continuing hypoxia after 08.30 am.

### Conclusions

88. **Breach.** For the reasons set out above at paragraphs 62-64, I find that there was no breach of duty by the midwives at night or in the morning. I find that the alleged incident of M going to the toilet in the night and reporting fresh blood to a midwife did occur but much later than M recalled and probably between 08.10 and 08.35 am. It was noted down at 08.35 am and led to the consultant being brought over to M within 13 minutes, so no breach arose.
89. For the reasons set out at paragraphs 71 to 76, I find that there was a breach of duty by Mr Siddig at 08.48 am. I consider that he should have read the notes, examined M and decided that M and C need delivery by a category II CS as soon as possible, with as much continuing FHR monitoring as was possible. A different breach arose after 09.40 am due to Mr Siddig failing to ensure similar FHR monitoring but that adds nothing to the earlier breach.
90. **Causation.** I find that C's severe brain injury was caused silently and tragically in the night, between 05.15 and 07.50 am, probably ending at or close to 07.50 am. It was caused by APHI. It was caused by a reversible mechanism. This was probably due to cord compression. This was not anyone's fault. It was not the midwives' fault and it was not M's fault. No one was to blame. By the start of the daytime CTG, at 08.30 am, the damage was either done or the course was set for the full damage to emerge over time in the usual 3 phase manner. Therefore, the breaches at 08.48 am and thereafter and those after 09.40 am made no difference to the outcome and made no contribution

to it. The primary damage had been caused, although the normal further sequelae of A PHI arose thereafter and further consequential damage would arise hours later.

END