Elisha Woods (A protected party by her mother and Litigation Friend Julie Woods) v Doncaster and Bassetlaw Teaching Hospitals Nhs Foundation Trust


No Substantial Judicial Treatment

Court
King's Bench Division

Judgment Date
11 June 2024

Case No: KB-2021-003001

High Court of Justice King's Bench Division

[2024] EWHC 1432 (KB), 2024 WL 02962822

Mrs Justice Lambert DBE

Date: 11/06/2024

Hearing dates: 22-29 January 2024

Representation

Richard Baker KC (instructed by Switalskis Solicitors) for the Claimant.
Caroline Harrison KC and Alex Ivory (instructed by DAC Beachcroft) for the Defendant.

Approved Judgment

Mrs Justice Lambert DBE:

1. The claimant is now aged 25 years old having been born on 14 October 1998. She brings this action for damages for personal injury arising from the alleged negligent delay in her delivery by the medical and nursing staff of the Doncaster Royal Infirmary ("the hospital") between 28 September 1998 and 14 October 1998. She has been assessed for the purpose of this litigation by Dr Ruth Kent, a consultant in neurological rehabilitation, who sets out the claimant's disabilities in her report of 29 January 2019. The claimant has only mild motor problems but her cognitive and behavioural difficulties are complex and extensive. They affect her memory, executive function, and her understanding of language. These cognitive and language difficulties in combination with their behavioural sequelae make the claimant particularly vulnerable. The defendant does not challenge the claimant's status as a protected party. The claimant brings this litigation through her mother and litigation friend, Julie Woods.

2. The claimant is represented by Mr Richard Baker KC and the defendant by Ms Caroline Harrison KC and Mr Alex

Ivory. I am grateful to counsel and their respective teams for their considerable help.

Background

3. The background and chronology below are drawn from the hospital records.

4. The claimant was Julie Woods' first child. Mrs Woods attended her general practitioner on 19 February 1998 when the pregnancy was confirmed, and she was given a hospital booking appointment for 4 March 1998. Mrs Woods attended the hospital for routine appointments. Traces of sugar were identified in her urine in April, and it was recorded that she should undergo a glucose tolerance test at 28 weeks' gestation. On 5 June Mrs Woods attended the hospital feeling unwell since the previous day and complained of feeling nauseous, lightheaded and with intermittent cramping pain in her lower abdomen. It was noted that she was not complaining of pain on arrival at hospital but reported a watery discharge since yesterday. A speculum examination was performed which demonstrated a white discharge. Swabs were normal. She attended hospital again on 1 September 1998 querying whether she had experienced a spontaneous discharge of the membranes.

5. On 28 September 1998 Mrs Woods self-referred to hospital. The pregnancy was 39 weeks' gestation. At 19.45 it was noted that she had a history of *"? leaking* " since midday and having felt contractions that morning. Fetal heart monitoring using a cardiotocograph ("CTG") was started at 19.42 and ran for around 100 minutes ("the first trace"). Shortly after the CTG started the midwife recorded that the fetal heart was 160 – 175 beats per minute ("bpm") and that the Senior House Officer, Dr Joynes, had been informed.

6. At 20.10, Mrs Woods was assessed by Dr Joynes who recorded in the clinical notes:

"Slight leakage – egg cup full

Some low abdo pain – 10 hr ago. Not now or since

On speculum: os closed, some white discharge

Triple swabs

Nitrazine negative

CTG – uterine contractions??

Baseline – 165 BTBV"

7. Dr Joynes recorded that the trace was to be reviewed by the senior SHO, Dr Samy. Dr Samy then recorded that " *CTG shows tachycardia around 160 bpm but good variability. Keep on CTG for longer and review."*

8. At 21.00 the midwife recorded: " *fetal heart remains 160 – 170, fetal heart down to 150, Dr Samy informed. To remain on CTG recording. Dr Samy will come to see her later."*

9. At 21.30 hours it was noted that Dr Samy had reviewed the first trace. The fetal heart rate was noted to be 155 – 160 bpm. He advised that Mrs Wood remain in hospital and have a repeat CTG in one hour's time.

10. By 22.40 Mrs Woods' care had been taken over by Midwife Sly. She made the following entry in the records: " *CTG recommenced. F(etal) H(eart) baseline 140 bpm. Accelerations were noted. No decelerations. Baby active. To be reviewed by Dr Samy.* "

11. The CTG referred to by Midwife Sly above ("the second trace") had been started at around 22.30. It ran for a total of around 33 minutes. Dr Samy reviewed Mrs Woods at or around 23.00. He made the following note:

> *"Reviewed (after being on CTG showing fetal movements). Fetal movements (++) felt by patient and shown on monitor. CTG baseline 140 bpm (in between movements). Variability 5 – 10 bpm. Accelerations repeated with movements (giving the impression of tachycardia but baseline between movements 140 bpm). Therefore, reactive trace. Maternal contractions shown on monitor but not felt by patient ?Braxton Hicks. Therefore, allowed home. Reassured. TCI [to come in] if feels contractions or reduced fetal movement."*

12. The claimant attended hospital again on 6 October 1998 when she saw Dr Samy. The pregnancy was term plus one day. Dr Samy recorded the presentation (which was cephalic), maternal weight and blood pressure and checked the urine. He noted that Mrs Wood should come in for induction of labour to take place on 19 October 1998. The clinical record makes no reference to any inquiry or discussion concerning fetal movements.

13. Overnight 13/14 October, Mrs Woods attended the hospital with a history of regular contractions. She was assessed at 02.15 when a history of contractions 1 in 5 since 02.00 was noted. It was noted that she had had a show on Friday but did not have spontaneous rupture of the membranes. A CTG was started at 02.16 which showed a baseline of 140 bpm but reduced variability down to 2/3 beats per minute. The CTG ran continuously until 05.00. At 03.20. Dr Ibrahim examined Mrs Woods, reviewed the trace, and discussed care with Dr Barekat who agreed that the membranes should be ruptured with further observation of the CTG trace. Mrs Woods' membranes were ruptured at 04.15 and Dr Barekat reviewed her at 04.40, noting the presence of thick meconium liquor and a flat trace with no variability at all since admission at 02.15 and a baseline of 140 – 145 bpm. He noted that there were no accelerations, decelerations, shallow variable. A fetal blood sample to assess fetal wellbeing was not feasible and Dr Barekat advised delivery by caesarean section.

14. Mrs Woods was transferred to the operating theatre at 05.02. At 05.10 the fetal heart was auscultated and found to be 136 bpm. The claimant was delivered at 05.20 with the cord wrapped once around the neck. It was noted that there was thick meconium-stained liquor, and the placenta was gritty. The claimant was delivered in poor condition with poor respiratory effort and a heart rate of less than 100 bpm. Her birth weight was 3.046 kg. The claimant's Apgar scores included the value of 3 at 1 minute. Her upper airways were aspirated, and she was given oxygen via a facemask. Resuscitation improved her condition. An arterial blood gas sample taken from the umbilical artery was noted to have a pH value of 7.0. No base deficit was recorded. She was transferred to the Special Care Baby Unit at 05.45.

15. The claimant and her mother were discharged home on 21 October. The discharge summary recorded that the claimant was a post term baby born by emergency caesarean section after profound late decelerations and thick meconium staining had necessitated delivery.

The Trial Issues

16. It is not contentious that the claimant's condition in the first 24 hours of life was consistent with hypoxic ischaemic encephalopathy caused by chronic partial hypoxia during the perinatal period. It is the claimant's case that the onset of the chronic partial hypoxia was no earlier than around three days before her birth: that is, no earlier than 11 October 1998 and that it persisted until her resuscitation very shortly after her birth. It follows that, on the claimant's case, had the delivery taken place before 11 October, the claimant would have been spared the brain damaging effects of the chronic partial hypoxia and would be neurologically intact. Whilst not disputing the likelihood of a period of perinatal partial hypoxia, the defendant asserts that the effect of the hypoxia was not brain damaging and the claimant's current neurological condition is likely to be genetic in origin. Earlier delivery would not therefore have altered the claimant's neurological condition.

17. The cause of the claimant's current condition and disability is not a matter for determination at this trial. By the case management direction of HHJ Pearce sitting as a judge of the High Court, it was ordered that there should be a trial of a preliminary issue " *as to whether or not the defendant is in breach of duty as alleged in the Particulars of Claim* ." In this context, the breach of duty issue should be read as whether the claimant should have been delivered before 11 October 1989. This involves consideration of breach of duty issues and some aspects of the case on causation.

18. The claimant's case focusses upon the obstetric management on 28 September 1998 when Mrs Woods attended the hospital complaining of a history of leaking per vaginum. The case, which is advanced by Mr John Hare, consultant obstetrician, is that the first trace demonstrated a tachycardia and two decelerations. The tachycardia should have led to the trace being classified as "non-reassuring" or "suspicious" and the decelerations should have been recognised as a "pathological" feature. The appropriate course was followed by Dr Samy however and repeat CTG monitoring was commenced. However, the second trace was not reassuring. The second trace was not normal. It was not possible to identify the baseline; there were no typical accelerations and for most of the trace the fetal heart was running at 150/160 (that is, a tachycardia). In the circumstances, it is alleged that the decision by Dr Samy to discharge Mrs Woods to be followed up routinely was unreasonable and a breach of duty. Instead, Mrs Woods should have been asked to come back into hospital and to undergo a further fetal heart monitoring, an assessment of liquor volume and an assessment of fetal size.

19. Had such a further assessment been undertaken, it is the claimant's case that the further CTG trace would not have been normal or reassuring. An assessment of amniotic fluid volume would have found the volume to be reduced and the claimant would have already been showing signs of growth retardation due to placental insufficiency with head and abdominal asymmetry. In combination, these features would have mandated delivery on or around 29 September. The pregnancy would by this stage be almost 40 weeks' gestation.

20. The claimant further asserts that Dr Samy ought reasonably to have questioned Mrs Woods concerning fetal movements when he saw her for routine antenatal care on 6 October. There is no note that he did so. Had he asked Mrs Woods about fetal movements he would have been told by her that they were reduced. This ought to have led to a decision to undertake a further period of fetal heart monitoring on or around 6 October. The trace would have been abnormal and a decision to deliver ought to have been made. By this stage, the pregnancy was just over 40 weeks' gestation (term plus 1 day).

21. I should add that, in his opening note for trial, Mr Baker included a case on informed consent. It was never clear to me how such a case fitted with the expert evidence. In any event, in his closing written submissions Mr Baker confirms that no such case is now pursued.

22. The defendant denies breach of duty. The defendant's case which is advanced by Mr Derek Tuffnell, consultant obstetrician, is that the first trace was difficult to interpret. It was therefore prudent to admit Mrs Woods and wait for an hour or so before commencing a second monitoring of the fetal heart rate. The second trace was wholly reassuring. It demonstrated a baseline of 140 bpm with no decelerations and accelerations from that baseline which were synchronous with fetal movements as experienced by Mrs Woods and as registered by the tocograph. In the light of the normality of the second trace, it was appropriate to discharge Mrs Woods for routine ante-natal review.

23. The defendant submits that, even if Mrs Woods had undergone further assessment on 28 or 29 September, those assessments would not have led to a decision to induce labour before 11 October 1998. The defendant denies that by 28/29 September the claimant was affected by placental insufficiency or suffering from intrauterine growth retardation. Any further monitoring on either the 28 or 29 September (or 6 October) would have been normal and reassuring. Liquor volume would have been normal, and the claimant's weight would have been within reasonable range. No action would have been indicated.

24. Given this bare outline above, the issues for me therefore drill down to the following main questions:
    i) Judged by reference to the standards of the day, was Dr Samy's conclusion that the second trace was reassuring a reasonable conclusion in the sense that it was consistent with that of a reasonable body of obstetric opinion?
    ii) If not, and further assessment was reasonably required, then what on the balance of probabilities would have been the outcome of further assessment on 28/29 September? Specifically, would it have led to a decision that labour should be induced and, if so, when should labour have been induced.
    iii) Ought Dr Samy have asked Mrs Woods about fetal movements on 6 October and, if so, would the inquiry have led to the claimant's delivery on or around 6 October?

The Evidence

25. Mrs Woods and her husband gave evidence.

26. Mrs Woods' statement (dated August 2009) records that on 28 September 1998 she had felt a gush of fluid from her vagina. She thought that her waters had broken. Although there is no note of this, she also remembers (or at least she did at the time of the statement) that she told the midwife at the hospital that she was concerned because she thought that the baby was not moving as much as it had before. She remembered a conversation in which she was asked whether she had been drinking coffee to which the midwife made a comment that the baby's heart must be beating fast because of the caffeine. She was surprised that she was not kept in hospital on 28 September because of her concern that the baby was not moving as much, her fluid loss and because her urine samples had been continuously "glucose +++". She also reported having been violently sick over the previous two weeks.

27. Mrs Woods said that, after 28 September, she remained concerned about the pregnancy and in particular the lack of fetal movement but, having been reassured by the midwives that reduced fetal movement later in pregnancy was usual because there is less space for the baby to move, she did not seek any further medical advice until she felt contractions. In

her evidence at trial, she said that the baby had in earlier pregnancy been very active which is why she was so acutely aware of the reduction. She agreed that, on 13 October, she had reported reduced fetal movements when she saw her general practitioner, but she said that this was in response to a specific question by the doctor. She said that she had been reassured by the advice she had been given by Dr Samy on 28 September and so had not thought to raise it herself. She had no recollection of the consultation on 6 October with Dr Samy.

28. Mr Woods gave evidence also. He confirmed Mrs Woods' account of events to the extent that he was able to.

29. The defendant called no factual evidence, but I draw no adverse inference from this fact (nor am I invited to do so by Mr Baker). I have been provided with a history of the chronology of the claim and of the efforts made to contact witnesses by the defendant. In summary, when a claim was initially intimated by way of a letter of notification in December 2006, the allegation of breach of duty concerned a delay in delivery on 14 October 1998. Although there was reference to the second trace and whether it had been sufficiently reassuring to defer induction of labour to 19 October 1998, no allegations of breach of duty were raised concerning the obstetric management on 28 September and so no attempt was made to obtain statements from witnesses who were involved in the management on 28 September. Thereafter, absence of communication from the claimant's then legal team led to the defendant's file being closed until a letter of claim was sent by the claimant to the defendant on 4 February 2019. Following the letter of claim of February 2019, the defendant's solicitors attempted to identify and locate Dr Samy without success. Midwife Sly had been dismissed from the Trust in 2008 for reasons which were not known and was thought therefore unlikely to provide evidence.

Expert evidence: Mr John Hare

30. The claimant's expert is Mr John Hare. He qualified in 1964. He was a consultant in obstetrics and gynaecology from 1976 until his retirement in April 1998 with posts at Cambridge and the Cambridge Heath District, then with Huntingdon and latterly Hinchingbrooke Healthcare Trust. He, like Mr Tuffnell, has had a deeply impressive career.

31. His report is dated March 2023. He gave evidence at trial. The following is a summary of the salient parts of his evidence.

32. The first trace ran for a period of around 102 minutes with the speed of recording of 1 cm per minute. On his review of the trace, it showed two abnormal features. A tachycardia and 2 decelerations.

33. For the majority of the trace the average baseline rate was 165 – 170 bpm. This rate is in excess of the internationally accepted 1985 FIGO (International Federation of Obstetrics and Gynaecology) Guideline which states that a fetal heart rate pattern is "suspicious" if the heart rate falls above 150 bpm. The evidence based clinical guideline concerning the use of electronic fetal monitoring published by the Royal College of Obstetricians and Gynaecologists defines a normal baseline heart rate as one which is running up to 160 bpm. However, Dr Donald Gibb, the author of Fetal Monitoring in Practice stated in the edition of the manual current in 1998 that " *Priority should be given to the revised definition of normal baseline FHR, 110 – 150 bpm* ." Gibb continued: " *any tracing with a baseline rate of greater than 150 bpm should be carefully scrutinised for other suspicious features* ." On either of these two definitions of the acceptable normal range for the fetal heart rate, the first trace demonstrated a tachycardia.

34. The two decelerations were timed at 20.30 and 21.00. The first deceleration lasted 6 to 7 minutes, the second deceleration was much shorter. In his report he said that the presence of the two decelerations meant that the trace should be classified as "pathological." In his oral evidence and in the joint note of his meeting with Mr Tuffnell he described the trace as "non-reassuring with pathological features."

35. The second trace is of poor quality. In its current (that is, as at trial) condition significant sections of the trace are illegible having degraded over time. The court therefore used a copy of the trace which had been obtained by Mr Hare at the time of his initial reporting of the case in around 2019. The trace quality had degraded even by 2019, but some sections, in particular the earlier sections, of the trace remained legible.

36. The trace ran for around 32 minutes. Mr Hare described the trace as showing the fetal heart varying in rate between 140 bpm and 160 bpm. Most of the time the heart rate was running at a rate between 150 and 160 bpm. Critically, he observed that at no point could the baseline be determined. There was only one short section of around 3.5 minutes at the beginning of the trace where it was possible to see a flat section of trace and there the heart rate was around 140 bpm. 3 or so minutes was not a sufficient period during which to declare a baseline. In order to determine the baseline, a heart rate which is stable for 10 minutes or a minimum of 5 minutes was necessary. The FIGO Guidelines define the baseline as " *the mean level of the fetal heart rate when this is stable, accelerations and decelerations being absent. It is determined over a time period of 5 or 10 min and expressed in beats/min (Bpm*)". The RCOG Guidelines confirm that the baseline is determined over a time period of 5 or 10 minutes. In Mr Hare's opinion there are no typical accelerations or decelerations. Mrs Woods appeared to be recording frequent fetal movements and the trace was impossible to interpret.

37. Mr Hare said that it was unacceptable for Dr Samy to have concluded that the trace was reassuring. The second trace had been obtained for clarification purposes and it had not served that purpose. It was impossible to interpret, and the only safe course was to conclude that it was non-reassuring. He disagreed with Dr Samy's stated conclusion (in the clinical notes) that the trace was accelerative and reactive to fetal activity. First, he said that the trace was not long enough: it had not been allowed to continue until fetal activity had ceased so as to permit the identification of the true baseline. Mr Hare said that if the tachycardia was thought to be due to fetal activity, then, as fetal activity is finite, the trace would have permitted identification of the resting baseline had it been continued. Second, Mr Hare made the linked point that it should not have been assumed that the tachycardia was due to fetal movement. The first trace ran for 100 minutes which was, in his opinion, just too long to be explained by the fetus being in an active state.

38. Mr Hare therefore said that the only reasonable management was a repeat CTG and an assessment of fetal wellbeing using some of the elements of a biophysical profile. Much time was spent at trial discussing whether biophysical profiling was in use in 1998 or whether it was outdated. However, as Mr Hare had set out in the joint note, his position was that it was only elements of biophysical profiling which should have been used to obtain more information about the pregnancy. There ought to have been a further CTG, a measurement of liquor volume and an assessment of the size of the fetus including head circumference. Mr Tuffnell agreed that these were reasonable further investigations if there was a concern over the condition of the fetus.

39. Mr Hare said that at birth, the claimant had intrauterine growth retardation ("IUGR") due to placental insufficiency and that this was the process which had led her to suffer a period of chronic damaging hypoxia in the days leading up to her delivery and accounted for the appearance of the CTG on 13/14 October 1998 and her condition following her delivery. He said that the process of IUGR due to placental insufficiency had started during the third trimester and was present on 28 September 1998. In his opinion, the features on the first trace were caused by a reduction in oxygen to the fetus due to reduced placental function. The oxygen reduction was not (in September 1998) brain damaging because the fetus was compensating for the reduction in oxygen by an increase in the heart rate.

40. It follows from Mr Hare's opinion on the underlying pathology that had the further investigations been undertaken on or around 28 September then: the further CTG trace would have been, at best, non-reassuring; the liquor volume would have been reduced and there would have been growth asymmetry with relative sparing of the head. On this basis, Mr Hare said that induction of labour would have been mandatory.

41. Mr Hare agreed that, given that the underlying cause for the trace features was placental insufficiency, then the appearance of the trace would not improve over time. Placental insufficiency is a deteriorating condition, and the trace would never go back to normality with a normal baseline and standard pattern. When it was put to him by Ms Harrison that the second trace was an improvement on the first, he did not accept this. Although there were good variability and good fetal movements, he said that it was impossible to detect the baseline and so it was impossible to interpret the trace in the way that it had been by Dr Samy. Nor did he agree that, if his theory concerning the underlying cause of the trace features was correct, the claimant would not have survived until 14 October. He said that in his experience the rate of deterioration might be very slow and may vary as the pregnancy progresses. He drew the court's attention to the FIGO Guidelines which set out at [4.3]: " *that the evolution of chronic fetal hypoxia is slow. In general, abnormal fetal heart rate patterns are observed over several weeks before ante partum fetal death* ." It was suggested to him that the FIGO Guidelines did not footnote the studies and evidence upon which they were based. Mr Hare said that the FIGO Guidelines were produced by 20 eminent scientists who would not have said this absent a good basis for doing so.

42. The abnormal features in the first trace and the absence of reassurance to be derived from the second trace were, said Mr Hare, complemented by other features of the pregnancy which, although individually did not require earlier delivery, either required further inquiry or supported the case for induction. Those features were: that Mrs Woods was taking a drug Colofac for treatment of her irritable bowel syndrome, the possible rupture of the membranes on 1 September 1998; multiple episodes of glycosuria during pregnancy and the absence of a glucose tolerance test during the third trimester; her report that she had been unwell in the two weeks leading up to the 28 September with persistent vomiting.

43. The only further feature which was, to use his phrase a "standalone" indication for active management was the maternal history of lack of fetal movements. Mr Hare told me that Dr Samy should have asked Mrs Woods specifically about her perception of fetal movements on 6 October 1998. He should have noted down her response. If, as she asserts, she was sensing a reduction in fetal movements then this should have prompted a repeat CTG assessment. This would, in turn, have proved to be at best non-reassuring and would have led to further assessment and induction of labour.

Mr Derek Tuffnell

44. Mr Tuffnell is a consultant obstetrician. He retired from NHS clinical practice in 2019 after 25 years in post at Bradford Hospitals. He has had an impressive career and in addition to his clinical work in Bradford he has occupied a number of roles with the National Institute of Clinical Excellence (NICE) and the RCOG.

45. Mr Tuffnell agreed that the first trace was not easy to interpret. The predominant feature of the trace was the heart rate. In his report he described the rate as being around 155 to 165 bpm with variability of around 10 bpm. He described the heart rate as falling " *near the top of the normal range* ." In his evidence at trial, he said that the heart rate was 155bpm – 170 bpm which was the upper limit of what is considered normal. He acknowledged that interpretation of any CTG when the baby is quite active was challenging. In his report he said that it had been noted that the baby was moving at this time. He described the clinicians as being cautious in deciding to repeat the CTG, but he clarified this in evidence by saying that,

had the trace not been repeated, then he would have been disappointed. He noted however the normal variability which was a reassuring feature. As he put it, the clinicians were faced with a trace which could not be described as normal but was not of such concern that the clinician would wish to intervene. In his view therefore, Dr Samy did the correct thing by allowing the trace to run for a while but, when still unsure of it, the trace was discontinued for a while before it was re-started.

46. The purpose of the second trace was to check whether the fetal heart remained elevated. Mr Tuffnell found the second trace to be clearly interpretable with a normal baseline of around 140 bpm. This was apparent from the first 3 or so blocks of the trace representing the first 3 or so minutes of monitoring. When challenged, Mr Tuffnell did not agree that it was necessary to have 5 minutes (at least) of stable baseline in order to determine the true baseline. He said that when the RCOG referred to the need to have 5 or 10 minutes of stable baseline the authors were referring to the fetal heart rate in labour and not during the antenatal period. Provided that there were plenty of accelerations and good variability, it was not necessary to have an interrupted period of 5 minutes minimum to be confident of the true baseline.

47. Mr Tuffnell said that following the initial period of baseline heart rate there were then a number of accelerations which coincided with fetal activity. Fetal activity was recorded in two ways on the trace. First, by Mrs Woods pressing a button when she felt the fetus move which generated an ink mark on the paper trace. Second, by the tocograph recording fetal movement when detected (although Mr Tuffnell acknowledged that this was not a particularly robust tool). Although some of the ink markings were faint, he was confident that the fetal heart rate was accelerating in response to fetal movements. As he put it, in the first section of the trace there were multiple fetal heart movements and multiple accelerations in association with the fetal activity.

48. The other key feature of the trace, and aid to its interpretation, was that beat-to-beat variability was maintained throughout. Mr Tuffnell said that in the "hierarchy of reassurance" the most reassuring feature is the presence of accelerations, but that normal variability is the second most reassuring feature. He told Ms Harrison that a tachycardia with normal variability was in his experience almost always caused by the baby moving and that whilst he could not rule out that a persistent tachycardia with normal variability may be due to hypoxia, this would be very surprising. It was not, he said, something which he had ever seen in his career.

49. Mr Tuffnell said that Dr Samy's note suggested that he had thought carefully about the trace and had reached the correct conclusion. It was the conclusion which he shared. The second trace was healthy, and the heart rate was reacting to fetal movements. He did not accept that the active phase usually lasted only 20 minutes or so. All babies have quiet and active phases, and some will move more than others during active periods. He said that it was quite appropriate to review the first trace in the light of the second trace and to reinterpret it. On this basis it was a safe conclusion that the elevated heart rate apparent on the first trace was a response to fetal activity and the so-called decelerations were not true decelerations but were a return to the true baseline of around 140 bpm. He referred the court to the edition of "Fetal Monitoring in Practice" by Donald Gibb and S. Arulkumaran which was current in 1998 which referred to difficulties in assessing the baseline heart rate of an active fetus. The authors state: " *Persistent accelerations may lead to confusion such that some traces have been termed "pseudodistress" patterns. When the fetus is very active it may show so many accelerations that it is misinterpreted as tachycardia with decelerations. This situation can arise in the antenatal period or in labour* ." This was, exactly, the assessment made by Dr Samy and which he endorsed.

50. Mr Tuffnell said that he would not have interpreted the first trace as showing a deceleration. He said that interpretation of CTG traces is all about pattern recognition and the two dips in the fetal heart trace did not, to him, look like decelerations. They were the wrong shape and if they had been true decelerations in conjunction with a true tachycardia then he would have expected them to be associated with loss of variability. Furthermore, a deceleration lasting

several minutes (as postulated by Mr Hare) would have been a late feature of placental insufficiency. Two things follow from this. First, the only correct management would have been immediate delivery (rather than further investigations) and second, the claimant would not have survived until 14 October 1998.

51. Mr Tuffnell accepted that if contrary to his opinion, the clinician ought to have sought further information then an assessment of fetal weight, liquor volume and a further CTG would be a reasonable investigation to undertake. He did not accept however that the claimant was suffering from IUGR in September 1998. He said that babies affected by IUGR fall into two groups. In addition to those which are affected by placental insufficiency in the third trimester or earlier, there is a group of babies who suffer from what he described as "late onset" IUGR. That group will have reasonably normal growth but towards the end of pregnancy the placenta begins to start failing and those fetuses tend to deteriorate over a few days and that process of deterioration is more rapid. He said that in September 1998 the liquor volume would have been assessed as normal; the trace would have been normal, and the fetal weight would have been assessed as within the normal range. In these circumstances, there would have been no indication to induce labour before Mrs Woods was well past term.

52. Mr Tuffnell agreed that, on 6 October 1998, he would have expected Dr Samy as a matter of routine to ask Mrs Wood about reduced fetal movements and he would have expected a competent clinician to make a note of the outcome of that inquiry. He told me however that notetaking in 1998 was not as diligent. He agreed that if there had been a complaint of reduced fetal movements then Dr Samy ought to have undertaken another CTG. He did not accept that a further CTG on 6 October would have been abnormal.

Midwifery Evidence

53. Both parties had served expert evidence from midwives. The claimant relied upon Ms Dawn Johnston and the defendant upon Ms Sue Brydon. The evidence of the midwives was of doubtful relevance. There were no criticisms of the midwifery management, and it was common ground that the midwives acted reasonably by, at all times, escalating their concerns to the obstetric team. The last note made by Midwife Sly was at 22.40 and there is no evidence that she had any input into the final assessment of the second trace or the decision to discharge Mrs Woods back into routine antenatal care.

54. Mr Baker's position was that the midwifery expert evidence added nothing and should not be called. Ms Harrison submitted forcefully that the court should hear from Ms Brydon on the basis that midwives were equally expert in interpreting CTG traces as obstetricians. In the event, I permitted both experts to give evidence. Neither, I regret to say provided me with much assistance on the issues which I have to grapple with.

55. Ms Johnston can only reasonably be described as a reluctant witness. She was loathe to be drawn into an assessment of the second trace given the poor quality of the trace. She did however agree that the initial section of the second trace showed a baseline of around 140 bpm and no decelerations and accelerations with good variability. But, she added, it would not be appropriate to interpret a trace on the basis of the first 10 minutes only. She accepted that an active phase might last as long as 40 minutes, no more than one hour. She said that all mothers are different, and the usual period would be no more than 30 or 40 minutes.

56. Ms Brydon's report extended to all aspects of the obstetric management including her interpretation of both CTG traces. Whilst conscious of her expertise in CTG interpretation I do however bear in the forefront of my mind that the critical issue in this case is the interpretation of the second trace and/or whether it was capable of sensible interpretation. In

this case, the critical interpretation was made by Dr Samy, an obstetrician and not a midwife. I therefore regard Ms Brydon's evidence in this light. There is a further issue which arises in this context. Ms Brydon's report refers to the second trace as being of 20 minutes duration. She makes the same mistake as Mr Tuffnell made in his report. Although Mr Baker put the position rather more strongly, I accept that this is a curious coincidence particularly given that Ms Brydon told me that she would have written her report without having sight of Mr Tuffnell's report. Her explanation that she was referring only to the legible portion of the trace was not immediately compelling. Had I decided that I should take into account her evidence on the critical issue, then I may have had to resolve this issue and reach a conclusion about the independence of her opinion. As it is, I do not have to.

57. In her evidence at trial, some time was spent seeking to interpret the degree of concern, or lack of concern, of the midwives caring for Mrs Woods on the basis of the records which were made at the time. This was obviously a difficult exercise and one which ultimately proved unhelpful. But Ms Brydon said that, from her perspective, it would not be uncommon for a CTG trace to demonstrate a tachycardia if the baby was active, but she would not be worried if variability was normal. She said that sometimes the fact of the CTG may provoke activity because it provides some acoustic stimulation and that active states can last for some considerable time. She told me that decelerations could be caused simply by a mother lying flat on her back and that if there were true decelerations, she would have expected there to have been more of them on the first trace.

58. As I have said, I bear this general evidence in mind whilst conscious that the focus of the case concerns obstetric decision making.

Findings/Conclusions

59. I start by making some preliminary points.

60. I am satisfied that the various additional factors or the so called "constellation" factors which Mr Hare relied upon to support the case for induction of labour either following the traces of 28 September or following the consultation on 6 October add nothing to the claimant's case.

i) The fact that Mrs Woods was taking Colofac as treatment for irritable bowel syndrome is irrelevant to the issues which I must decide. The drug is not contraindicated in pregnancy. The data sheet advises that it should, "in common with most drugs," be prescribed with care. As Mr Hare accepted, there have been no reported cases of the drug causing harm to pregnant women or foetuses in its 60 years of manufacture. It was used by Mrs Woods throughout her pregnancy with good effect on her symptoms and she was prescribed the drug throughout her second pregnancy also. It was an appropriate medication for Mrs Woods to be prescribed and the fact that it was being taken by Mrs Woods had no bearing on the management of her pregnancy.

ii) Mr Hare also raises the possibility that Mrs Woods may have had gestational diabetes on the basis that there were a number of instances of raised urinary sugar levels. He accepts that a glucose tolerance test was taken at 28 weeks and was normal, although the exact values are not in the records. His case is that further testing should have been done later in the pregnancy, although in response to questions from Ms Harrison, he accepted that had it been done then on balance it was likely to produce a normal result. In any event, it is not clear to me how any such failure to undertake further testing would have impacted upon a decision to induce labour. As such, I put the point to one side.

iii) Mr Hare is concerned by the possibility that Mrs Woods had suffered a leak of amniotic fluid. He agreed that on 1 September and on 28 September nitrazine tests had been carried out and had been negative. This ruled out membrane rupture on the balance of probabilities. Mr Hare maintained however that the possibility that there had been a membrane rupture remained "a very minor factor" and not a "standalone" factor when managing the pregnancy post term. It was, he said, part of the constellation of factors which might influence the decision to induce. I disagree. Having ruled out membrane rupture on balance, I am satisfied that even as a minor factor the

history of leakage, appropriately investigated as he accepted, would have been irrelevant to any decision to induce.
iv) Finally, one of the particulars of negligence concerns the failure to investigate adequately Mrs Woods' history of vomiting. Much time was spent in cross examination of Mrs Woods on this subject. In fact, Mr Hare accepted in his evidence that whilst it was something which he might have expected to have been investigated it would not have had any impact on the decision to induce or not on 28/29 September. As such, I put this point to one side also.

61. At various stages of their questioning and in their closing submissions counsel accused the expert on the other side of failing to provide objective and unbiased opinions, both in their reports and in their evidence at trial. Counsel sought to challenge the experts by suggesting to each that their reports had been prepared with the respective statements of case in mind. It was expressly put to Mr Hare that his analysis had been influenced by his knowledge that, in order for the claim to be successful, it was imperative to deliver the claimant before 11 October 1998, a criticism which he rejected emphatically. A similar point is taken against Mr Tuffnell in Mr Baker's closing submissions in which he describes as " *a further worrying feature* " of Mr Tuffnell's evidence that his report was prepared having read and considered the Defence, the " *obvious implication being that Mr Tuffnell prepared his report to meet the Defence* ." I am aware that both experts are experienced and have broad shoulders. However, I am quite satisfied that there was no basis for either counsel to challenge the objectivity of the experts in this case. It would be distinctly odd if an expert's final report, prepared for the purposes of disclosure had not been prepared having had sight of the statements of case.

62. Having cleared the decks of those points, I start by recording some aspects of Mr Tuffnell's evidence which, notwithstanding my conclusion above, I found to be problematic. A number of them feature in Mr Baker' closing submissions.

63. As Mr Tuffnell acknowledged, his report of 2023 had been prepared by him without a recent review of the second trace. In his covering letter to the report dated 7 June 2023 he wrote: " *I have completely redrafted my report because the case that is being put now is different to the case that we dealt with in 2007. However, my interpretation of the CTG is based upon the interpretation I made at that time on 28 September 1998. I do not seem to have another copy…I have based this report upon my previous interpretation of the CTG* …" I assume that the reference to 28 September 1998 is an error and that he meant, in 2007. When asked about the preparation of his report in court he said that he may have had a copy of the second trace, but it was not legible. This is not what he said in his covering letter though. Whatever the problem, he confirmed that he had imported into his July 2023 report that section of the report from 2007 which set out his interpretation then of the second trace.

64. On any assessment, this was wholly unsatisfactory. In 2007 when the section of the report concerning the second trace had been written, the second trace was not the subject of criticism. By 2021 when the case was issued however, the second trace was the focus of the claim. A better and more legible copy of the second trace was available. Even if the copy retained by the defendant had degraded, the claimant's team were in possession of a copy which had been made by Mr Hare before the trace had deteriorated badly. It is unclear whether it was sent to Mr Tuffnell. If it was then it does not appear that he reviewed it before the meeting with Mr Hare. In the joint expert minute, he observed that his " *consideration of the CTG has to be based upon my first review of this case which happened in 2007. The current available copy of the CTG actually has no heart rate visible, so I can only rely on my opinion at that earlier time. I cannot review it* ." In fact, a better copy of the second trace formed part of Mr Hare's report. Although the later part of the trace remains difficult, if not impossible, to interpret, it is possible to identify (although perhaps not clearly) the heart rate, the variability and the ink markings associated with fetal activity in the earlier sections. The second trace appeared within the body of Mr Hare's report (rather than as an appendix). It is difficult to see how, had Mr Hare's report been considered by Mr Tuffnell, he could have missed the copy of the second trace.

65. There are some other parts of Mr Tuffnell's evidence which are concerning. In the section of his report which

considers the first trace, he sets out that it had been noted that the baby was moving at the time of the trace. There is however no such reference in the notes. His report refers to there being no history of continuing leakage and/or leakage on the day of attendance. This statement must be seen in the context of a documented report from Mrs Woods of leakage, indeed on the basis of the midwifery note, this was the presenting complaint and the reason for the self-referral. Mr Tuffnell explained that this was clumsy wording and that he was intending to convey that there had been no further leakage following admission but, as Mr Baker put to him, this is not in fact what he wrote in the report.

66. Both of the points above are small points. But in conjunction with the circumstances in which the report was put together by Mr Tuffnell they go to create an unfavourable picture. I am reluctantly driven to the conclusion that, in this case, Mr Tuffnell's preparation has lacked the attention to detail which the case demanded. Predictably, it led to matters being raised for the first time in evidence (not having featured in his report or expert meeting and not having been put in cross examination) in circumstances when it was impossible to know whether Mr Tuffnell had thought about these things beforehand (but not set them out in writing) or whether they were occurring to him for the first time in the witness box. He relied on little, if any, literature even when he was challenging the use of Guidelines (for example, FIGO/RCOG and the need for 5 minutes minimum of stable baseline). Whilst I understand that in his oral evidence, he may have been trying to convey the reality of practice in a busy but competent antenatal ward, I regret to say that the overall impression was of a rather casual approach to the issues in the litigation. As Mr Baker highlights, this is in stark contrast to Mr Hare who gave the impression of having considered the issues in the case with real care and who provided thoughtful and measured responses to the questions posed.

67. It is against this background that I set out my finding and conclusions below.

68. The first issue that I must resolve is whether the second trace was sufficiently reassuring to justify discharging Mrs Woods back to routine antenatal care. Whilst dealing with this question is bound to entail some discussion of the trace features, it does not require me to fine tune my conclusions as to the interpretation of every shape and feature on each of the traces. I bear in mind that Dr Samy decided to repeat the trace, and no one is critical of that decision. The question for me is in some ways straightforward: it is whether the second trace was reassuring or not. This does not require me to determine the proper interpretation the various features on the traces. I set out my conclusion and reasoning below.

69. I am satisfied that the decision by Dr Samy to instigate further CTG monitoring after a break of one hour was appropriate and reasonable management. It was the only reasonable management in the light of the features on the first trace. I do not find, as Mr Tuffnell suggests, that it demonstrated a particularly cautious approach. A repeat trace was the only appropriate and reasonable response to the difficulties in interpretation of the first trace.

70. The first trace demonstrated throughout its course a heart rate in excess of the normal baseline. As Mr Tuffnell accepted in his oral evidence, the heart rate was beating at a rate of between 155 and 170 bpm. I do not accept that the FIGO definition of the limits of the normal baseline was " *more the American way* " as Mr Tuffnell suggested in his evidence. Dr Gibbs' pocket manual counsels doctors and midwives to give " *priority* " to the " *revised definition of normal baseline FHR 110 – 150 bpm* ." The manual describes a baseline of 150 -170 bpm as a moderate tachycardia. Even if the appropriate upper limit of normal was 160 bpm, the claimant's heart rate exceeded this value. The heart rate was not, as Mr Tuffnell suggested, towards the "upper limit of what is considered normal." It was over the limit of what is or should have been considered normal and by some margin. Although the reason for the tachycardia may not have been known prospectively the heart rate was undoubtedly tachycardic and had continued to be so for almost 100 minutes.

71. I do not accept Mr Tuffnell's opinion that the tachycardia should have been considered a feature of limited concern due to the retained beat to beat variability. His evidence was that tachycardia with retained beat to beat variability was almost always due to fetal activity rather than hypoxia and that whilst he could not rule anything out, in his career he had never encountered such a feature. As Mr Baker acknowledges, this statement was later qualified to an extent when Mr Tuffnell remarked that fetal activity was the cause in the " *vast majority of situations* ." The point does not feature in Mr Tuffnell's report. It was not mentioned in the joint expert note nor was it put directly to Mr Hare in cross examination.

72. Mr Tuffnell's interpretation of a tachycardia with retained variability as being almost always, or in the vast majority of situations, due to fetal activity is an overstatement. The RCOG define a tachycardia as a non-reassuring feature. The FIGO classify its presence as suspicious. Neither definition discriminates between a tachycardia with or without retained beat to beat variability. Dr Gibb's manual sets out that a very active fetus may show so many confluent accelerations that it is misinterpreted as a tachycardia with decelerations. It makes the point that if beat to beat variability is maintained then the trace will not generally represent an hypoxic fetus. But the manual also counsels that " *any tracing with a baseline rate of greater than 150 bpm should be carefully scrutinised for other suspicious features* ." In these circumstances I accept the claimant's case that it would be unsafe to assume that a tachycardia with retained variability was due to fetal activity.

73. The first trace also demonstrates two episodes when the fetal heart rate drops to around 140 – 150 bpm. Again, I am unable to accept Mr Tuffnell's opinion that because beat to beat variability was maintained throughout the course of the episodes, they should not be classified as decelerations and otherwise treated as benign features. This interpretation does not fit with the textbooks which describe the presence of a deceleration as a non-reassuring feature but one which if associated with additional loss of variability would justify the classification pathological. Viewed prospectively, it was impossible to judge the cause of the deceleration and whether it was due to hypoxia or, as Ms Brydon said in evidence, maternal position or for some other reason.

74. It follows that I accept Mr Hare's evidence that the first trace had two apparently abnormal features, tachycardia, and decelerations, and that there was a need therefore for further information.

75. The second trace did not provide sufficient reassurance to justify allowing Mrs Woods to go home with routine antenatal follow up. There are a number of reasons for this conclusion.

i) I accept Mr Hare's evidence that in order to determine a baseline heart rate, a minimum period of 5 minutes is required. Mr Tuffnell asserts that the minimum requirement for 5 minutes stable heart rate is relevant only to traces in labour, but this caveat does not appear in the FIGO or RCOG Guidelines. On the second trace Mr Tuffnell identifies a block of no more than 3 or so minutes when the heart rate was stable at 140 bpm. Thereafter he identifies a few episodes of a minute or so between accelerations when the rate is 140 bpm. I agree with Mr Hare that the second trace is not capable of being interpreted as demonstrating a fetal heart rate baseline of 140 bpm.

ii) I accept that the trace appears to show accelerations in conjunction with periods of fetal activity. However, if the tachycardia were due to the fetus being in an active state, then the active state must have continued for an unusually long period of time. The first trace lasted around 100 minutes and was tachycardic throughout its course save for the two decelerations. Mr Hare stated that the active phase would not usually persist for longer than 20 minutes or so. Mrs Johnson said that she could not recall an active phase lasting for more than an hour and thought that around 30 minutes was the norm. Mr Tuffnell did not give a timescale but indicated that he might have women on the antenatal ward for an hour or so with traces which are difficult to interpret. If the baby was active and variability maintained, then he would advise stopping the trace and starting it again. He said that the trace would come back normal. This of course is not what occurred in Mrs Woods' case where the trace did not come back normal. On the basis of this evidence as a whole, it does seem to me that if the tachycardia were to be explained by fetal activity, the active phase was very long indeed. It was a minimum of 1 hour and 40 minutes (subject to the short blocks of deceleration) followed by another period of around 30 minutes. This would assume that the baby was in a quiet phase between the two episodes of monitoring. If the baby was not in a quiet phase between the two

episodes of monitoring, then the active phase continued from 19.45 when the first trace started until around 23.00 when the second trace was disconnected.

76. Dr Samy's decision to let Mrs Woods go home on 28 September was not a reasonable decision. Having determined correctly that the first trace could not be interpreted and that a further trace should be performed to assist in its interpretation, the further trace did not serve its intended purpose. The second trace did not provide the necessary reassurance for the reasons set out above. As Mr Hare put it, the active phase is finite. The trace should have been allowed to run on beyond the active phase in order that the true baseline could be established. Having determined that another trace should be obtained it was wholly illogical to stop the trace before the active phase was over.

77. I therefore turn to the question of the further investigations which ought to have been undertaken and the results of those investigations. In reality, there is no difference between Mr Hare and Mr Tuffnell as to the types of further investigations. Mr Hare overstated matters in his report by referring to the need for biophysical profiling when, in effect, he was stating only that certain elements of the biophysical profile were required. Those elements were a further CTG, an assessment of liquor volume and an assessment of fetal growth. Mr Tuffnell did not disagree that if the obstetrician wished for more information and was concerned about the possibility of growth retardation secondary to placental dysfunction then those investigations were appropriate.

78. I am satisfied that had those further investigations been performed they would have led to a decision to induce the labour on or around 29 September 1998. I set out my thinking below.

79. The logical starting point for my determination of this issue is consideration of whether, in fact, the claimant was affected by IUGR due to placental dysfunction at birth. On this issue, it seems to me that all of the evidence points one way. There was an asymmetry between the claimant's head circumference and her birth weight. There was relative head sparing and the head circumference was on 50th centile whilst her birthweight was (using Mr Tuffnell's app) between the 9th and 25th centile. Both experts agreed that asymmetry is one of the features (or can be one of the features) of IUGR. The placenta was gritty, and the liquor was thick, and meconium stained. Both Mr Hare and Mr Tuffnell said that a gritty placenta is or is usually caused by calcification due to it not functioning properly. The thick liquor is due to reduced liquor volume which was, in Mr Hare's opinion, an important pointer towards IUGR. Mr Tuffnell accepted that thick meconium is commonly associated with reduced liquor which can be associated with IUGR due to reduced blood flow to the kidneys and so less liquor. Given all of these features, I find it likely that the claimant was affected by IUGR due to placental dysfunction at the time of her birth.

80. The next question is whether the claimant was affected by IUGR in September 1998 such that it would have been detected on further investigation on 28/29 September 1998, two weeks before her delivery and, on the claimant's case 11 or so days before her condition deteriorated and she began to sustain brain injury.

81. Mr Hare has explained the pathological process which he asserts was ongoing on 28/29 September. As a result of placental dysfunction, less oxygen reaches the fetal circulation and so the fetal heart beats faster to make up the deficit. For a period of time the deficit will be compensated and there will be no fetal brain damage. Once the heart is unable to compensate then brain tissue damage may occur. Mr Hare described the process as a slow one which may take place over a period of weeks. His experience suggested that on occasions it started at the beginning of the third trimester. The speed of the process he describes is reflected in the FIGO Guidelines which set out that: " *the main target of antepartum fetal heart monitoring is the detection of chronic fetal hypoxia related to chronic placental dysfunction… the evolution of chronic fetal hypoxia is slow . In general, abnormal fetal heart patterns are observed one or several weeks before antenatal death*

."

82. Mr Hare's case that loss of variability occurs only after the fetal heart is unable to compensate is reflected in Parer: " *in the presence of normal FHR variability, no matter what other FHR patterns may be present, the fetus is not suffering cerebral tissue asphyxia because it has been able to successfully centralise the available oxygen and is thus physiologically compensated. In the presence of excessive asphyxia stress however as evidenced by severe periodic changes or prolonged bradycardia this compensation may break down and the fetus may have progressive central tissue asphyxia. In this case it is theorized that FHR variability decreased and eventually is lost* ."

83. Mr Tuffnell's opinion that, had the trace demonstrated a tachycardia and decelerations, the claimant would not have survived until 14 October must be seen in the light of the literature above. The trace features on 28 September were not due to fetal hypoxia. On Mr Hare's analysis, on 28 September, the fetal heart was still compensating adequately for the placental deficiency. Indeed, the two episodes of deceleration on the first trace may have been, as Ms Brydon said, simply episodes when the fetal heart dipped due to maternal posture. The FIGO Guidelines and Parer support Mr Hare's opinion of a process which is slow with trace deterioration and loss of variability taking place only once the fetal heart is no longer able to compensate for the placental insufficiency.

84. The defendant alleged an inconsistency in Mr Hare's evidence when in the joint statement he recorded: "As baseline variability was not impaired this pattern indicates that this was an early stage of fetal compromise. The fetus would have been receiving adequate oxygen for the health of the brain to be maintained, albeit by increasing the blood flow to the brain by an increase in the heart rate. It is perfectly feasible that this should continue for several days before damaging hypoxia developed." (my emphasis). The point made by Ms Harrison at trial, and which I have been asked to address now following circulation of the draft judgment, is that the reference to " *several days* " is inconsistent with a time interval between 28th September and around 11th October when, on the claimant's case, the brain damage commenced. I don't accept that there is any inconsistency. On any plain reading "several days" is capable of referring to 13 days. In any event, the point which Mr Hare was making in the joint report, as in his oral evidence and which is supported by the literature, is that the downward trajectory of the fetal heart's compensatory mechanism may be very slow when the underlying mechanism is placental dysfunction. Mr Hare referred to "several days"; the 20 authors of the FIGO Guidelines referred to "several weeks." I see no inconsistency in Mr Hare's evidence and his evidence is supported by the literature.

85. I accept Mr Hare's account of the underlying process which culminated in the claimant's delivery on 14 October with IUGR. Whilst noting that Mr Tuffnell asserted that IUGR may be a "late stage" condition, there is no evidence in this case that it was so. The point was not raised in Mr Tuffnell's report and not put to Mr Hare, and I have no literature or information on the topic. Mr Hare's clinical experience was that placental dysfunction can start relatively early in pregnancy and in his experience, it can start at the beginning of the third trimester. I accept this evidence. Mr Tuffnell acknowledged that the gritty placenta was not something which would "happen overnight," accepting that it indicated that the placenta had been sub optimal in the last week or two before delivery. This may be a small concession by Mr Tuffnell, but it points in the direction of a process which had been ongoing since, even on his assessment, 14 days before delivery i.e., on or after 1 October.

86. I am therefore satisfied on the balance of probabilities that, had a longer CTG trace been undertaken on 28/29 September 1998, it would have continued to be abnormal as Mr Hare has said. It would have continued in this way without improvement until the fetal heart was ultimately unable to compensate for the oxygen deficit caused by the placental dysfunction. Although it does not fall within the remit of this trial, I understand the claimant's case to be that this was on or around 11 October 1998 when the claimant was subject to a period of chronic partial hypoxia. I also find that if a liquor volume assessment had been performed it would have demonstrated a lower-than-normal volume and that fetal assessment

would have demonstrated the asymmetry which was apparent at her birth. The gestation was term, or near term. In these circumstances, I accept that reasonable management mandated induction of labour on or around 29 September 1998.

87. These findings are determinative of the claim. However, I set out my findings in connection with the 6 October, since this was an issue which was argued before me. I am satisfied that Dr Samy failed to ask Mrs Woods about fetal activity on 6 October. Had he done so then he would, or ought, to have noted her response. The expert evidence on this point is all one way. Although I take into account that standards of note keeping may have improved since 1998, I also bear in mind that any note need only have been short. I therefore infer from the absence of any note that Dr Samy did not ask about fetal movements. Had he done so however I am not satisfied that Mrs Woods would have reported any reduction. Had she experienced a reduction in fetal movements following the 28 September, then I am confident that she would have reported them whether questioned or not. She was a concerned parent who self-referred when necessary. Given the advice which she received on 28 September to come back to hospital if she felt a reduction in movements, then I have no doubt that she would have abided by that advice, irrespective of any earlier reassuring conversation with the midwife.

88. Judgment should be entered for the claimant. I invite the parties to draw up the appropriate order.