Case No: HQ12X05057

Neutral Citation Number: [2015] EWHC 3395 (QB)
IN THE HIGH COURT OF JUSTICE
QUEEN'S BENCH DIVISION

Royal Courts of Justice
Strand, London, WC2A 2LL

Date: 24/11/2015

Before:

MR JUSTICE JAY

- -

Between:

MISS MELISSA RICH
(a protected party by her Mother and Litigation Friend HELEN RICH) **Claimant**

- and -

HULL AND EAST YORKSHIRE HOSPITALS NHS TRUST **Defendant**

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Simeon Maskrey Q.C. (instructed by **Stamp, Jackson & Procter LLP**) for the **Claimant**
Philip Havers Q.C. (instructed by **DAC Beachcroft LLP**) for the **Defendant**

Hearing dates: 3rd-6th, 9th-10th November 2015

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Judgment

MR JUSTICE JAY:

Introduction

1. This is the trial of Miss Melissa Rich's claim for damages for clinical negligence in the period leading up to her birth at the Defendant's hospital, the Hull Royal Infirmary, on 17th June 1993. The Court has previously ordered that the issues of liability and causation should be addressed at this trial, with the issue of quantum, should the need arise, falling to be determined subsequently.

2. The key issue in this case is whether corticosteroid drugs should have been given to Melissa's mother, Helen, before her delivery by emergency Caesarean section ("CS") at 32[1] weeks' gestation. The Claimant alleges that this failure caused or materially contributed to her developing post-natal Respiratory Distress Syndrome ("RDS") as a result of which she required mechanical ventilation. Consequently, so the Claimant's case runs, she suffered cerebral ischaemia resulting in cerebral palsy due to Periventricular Leukomalacia ("PVL").

3. The resolution of this issue, which itself sub-divides into a number of issues under the overall rubrics of breach of duty, factual and medical causation, turns principally on my assessment of complex lay and expert evidence in the fields of obstetrics and neonatal medicine. However, before coming to the disputed matters, I need to set the scene by setting out some essential background to this case.

Essential Factual Background

4. Helen Rich had a complex obstetric history which it is necessary to summarise because it is relevant to the degree of risk of pre-term delivery when she was pregnant with Melissa.

5. Helen Rich was born on 17th December 1965. Her first child, David, was born in May 1983 at full term following vaginal delivery. Her second child, Gareth, was delivered by CS at 37 weeks' gestation, on 20th September 1984, following signs of antepartum haemorrhage which the experts agree is likely to have been caused by placental abruption. It is also agreed that the risk of placental abruption was enhanced in any subsequent pregnancy. Her third child, Adam, was delivered by CS at 40 weeks, on 31st October 1985, after trial of labour failed to progress. The operation note records the lower segment of the uterus as being *"very thin"*. This factor increased the risk of dehiscence (i.e. separation of the uterine scar) or scar rupture, although it did not eventuate. There was no evidence on this occasion of any placental abruption, haemorrhage or dehiscence.

6. Helen Rich's fourth, fifth and seventh pregnancies all ended in early miscarriages.

7. Helen Rich's fourth child, Lauren, was born delivered by CS at 30 or 31 weeks, on 21st November 1987, following her admission to hospital with severe abdominal pain. The indication for emergency CS was pain consequent on possible abruption or

uterine rupture. However, the operation note records that there was no evidence of dehiscence of the scar or rupture of the uterus, and no signs of placental abruption.

8. Helen Rich's fifth child, Hannah, was delivered by CS at 33^5 weeks, on 8th May 1991, following complaints of severe, extensive pain. The pain was so severe that Mrs Rich would not allow the doctors to touch her lower abdomen. The operation note, as opposed to the antenatal notes, records that the reasons for the procedure were the three previous sections, the onset of labour, and suspected abruption of the placenta. The findings at operation included:

> "… no adhesions in abdominal cavity. Lower segment exposed and excised, thick, not vascular … Liquor clear, no evidence of fresh abruption. Placenta complete, signs of old abruption on small surface …"

The experts agree that on this occasion the lower segment of the uterus was thick, not thin – in contrast with the operative findings when Adam was born. It is also agreed that the reference to *"old abruption"* must be to a relatively mild placental abruption during the course of this pregnancy.

9. Helen Rich's ninth, and last pregnancy, was with Melissa. According to the booking sheet, the expected date of delivery was 11th August 1993 and she was to be under Consultant management. It was clear that the birth would need to be by CS, and on a date which cannot be deduced from the antenatal notes the mother was advised that the procedure might have to be undertaken at 37 or 35 weeks, owing to pain and the previous obstetric history. According to these notes, there was tenderness in the right iliac fossa as early as January 1993, and by March Helen Rich was complaining of abdominal discomfort on the right side of the scar similar to the symptoms she had experienced previously. On 13th April she had to be admitted to hospital with complaints of abdominal pain *"especially around scar"*. The pain settled and Helen Rich was discharged after two days.

10. On 18th May 1993 Helen Rich was seen for the first time by Professor David Purdie, Honorary Consultant Obstetrician and Gynaecologist at the hospital. He was not the Consultant allocated to Mrs Rich at booking, but nothing turns on this. His clinical note reads:

> "Well. Recurrent right abdominal pain under scar. To report stat[im] [i.e. immediately] if any acute onset pain with or without bleeding or if foetal movements decline sharply [or it might read "markedly"]."

Professor Purdie's letter to the GP reads:

> "… she continues to have recurrent lower right-sided abdominal pain, similar to the pain which she has experienced with her abruption and prior to her last emergency CS. There is no objective evidence of scar dehiscence, nor of abruption this time, but we will need to get a further scan done before she is reviewed in a week to check that there is no retroplacental collection.

> She is aware of the need for a further section this time and that this may again be premature. Please let us know at any time should problems arise. She knows to report to you at once should there be an acute onset of abdominal pain with, or without, bleeding or should foetal movements rapidly decline … Cons[ultant]/ S[enior] R[egistrar] only."

11. In his oral evidence, Professor Purdie explained that he believed that the origin of the pain was within the deep tissues underneath the skin incision rather than the uterine scar. His letter to the GP is consistent with that evidence.

12. On 19th May 1993 Helen Rich was admitted to the labour ward as an emergency, complaining of pain under the CS scar. According to her witness statement, which was not cross-examined, "*the pain must have been extreme for me to call an ambulance"*. On examination, she was found to be tense and tender over her lower abdomen, with the pain being most severe in the region of the left iliac fossa. By 16:30 the pain was noted as *"beginning to settle"*. Examination of the abdominal area showed that the scars were *"mildly tender"*. Helen Rich was then seen by a more senior doctor who confirmed the impression that there was no evidence of uterine scar rupture, and Tylex was prescribed. This is a paracetamol/codeine mixture which is recognised as being one step up in strength from paracetamol alone. By 23:00 the pain had settled and my interpretation of the midwifery notes is that she then slept well. Helen Rich was discharged home the following day.

13. On 25th May 1993 Helen Rich was seen in the antenatal clinic by someone who has not been identified. His or her clinical notes read as follows:

> "… now pain under all scar … lying at home all day – never goes out. Only up to toilet and children."

14. The phrase *"now pain under all scar"* is difficult to interpret, but read as a whole this brief clinical note tends to suggest that Helen Rich's pain was debilitating although it probably was not as acute as it had been on 19th May, given that she was not seeking re-admission to the ward. However, this is in the context of her having been prescribed fairly strong painkillers, which it is reasonable to infer that she had been taking, although I accept Mr Havers' submission that, given that there is no clinical record of the prescription, we do not know for how long. According to her witness statement, by then she had been in *"constant"* pain for over 6 weeks. However, I cannot regard this as an entirely reliable statement of the course, duration and intensity of the pain she suffered over two decades beforehand.

15. The clinical notes show that on 1st and 3rd June the midwifery team telephoned Helen Rich to arrange a further home visit (there had been one in April), but on both occasions there was no reply. This issue was not explored in evidence.

16. On 8th June 1993 Helen Rich attended the antenatal clinic and saw Professor Purdie. Her witness statement suggests that this was because her pain had worsened, but the inference I draw from the clinical notes is that this visit had been pre-arranged. According to Professor Purdie's note:

> "Still c/o right iliac fossa pain. Probably adhesions ± old scar."

According to the letter Professor Purdie wrote to Helen Rich's GP on the same day:

> "She is well but still has recurrent RIF pain which has caused such trouble during the pregnancy. Again, there is no clinical problem on abdominal examination today. The baby is well grown. Helen is normotensive and urine analysis is negative. I suspect that this is related to adhesions or to tensions of the old scar which, however, shows no sign of dehiscence. Should there be any acute onset of pain and/or bleeding she knows to report to you at once."

17. On 17th June 1993 Helen Rich recalls being in her kitchen at her old house: she turned round and felt a sudden pain in her abdomen. She says that the pain came on unexpectedly. An ambulance was called and Helen Rich was admitted to hospital. According to the midwifery notes:

> "Admitted with history of severe pain around the scar site, more on the right side of the lower abdomen at the right side of the scar … pinky discharge … previous two admissions with abdominal pain around scar site. Pain now much worse."

18. Helen Rich was examined and was found to be in a lot of pain, with extreme tenderness around the scar site. By the time the Registrar came to examine her, she was *"obviously in extreme agony"*. It was decided after Consultant involvement to perform an emergency CS, and Melissa was delivered at 16:49. The finding at operation was of scar dehiscence on the right side of the uterine scar, such that there was a *"palpable window"* of approximately 3cm. No inferences can be drawn as to when this scar dehiscence started.

19. Melissa's birth weight was 1,980 grams which is large for a baby born at 32[1] weeks. Her Agpar score after 1 minute was 8, which is good. However, at 17:45 a deterioration in her condition was noted, and at 18:00 she was intubated and placed in a ventilator, where she remained for five days. It is common ground that Melissa suffered from RDS which led to her needing to be ventilated.

20. Melissa's case is that PVL results from instability and reduction in brain perfusion. Put simply, premature infants have low blood flow to the periventricular areas, and small changes to cerebral perfusion can result in critically low perfusion to these areas and consequent ischaemia. Melissa's case is that it is likely that she was poorly perfused in the 70 minutes or so before she was intubated, and that there may have been smaller changes in perfusion pressure whilst she was being ventilated. Thus, Melissa's PVL or white matter damage was caused by (i) her RDS, and (ii) her need to be ventilated, with item (ii) flowing from item (i).

21. The Defendant's case was originally that Melissa's PVL was not caused by her RDS, or could not be shown to have been. However, on the fourth day of the trial Mr Philip Havers Q.C. for the Defendant sensibly abandoned that contention, having noted my provisional indication that I was struggling with it.

The Key Issues Arising

22. At paragraph 39 of the Skeleton Argument he lodged at the beginning of this trial, Mr Simeon Maskrey Q.C. for Melissa submitted that there were five key issues for me to resolve. The Defendant has conceded one of these, and in my view another passed out of the picture during the course of the hearing. In my judgment, the matter may be simplified still further and distilled into the following two key questions, both of which must be answered affirmatively if this claim is to succeed:

    (i) should Professor Purdie have considered prescribing a course of corticosteroid drugs when he reviewed Helen Rich on 8th June 1993 (I focus on that date, and not on either of the May dates, because Melissa cannot succeed on causation had her mother received corticosteroids on one or other of those occasions); and, had he done so, would or should he have recommended corticosteroids?

    (ii) on the balance of probabilities, would corticosteroid treatment on that date have avoided the RDS altogether or ameliorated it to such an extent that Melissa would have avoided PVL; or, alternatively, would have ameliorated it to a degree such that Melissa may properly claim that in law the entirety of her injury is attributable to the Defendant's tort?

23. The sub-issues I adverted to under paragraph 3 above will be addressed under the overall umbrella of these two issues. To the extent necessary, it will be convenient to discuss the factual causation questions Mr Maskrey adeptly deployed in his closing submissions under the banner of the first issue.

A Brief History of Developing Clinical Practice in the Use of Corticosteroids

24. The parties have collected a considerable weight and volume of academic literature bearing on the issues of the evolution and effectiveness of corticosteroid use for pregnant mothers at risk of delivering their babies pre-term. Given, however, the measure of common ground which was achieved during the course of the trial, I may take the issue of developing clinical practice in this domain quite briefly. The related issue of the effectiveness of this intervention deserves a fuller treatment under a separate rubric.

25. Because the foetal lung matures late in the process of gestation, pre-term babies are at high risk of pulmonary problems. RDS has been, and perhaps still is, the commonest complication of pre-term birth: in the absence of any prophylactic measures, affecting over 50% of babies born before 34 weeks' gestation. In the past, many of these babies died, but the advent of mechanical ventilation has improved survival rates, albeit not being without its own risks.

26. The pharmacological and physiological basis of treatment of the expectant mother with corticosteroids is that these powerful agents mature the foetal lungs *in utero* thereby reducing the risk of pulmonary deficit and RDS at pre-term delivery. Nowadays, surfactants are often also used post-natally to achieve the same outcome, but these agents were not deployed in Melissa's case (non-negligently) and I was told that they are far more expensive than corticosteroids.

27. In 1969 Dr Liggins published a paper which showed that prematurely delivered lambs exposed prenatally to corticosteroids survived longer than their placebo-controlled counterparts. He hypothesised that the reason for this may have been that which I have already sought to encapsulate. Subsequently, he mounted a randomised, placebo-controlled trial of antenatal betamethasone administration in expectant mothers, and demonstrated statistically significant reductions in RDS in babies born before 32 weeks' gestation (Liggins and Howie, 1972). Dr Liggins' work has been internationally recognised as trailblazing in the field of obstetrics.

28. Since this pioneering work, numerous further investigations have indicated benefit from antenatal administration of those corticosteroids that cross the placental barrier: for present purposes, I am limiting the discussion to betamethasone and dexamethasone. It is unnecessary for me to examine these investigations. In 1989, Oxford University Press published what came rapidly to be regarded as a leading textbook on obstetrics in the UK, Effective Care in Pregnancy and Childbirth, edited by Chalmers, Enkin and Keirse. Chapter 45 on Promoting Pulmonary Maturity was written by Dr Patricia Crowley, another pioneer in her field, practising in the Republic of Ireland. She analysed and synthesised the relevant literature, and concluded that clear evidence of benefit had been established. Her conclusion was as follows:

> "This reduction is of the order of 40-60% and is independent of gender. Furthermore, the benefit of antenatal corticosteroids appears to apply to babies born at all gestational ages at which RDS may occur. While the greatest benefits are seen in babies delivered more than 24 hours and less than 7 days after commencement of therapy, babies delivered before and after this optimum period also appear to benefit … the benefits of antenatal corticosteroids have been established. No further trials are necessary with the exception of certain specific situations … or to establish other dosages or routes of administration."

29. Witnesses were also taken to the paper by Crowley et al entitled The Effects of Corticosteroid Administration before Pre-term Delivery: an Overview of the Evidence from Controlled Trials [*British Journal of Obstetrics and Gynaecology*, January 1990, Vol. 97, pp. 11-25]. I do not understand the findings and conclusions of this review paper to differ materially from Dr Crowley's textbook chapter. However, the introductory section of the paper did point to the wide variation in clinical practice in the UK and elsewhere, with only 42% of senior obstetricians using this treatment *"frequently"*.

30. Another leading light in this field is Professor Richard Lilford, currently Professor of Public Health at Warwick University but between 1985-1992 Professor of Obstetrics and Gynaecology at Leeds University. In 1992 he was appointed chair of the Audit Committee of the Royal College of Obstetrics and Gynaecology. He gave evidence before me, and explained that during the currency of his chairmanship of that committee he was instrumental in producing a bundle of 21 guidelines relating to separate aspects of obstetric care. One of these covered the use of prenatal corticosteroids. Unfortunately, it has not proved possible to locate a copy of the guideline promulgated by the Audit Committee, and there is some doubt as to exactly what it said and the timing of its publication; but in my view nothing really turns on

this. In or shortly before December 1992 the Scientific Advisory Committee ("SAC") of the RCOG published a document entitled Antenatal Corticosteroid Administration Reduces the Incidence of Neonatal RDS. This did no more than summarise the findings of the Crowley chapter (see paragraph 28 above) and another paper, but warned clinicians to guard for contra-indications. The recommendation was as follows:

> "Recommended Drug Regimen
>
> Betamethasone 4mg or Dexamethasone 4mg, twice a day for two days by intramuscular injection.
>
> WE WOULD ENCOURAGE ALL OBSTETRIC UNITS TO CONSIDER THE USE OF SUCH THERAPY WHEN DELIVERY IS LIKELY BEFORE 34 WEEKS."

31. For present purposes, I am content to infer, having due regard to Professor Lilford's oral evidence, that the Audit Committee's guideline (as he characterised it) was either identical to or not materially dissimilar from the SAC's. I do not exclude the possibility that Professor Lilford's memory is playing tricks with him, and that there was only one document – the SAC's. If there was, I should make clear that one way or another Professor Lilford was the spur to its genesis.

32. The attention of Fellows and Members of the RCOG was drawn to the SAC's recommendation in the President's letter dated December 1992. This was under the overall rubric, Items of Interest from the SAC during 1992. The capitalised part of the SAC's recommendation was emboldened in the text of the letter.

33. According to a note published online by the Wellcome Trust:

> "The guidelines seemed to do the trick. Published in 1992, almost overnight they led to a huge change in medical practice. Bizarrely, though, doctors not only took up steroids but began to use higher or multiple doses, without any evidence that this was effective or safe …"

34. Both Professor Lilford and the Wellcome Trust have described the SAC document as "guidelines", which the Defendant says is a misnomer. Technically, so its argument proceeds, the RCOG did not publish any guidelines *stricto sensu* until April 1994, being the first of the "green top" guidelines mentioned in the expert evidence. In April 1996 the RCOG published its seventh guideline, entitled Antenatal Corticosteroids to Prevent RDS. Its introductory section stated, referring to an unpublished work, that the use of antenatal therapy has been hesitant. The use of the conjunction *"however"* suggests that this hesitancy ran counter to the robust evidence base.

35. The following points emerge from a consideration of this seventh guideline. First, the optimal period for treatment was within a "window" of 1-7 days before delivery, although there was a trend towards benefit both before and after this period. Secondly, the guideline recognised that a practice (by no means universal) had arisen whereby repeated courses of the drug were being administered without there being a good evidence base for it. The guideline stated that decisions to repeat the treatment should

be made on a case-by-case basis. Thirdly, the indications for treatment were expressed to be as follows:

> "Every effort should be made to initiate antenatal corticosteroid therapy in women between 24 and 36 weeks' gestation with any of the following:
>
> Threatened pre-term labour
>
> Antepartum haemorrhage
>
> Pre-term rupture of the membranes
>
> Any condition requiring elective pre-term delivery [there was some discussion with Mr Hare as to exactly what this means, but ultimately it seems to me that I do not have to resolve this issue]
>
> As pregnancy advances, the number of women that will have to be treated with corticosteroids to prevent a single case of RDS increases."

Fourthly, the guidelines set out a series of contraindications and precautions, none of which are relevant to Helen Rich's pregnancy. Fifthly, the dose and route of administration was *"two doses of betamethasone 12mgs given intramuscularly 24 hours apart or four doses of dexamethasone 6mgs given intramuscularly 12 hours apart".* Finally, the guideline included the following standard caveat:

> "This guideline was produced under the direction of the SAC of the RCOG as an educational aid to obstetricians and gynaecologists. This guideline does not define a standard of care, nor is it intended to dictate an exclusive course of management. It presents recognised methods and techniques of clinical practice for consideration by obstetricians/gynaecologists for incorporation into their practices. Variations of practice taking into account the needs of the individual patient, resources and limitations unique to the institution or type of practice may be appropriate."

The First Issue: Breach of Duty

Corticosteroids: The Standard of Care in 1993

36. On one reading of paragraph 21(1) of the Defence it was being suggested by Mr Havers that there existed a responsible body of medical opinion in 1993 which would not have supported the use of corticosteroids such that in Bolam terms it was not mandated. Whereas it is correct that the use of these drugs was not universal across maternity units in the UK, I cannot accept that it would have been rational to exclude their administration; and, to be fair to the Defendant, that was not ultimately its case. The combined effect of the evidence of Professor Lilford, Mr Simon Tyrrell and both

obstetric experts (Mr John Hare FROCG for the Claimant and Mr Richard Porter FRCOG for the Defendant) was that by 1992 at the latest the evidence base was sufficiently robust that the use of corticosteroids ought to have been considered by any competent obstetrician in cases of pre-term delivery. I rather liked the way Mr Hare put the point, namely that in the early 1990s it took time for obstetricians to get their act together.

37. In these circumstances, it is otiose for me to address in any detail the Defendant's semantic argument that the 1992 SAC document was not a "guideline", and was therefore not mandatory. Whereas I entirely agree that the SAC document did not explain the circumstances in which corticosteroids should be administered, beyond the very general recommendation that a single course should be considered if delivery was likely at less than 34 weeks, it seems to me that debate about the difference between "guidelines", "guidance" and "recommendations" is somewhat arid. In answer to my question, Professor Purdie agreed that the normative force of the 1992 and 1996 documents was *"very similar"*. In any event, the point becomes even more arid in the light of Mr Tyrrell's evidence that the use of corticosteroids was standard practice at this hospital since around 1989.

38. My finding that corticosteroid drugs ought to have been on any reasonably competent obstetrician's palette of interventions in cases of pre-term delivery is not, in my judgment, sufficient for Melissa's purposes. The circumstances in which these drugs should have been considered are an inseparable part of the discussion.

39. The real debate concerns the timing of administration. Helen Rich was a good candidate for the use of corticosteroid drugs – and in her case Professor Purdie told me that the drug of choice would have been betamethasone – because it is accepted that there was a real risk that she would deliver pre-term, and in her case there were no contraindications. However, given that (i) the practice at this hospital was to administer only one dose (and Mr Hare accepts that this was not sub-standard), and (ii) the optimal benefit was in the 1-7 day "window" between treatment and delivery, with a trend towards some benefit both before and after, it is obvious that an element of clinical judgment is required. Give the dose too late and the baby will be delivered before 24 hours has elapsed, with the benefit being (at best) sub-optimal. Give the dose too early and the mother does not deliver within 7 days, with the optimal benefits being wasted. Beyond specifying the 34 week temporal boundary, the 1992 documents said nothing about circumstances and timing, presumably leaving it to clinicians to devise their own protocols and practices in this respect.

<u>The Evidence of Professor Lilford and Mr Tyrrell</u>

40. I heard interesting and impressive lay evidence from Professor Lilford and Mr Simon Tyrrell on this issue. Although the SAC recommendation used the recommendation *"likely"*, Professor Lilford told me that in his best judgment none of his colleagues would have interpreted that to mean *"more likely than not"*. *"Likely"* in many contexts is a synonym for *"probable"*, but in a clinical context it conveys the idea of *"significant degree of likelihood"*; in other words, it is more attuned to the concept of risk than any balance of probability. This is how Professor Lilford put the matter in more concrete terms:

> "One way of interpreting 'likely' is, a material risk of the birth taking place within the period of time the steroids are believed to be active. I would have recourse to how doctors generally make decisions: on the basis of a calculus, albeit intuitive, of the ratio of benefits to the risks."

41. Professor Lilford further explained that in his view a risk of between 5-20% would be sufficient to trigger the prescription of the drug, but on my understanding of his evidence he would countenance some upwards adjustment of this band to reflect divergences of view amongst his colleagues. Professor Lilford added that the 5-20% bracket was his *"Bayesian prior probability"*. By this he meant, or I think he meant, the generic probabilities before considering the circumstances of the individual case. When he was asked about those in cross-examination, and in particular the role of clinical judgment, he said this:

> "A. The particular case has more influence on the probability that pre-term labour may occur. The particular case has less bearing on whether or not you give the steroids, given the probability.
>
> Q. Should it not all come down to the individual case?
>
> A. This is the big question for evidence-based medicine. You must consider the average effect. It is a decision in the individual case informed by the epidemiology."

In re-examination, Professor Lilford was asked to clarify the somewhat gnomic terms of the first of his answers cited above, and he did so in this way. The determination of whether a degree of probability in an individual case has been attained is a matter of clinical judgment. Separately, the determination of what level of probability must exist before any particular treatment is warranted or mandated is determined by the evidence base as explained by any relevant guideline. The default assumption is that the clinician administers the recommended treatment unless there is a contraindication.

42. If, in re-examination, Professor Lilford was being led by Mr Maskrey into saying that in the present situation the decision-making process possessed two separate stages, I would not altogether agree with that. I would agree that in many clinical situations a clear guideline may set out the general circumstances in which an intervention should be considered, leaving the practitioner to exercise his or her clinical judgment in the individual case as to whether (a) those circumstances exist, and (b) this individual patient has characteristics which indicate a different approach. However, I reiterate the obvious point that in the present situation the SAC recommendation is very open–textured. Beyond advising that corticosteroids should be considered if delivery is likely before 34 weeks, no further assistance to clinicians is given. Specifically, the SAC recommendation is of no real assistance in addressing the temporal conundrum I outlined under paragraph 39 above.

43. But what I do take from Professor Lilford's evidence is that *"likely"* does not mean *"probably"*, and that in his view if there is a real risk of delivery within the stated window of opportunity (which risk he puts at 5-20%), then corticosteroids should be

actively considered. Beyond saying that this was a decision to be made in the individual case, Professor Lilford was not precise as to whether there had to be any specific indications for delivery within this window. His evidence was somewhat more general: the risk has to be evaluated in the individual case, and a clinical judgment then exercised.

44. I suspect that the reason why Professor Lilford was not asked to be more precise was that Mr Maskrey well knew that there were constraints on how far a lay witness could properly opine on an issue of this nature. I should not therefore be understood as criticising Professor Lilford in any way. It would also be fair to say that in making his careful and insightful contributions he did occasionally overstep the boundary between fact and opinion evidence, but this too was not his fault.

45. Mr Simon Tyrrell FRCOG, currently working as a Consultant at the Gladstone Hospital, Queensland, Australia, also gave lay evidence, but on behalf of the Defendant. Between December 1989 and 2014 he worked as a full-time Consultant at Hull Royal Infirmary having previously been junior to Professor Lilford at Leeds. He agreed that by 1990 it was evident that corticosteroids had a beneficial effect in cases of pre-term delivery before 34 weeks. The known risks were minimal. Accordingly, he disagreed with paragraph 21(1) of the Defence, or – more precisely – one of the interpretations I have placed upon it at paragraph 36 above. Indeed, in 1993 he said that all consultants in Hull shared his point of view, and would therefore have subscribed to the opinion that corticosteroids should be given within 1-7 days of delivery.

46. Mr Tyrrell was of great help to me on the issue of timing. Given that the unit in which he worked did not administer multiple doses, he and his colleagues were naturally alive to the conundrum I have previously identified. Mr Tyrrell emphasised that the factors bearing on the exercise of clinical judgment in an individual case were complicated. It was difficult to express the risk or degree of likelihood in a verbal formulation, because words mean different things to different people. He agreed with Mr Maskrey's "5-20%" band, subject to this important qualification:

> "The decision depends on the available evidence, the clinical findings and your experience. It is too simplistic to be prescriptive about this. In most cases, it is a matter of clinical judgment. If you believe that delivery is likely within the next 7 days, then steroids should be given … I agree that a risk of 5-20% would have caused me to give very considerable thought to the use of steroids, but I would add to this that you need a clinical suspicion to give that number … you need some evidence to support that view – that you are likely to want to make a decision to carry out a CS, or some evidence that labour is about to happen. This must be part of the clinical picture." [here, I have amalgamated parts of Mr Tyrrell's cross-examination and re-examination, re-ordering his evidence as appropriate]

47. Mr Tyrrell added that what he called the prior risk of pre-term delivery did not matter much, because *"you warn, but ultimately you wait"*. He agreed with Mr Maskrey that policy and practice at Hull would create difficulties if the clinician believed that an

emergency CS would be required but he did not know when: once the emergency arose, there could be no opportunity for the corticosteroids to take effect. At the end of the day, this was a *"very imprecise art"*.

48. Mr Tyrrell's final position in his oral evidence may not have been quite the same as the formulation he advanced under paragraph 10 of his witness statement, which predicated *"a clinical suspicion that labour is or was starting or if it appears likely that the obstetrician will advise iatragenic premature delivery because of a pregnancy complication"*. The second limb of this clause, after the conjunction *"or"*, may be variously interpreted, not least because *"likely"* in this context could be read as synonymous with *"probable"*. On the other hand, Mr Tyrrell used the adjective *"probable"* in re-examination, in the context of having previously accepted that a 5-20% risk, taken together with a clinical suspicion, would suffice. Yet it could be argued that once the clinical suspicion is fed into the decision-making process, what started off in Bayesian terms as being a prior 5-20% risk has *in fact* been turned into a much higher risk, akin to probability. If, for example, there are signs of pre-term labour, delivery is probable within 7 days. If a clinician believes on account of specific clinical signs, that a CS will be required, in very many cases it will be performed in the near future.

49. The difficulty with this type and level of analysis is that it is artificial to reduce clinical judgments to the sort of exact formulations to which lawyers aspire. I will need to return to this point when returning to a final evaluation of Mr Tyrrell's evidence in the context of the expert evidence I heard.

50. Finally, I should make clear in relation to Mr Tyrrell's otherwise clear and helpful evidence that I must discount his suggestion, as per paragraph 9 of his witness statement, that in this hospital in 1993 corticosteroids *"were not offered to a woman in the situation Helen Rich found herself in"*. Mr Tyrrell is entitled to speak about general practice, but in my view he cannot opine about an individual case.

<u>The Witness Statements of Professor Purdie</u>

51. Professor David Purdie FRCP was Honorary Consultant at Hull Royal Infirmary between 1989 and 2004. He retired from obstetric practice in 1994 to concentrate on gynaecology. He also held an academic post at the Hull-York Medical School over the same period. His first witness statement is dated 24th January 2013 and therefore post-dates the Particulars of Claim, which contains the core allegation that corticosteroids ought to have been administered pursuant to RCOG advice. Professor Purdie's witness statement is quite brief (save for setting out what the medical records say), and makes clear that he could not now recall whether maternal steroids were being given in 1993. The overall tenor of paragraphs 9-11 of this statement is that had he thought that delivery was imminent or impending, or that there was an immediate risk of this, he would have admitted Helen Rich for observation, and steroids would have been considered *"subject to local practice"*. Given that Professor Purdie could not apparently say in January 2013 whether it *was* local practice in Hull to give steroids, the reasonable inference to draw is that the factor driving any decision to admit was the condition of the mother – in the context of the likelihood of her delivering – rather than any need to administer steroids. However, I take the point that

if it was local practice to administer steroids in these circumstances, then Helen Rich might have benefitted from that policy.

52. I also have some difficulty with paragraph 12 of Professor Purdie's witness statement, which provides:

> "… My understanding is one might give steroids to mothers with babies prior to 32 weeks. Further, my use of the words, *"may again be premature"* [letter dated 18th May 1993] is quite distinct from me expressing any concern about whether there was a significantly increased risk of delivery before 32 weeks. After this point, my understanding is one gives surfactant and manages them in the neonatal period."

I asked Professor Purdie whether the reference to *"32 weeks"* was a typographical error, and he told me that it was not. I find this surprising, given that the SAC advice, which as an obstetrician with an academic background he must have known about, clearly refers to 34 weeks. It is quite true that the Liggins et al paper refers to 32 weeks, but subsequent literature goes up to 34 weeks if not beyond that. Furthermore, I am also surprised that Professor Purdie appears to believe that, given Helen Rich's history, there was no enhanced risk of delivery before 32 (or 34) weeks.

53. On 10th December 2013 Professor Purdie filed a supplementary witness statement. By this stage, he clearly had seen the President's letter and the SAC document, and confirms that he would have seen it before May 1993. Accordingly, Professor Purdie believes that it is very likely that he was aware of the potential benefits of steroids at the material time, and therefore would have been prepared to consider their use in appropriate cases. Paragraphs 9-13 of Professor Purdie's second witness statement merit scrutiny. In contrast with his first witness statement, he now agrees that the relevant window of opportunity was 34 weeks, not 32 weeks. In these paragraphs, Professor Purdie appears to use the epithets *"immediate"* and *"imminent"* interchangeably. Likewise, he appears to equate *"an immediate threat of delivery"* with *"imminent delivery as a consequence of premature labour"*, thereby excluding the possibility of premature delivery by CS. Save for her 1987 pregnancy with Hannah, premature labour was not a feature of Helen Rich's previous history, and in any event the plan was to deliver by CS. Paragraph 12 of Professor Purdie's second witness statement encapsulates his position (at least, in December 2013):

> "In summary, I believe when I saw the Claimant's mother in May and June 1993 I would have considered, but rejected, steroids because I did not consider that premature labour with delivery before 34 weeks was likely. Even if I had considered using them I would not have done so unless and until delivery was imminent."

54. No accusation could fairly be raised against the Defendant's legal team that Professor Purdie's second statement has been heavily "lawyered". Taken at its face value, it far from assists the Defendant's case. In reality, Professor Purdie would not apparently have considered corticosteroids unless and until delivery was about to happen. The risk is not 5-20%, or thereabouts, but closer to 100%. Of course, if delivery is about to

happen, there could be no opportunity for enough time to elapse to permit corticosteroids to attain any significant benefit.

55. Given that Professor Purdie's second witness statement does not even mention the possibility of pre-term delivery by CS, it may be difficult to infer the circumstances in which corticosteroids might have been considered in that context. One possible approach might be to say that Professor Purdie simply would not have considered corticosteroids in the event of delivery by CS, but that would be completely illogical: so illogical that he could not have thought it. On the other hand, it is an approach which I cannot necessarily exclude. In any event, if Professor Purdie should be given the benefit of the doubt, then on the basis of his second witness statement it seems that he only would have considered corticosteroids if delivery by CS were imminent. That, as before, would have been too late.

Professor Purdie's Oral Evidence

56. When he entered the witness box, Professor Purdie did not seek to revise or correct his witness statements in any way. However, during the course of his extended cross-examination by Mr Maskrey, Professor Purdie qualified, corrected, retracted from and supplemented his witness statements in a number of respects, without necessarily being aware that he was doing so. First, he made clear that he was not seeking to suggest that 32 weeks' gestation was the cut-off point, and that the practice was to exhibit steroids prior to 37 weeks if the clinical presentation warranted this course. However, he did not give a satisfactory explanation for the clear wording of his first witness statement. Secondly, he tried to defend his failure to mention the President's letter and Hull's policy (as clearly explained to me by Mr Tyrrell) in his first witness statement. At one stage he said that he was not sure that this was relevant and important evidence to give. Eventually, he was compelled to agree that it was a slightly surprising omission not to refer to the President's letter. Thirdly, he said that by *"imminent"* he in fact meant *"within 1-7 days, being the window of opportunity"*. This he told me was the policy of the unit in 1993. Given, too, that the drugs would be administered only once, the policy was conservative, with clinicians hoping to achieve increasing maturity of the foetus. Fourthly, Professor Purdie told me that he rather regretted confining his second witness statement to pre-term labour, and not expressly mentioning pre-term CS.

57. During the course of his cross-examination, Professor Purdie's evidence became more precise and concrete. He had faced a close series of questions addressing the temporal conundrum I have previously mentioned. Quite late on in his cross-examination, he said this:

> "There was a discussion in the department as to the triggering circumstances [this is my paraphrase]. There was a Consultants' meeting where the issue was discussed. I reviewed the literature, and came up with a set of indications:
>
> - incipient pre-term labour [Professor Purdie explained elsewhere that there could be prodromal signs from which it

could be deduced that the establishment of labour was to be anticipated]

- scar dehiscence
- antepartum haemorrhage
- membrane rupture
- elective pre-term delivery"

58. Professor Purdie made clear that this was an informal discussion amongst senior staff rather than the prologue to the formulation of any protocol. In answer to my questions, Professor Purdie explained that some of these indications would not necessarily require immediate CS: for example, an apparently slow scar dehiscence might not. Thus, in such circumstances there might well be time to administer corticosteroids within the window of opportunity. It follows that these indications leave open the possibility of administering steroids before it would become necessary to perform an emergency CS.

59. This Consultants' meeting receives no mention in Professor Purdie's witness statement, nor did he seem to recall it at any earlier point in Mr Maskrey's cross-examination. I must ask myself how much reliance I may safely place on Professor Purdie's apparently late recollection. I discount altogether the possibility that Professor Purdie has simply made this up, and Mr Maskrey did not suggest that he did. One possibility is that Professor Purdie's witness statements were somewhat perfunctorily assembled by him in circumstances where he is long-since retired, irritated by the fact that his practice has been called into question, and that he is now being asked to ransack his memory for the fine detail of what occurred many years ago. If this is the valid explanation for what has occurred, I might be prepared to find that the witness statements fail to do justice to Professor Purdie's evidence. Another possibility is that in attempting an *ex post facto* reconstruction of what *his* practice (as opposed to Hull's practice) was, he has subconsciously permitted himself to misremember what exactly occurred, and when.

60. Professor Purdie agreed that he would consider whether to give corticosteroids if he assessed there to be a risk of delivery within 34 weeks. Later in his cross-examination, he clarified his practice as follows:

> "In a patient with this history, premature delivery is always a possibility. In any consultation, the possibility of using steroids exists, in the background. I would only give active consideration, or bring the issue to the foreground, if the clinical situation mandated it; i.e. the occurrence of one of the complications of pregnancy which made delivery within 7 days likely."

61. If this indeed represented Professor Purdie's practice in 1993, then – subject possibly to a terminological issue as to the difference between background and foreground considerations - it would have been very similar to Mr Tyrrell's. Again, however, I must consider whether this was so.

62. Professor Purdie was asked about Helen Rich's obstetric history. He agreed that the earlier CSs (including pre-term CSs), the two placental abruptions, the finding at operation of a very thin uterine wall in 1985 (albeit not subsequently), and the history of pain were relevant and important factors in assessing the risk in the index pregnancy. Nonetheless, I would observe at this juncture that these are all fairly "soft" factors whose relevance and, in particular, quantification (in terms of the risk in the instant case, and the timing of its coming closer to fruition) must ultimately turn on clinical judgment.

63. When Helen Rich saw Professor Purdie on 18th May 1993, he knew that she had been complaining of abdominal pain since April, if not before. This was similar to the pain experienced in previous pregnancies, although it was not as severe. He appreciated that there was a risk of premature delivery in this pregnancy, which could occur at any time. His clinical judgment was that the pain was referable to a location deep underneath the skin incision and not the uterine scar. There was no objective evidence of scar dehiscence. Given that Mr Hare no longer contends that the administration of corticosteroids on 18th May was mandatory, I do not have to examine this consultation any further.

64. Professor Purdie did not of course examine Mrs Rich on 19th or 25th May, but the clinical notes relating to these dates were available to him on 8th June. Thus, his interpretation of them is relevant for present purposes. As for 19th May, he pointed out that Mrs Rich's pain settled quite quickly and that he was aware of the analgesia she was given. As for 25th May, the notes do not make clear how severe the pain was, although Mrs Rich's mobility was clearly compromised. The notes also do not make clear whether she was still taking Tylex. I have already pointed out that the note, *"now pain under all scar"*, is not easy to interpret, but Professor Purdie told me that he presumed that it meant under the scar itself. His note of 18th May states that the pain was both right-sided and under the scar, and so whether it had migrated on 25th May is open to question. The rudimentary abdominal sketch carried out on 13th April shows two transverse abdominal scars with their mid-points over the mid-line of Mrs Rich's stomach. It follows that the reference to *"pain under all scar"* was intended to indicate that the pain had now extended to the mid-line and left-side area.

65. When Professor Purdie saw Helen Rich on 8th June, he was aware that she had not returned to the hospital for any reason since 25th May. His assessment was that the pain was more or less in the same location as it had been on 18th May. He told me that he thought that the presentation amounted to *"a continuation of the clinical findings on 18th May"*, and that Mrs Rich was no worse on 8th June than she had been when he last saw her.

66. A key issue it seems to me is the location and source of Mrs Rich's pain on 8th June. Professor Purdie agreed with the general proposition that an increase in uterine pain, as opposed to abdominal pain, increases the risk of pre-term delivery. It was put to him that Helen Rich must have been experiencing uterine pain on 8th June. His answer was as follows:

> "Pain from a uterine rupture does not settle. Pain from scar dehiscence continues, until delivery. We had no objective evidence of scar dehiscence at this point."

67. According to Professor Purdie's note, this was recurrent pain. According to his letter to the GP, he examined his patient. In answer to Mr Maskrey's questions, Professor Purdie was more specific:

> "My view on 8th June was that this was adhesion pain. Previous abdominal surgery is implicated in adhesions. The pain is RIF, not over the uterus and not over the position of the [uterine] scar. Pain was uniquely in the RIF. I had a suspicion that adhesions were the cause of the pain. It was not possible to get a clinical conformation of that. It is significant that it was not pain over the [uterine] scar."

68. If the pain was in the same location as it had been on 18th May, I have already made the point that on one interpretation of Professor Purdie's clinical note it was under the right-side of the abdominal scar or scars. That does not necessarily mean that the pain related to the uterine scar. On the other hand, Professor Purdie's letter to the GP said that he suspected that the pain was related to *"adhesions or to tensions of the old scar which, however, shows no sign of dehiscence"*. If Professor Purdie's grammar is correct, this reference to the old scar must be to the uterine scar. Further, I understand that there was only one of these scars, not two (c.f. the abdominal scars).

69. I will be returning to these difficult and vexing issues later in this judgment.

The Expert Evidence of Mr John Hare FRCOG

70. Mr Hare took early retirement from clinical practice in 1998 and since then has worked as a mediator, has written a number of articles, and has written medical reports for the court. In the last ten years, his instructions have almost invariably come from Claimants, but I agree with Mr Maskrey that this is a jury point. Mr Hare was a clear and impressive witness. Ultimately, the points of distinction between his evidence and Mr Porter's may be clearly identified, in which circumstances I may take his evidence more succinctly than I did Professor Purdie's.

71. Having said that Mr Hare was a clear and impressive witness does not mean that I accept all his evidence. The strength of his premises, and his reasoning, merit careful examination.

72. Mr Hare's report dated 13th February 2014 was written on the premise that in 1993 the average Consultant would in his opinion have given repeat doses of corticosteroids. Mr Hare now accepts that the practice in Hull – namely, to prescribe a single dose – was not sub-standard in the Bolam sense. It follows, as Mr Hare agreed with Mr Havers, that if one is confined to a single dose, the balancing exercise which is required, in the context of the temporal conundrum I have described, becomes more difficult.

73. Mr Hare's oral evidence was that the threshold for prescribing corticosteroids was along the lines of a real risk of delivery before 34 weeks. That risk would be assessed in the instant case by reference to the previous obstetric history and the manner in which this pregnancy had developed. For Mr Hare a risk in the region of 10% would

be sufficient. He agreed that he had not derived this test from the 1992 documents or from the literature; rather, it was a clinical opinion shared by his colleagues. Mr Hare agreed that his position was not the same as Professor Lilford's in that the latter would have acted on the basis of a 5-20% risk of delivery within the next 7 days.

74. The question does arise as to the precise extent to which Mr Hare's test needs modification if the practice of a unit is not to administer repeat doses.

75. Mr Hare's original view had been that Professor Purdie should have prescribed steroids as early as 18th May (see the summary section to his report). He no longer holds that view. His advice to me was that it was sub-standard practice not to prescribe steroids on 19th May, 25th May and 8th June. Yet, until a very late stage in his oral evidence it was not clear to me why Mr Hare had revised his opinion in relation to Professor Purdie's first consultation. On the one hand, I can see that if the clinician intends to administer only one dose, the test for intervention becomes stricter – in other words, the quantum of risk must be higher. Yet, Mr Hare did not appear to embrace this logic because he told me that if a clinician is not prescribing multiple doses, then he would require a 10% risk within the 1-7 day window of opportunity. This, to my mind, is the same test as the one he would apply for multiple doses; the only adjustment to it is the 1-7 day period. Arguably, the application of this test renders intervention *less* likely because, other things being equal, the risk of delivery within 1-7 days must be lower than the risk of delivery before 34 weeks - *unless,* of course, there is a clinical indication for delivery within that timescale. Mr Hare said at the Joint Experts' Meeting that in Mrs Rich's case what I take to be the *ex ante* probability of delivery within 34 weeks was in the region of 50%. Further, Mr Hare accepted that if Professor Purdie would only have given a single dose, then on 18th May there was no clinical indication for delivery within the next 1-7 days. On the other hand, Mr Hare was far from conceding that the presence of a clinical indication was a necessary component of the test for intervention.

76. Towards the end of his evidence, and in answer to my question, Mr Hare said this:

> "If only one dose was to be given, it should have been on 8th June. Apart from the incidents in April, there was an episode of pain in May which was worse on 19th May. At that point, she was given strong analgesia. On the following morning, she was complaining of less pain, but the scar was still tender. Tylex is prescribed [off licence] only if the pain is considerable. During that week, she was in considerable pain; she was virtually immobilised. This is highly indicative – considerable pain, despite the Tylex. On 25th the Senior Registrar noted *"now pain under all scar"*; this could be a progression. He was unable to determine the presentation of the baby or the level of the presenting part. Almost certainly this was because the abdomen was too tender, and he couldn't palpate. On 8th June there was no record of tenderness. [Professor Purdie] has omitted to record the signs on palpation, or the state of the foetus. I cannot accept that the scar pain has gone away; it hasn't been recorded. She had been on strong analgesia for two weeks. Scar pain is a herald of dehiscence or rupture. These features

> mandated recognition of the likelihood, that is 10% risk, of delivery within 7 days."

In my view, this is the high watermark of Melissa's case on breach of duty and, should it arise, factual causation. There are three matters I need to add to the mix. First, Mr Hare is not requiring the presence of any *specific* clinical indication for delivery within the next 7 days. Instead, his approach involves looking at the presentation more broadly – as was put in closing argument, more holistically. Given the past obstetric history, the longitudinal course of the index pregnancy, and the overall clinical picture on 8th June, the position had been reached whereby intervention became mandatory. Secondly, Mr Hare made clear elsewhere in his evidence that he did not accept that Professor Purdie's clinical note and GP's letter written on 8th June were comprehensive and/or accurate. However, he did accept that if these documents were taken at their face value then, without prejudice to his contention that none was required, on 8th June there was no specific clinical indication of delivery within 7 days. Thirdly, I did not understand Mr Hare to be abandoning his contention that it was sub-standard practice not to administer corticosteroids on 19th or 25th May. It was implicit from his extended answer to my question that the case for intervention was even stronger on 8th June.

77. Mr Hare gave detailed evidence as to the correct interpretation of the contemporaneous clinical notes. I will take this account in reaching my conclusions on breach of duty.

The Expert Evidence of Mr Richard Porter FRCOG

78. Mr Porter has worked as a Consultant at the Royal United Hospital, Bath since 1989. He is an extremely experienced Obstetrician and Gynaecologist, and once he had got over Mr Maskrey's initial fusillade, he gave his evidence in a measured and assured manner.

79. Before turning to the essential features of his evidence, I should address the concern Mr Maskrey raised in his closing submissions. I have already been heavily critical of Professor Purdie's witness statements, and I would expect any experienced Consultant Obstetrician and Gynaecologist to have shared those concerns. However, at no stage before trial, or even in his evidence in chief, did Mr Porter see fit to advise me accordingly. One of the questions put to the experts in their Joint Meeting was directed to paragraph 10 of Professor Purdie's second statement and the assertion that *"I would have considered use of steroids if I felt it was likely there would be an imminent delivery as a consequence of premature labour"*. The question was directed to the point that Professor Purdie appeared to be limiting his practice to premature labour, thereby excluding the possibility of pre-term CS, and whether that mattered. Mr Porter's answer was as follows;

> "No … it is for Professor Purdie to expand on this matter and specifically to address the implied assertion that he would only consider prophylactic antenatal corticosteroids if there were contractions."

I have no difficulty whatsoever with Mr Porter's *"no"* (which is correct), but in my view he should have commented on what he called Professor Purdie's *"implied assertion"*. In his oral evidence, admittedly under considerable pressure from Mr Maskrey, Mr Porter tried to persuade me that the implied assertion he was referring to was that implicit in the Solicitors' question, not in paragraph 10 of Professor Purdie's witness statement. I regret to say that in my judgment Mr Porter was being disingenuous.

80. In relation to the Joint Experts' Meeting, I also criticise Mr Porter for his answer to question 3 on the Claimant's agenda –*"can the experts agree that the indication for delivery in the 1991 pregnancy was the extent and severity of the symptoms of pain?"* – namely, *"[t]he indication noted in the CS operation note is '[t]hree previous sections, onset of labour ?abruption of placenta"*. Mr Porter explained to me that he should have inserted *"I would add"* before giving this answer, thereby making clear that the treating obstetricians in 1991 believed that the extent and severity of Mrs Rich's symptoms of pain were important. In my judgment, his failure to make that insertion when he signed off the notes of the Joint Experts' Meeting has not been satisfactorily explained.

81. Although not specifically criticised by Mr Maskrey, I also have difficulty with Mr Porter's answers to questions 11(2), 11(3) and, in particular, 11(5).

82. I reject the remainder of Mr Maskrey's stringent criticisms of Mr Porter as advanced under paragraph 3 of his closing submissions. Whereas I would agree with Mr Maskrey that errors in Mr Porter's approach to this case have been identified, in my view these are not of the same order of magnitude as those I have specifically mentioned.

83. An expert's obligation to the Court is clear: see the decision of Creswell J in The "Ikarian Reefer" [1993] 2 Ll. Rep 68, as summarised in note 35.3.3 of Volume 1 of the White Book. In my judgment, Mr Porter has failed to give objective and independent evidence to the Court on a number of important matters. I direct that the Defendant's solicitors should send him copies of my judgment and the relevant extract from the Supreme Court Practice.

84. Mr Porter's breach of duty as an expert is not so serious as to oblige me to disregard his evidence altogether: c.f. Stevens v Gullis & Pile [1999] B.L.R. 394. However, I do take it into account in evaluating the reliability of his evidence overall.

85. The essence of Mr Porter's evidence was that it was a reasonable approach in 1993 for a clinician to proceed on the basis of a 5-20% risk of delivery within the following week, subject to there being a clinical suspicion based on some evidence. Mr Porter also said that on none of the relevant dates in May and June 1993 were there clinical signs that Mrs Rich was going to deliver within the next 1-7 days. There was always the background possibility, but nothing more than that.

86. Mr Porter was asked about the 1992 documents and sought to persuade me that a distinction fell to be drawn between "guidelines" and "guidance". For the reasons I have already given, I cannot accept that semantic finery. However, I do take Mr Porter's related point that in the early 1990's difficulties arose in defining who should be given corticosteroids, in what circumstances, and (in particular) when.

87. Mr Porter told me that the risk of scar dehiscence increases as a pregnancy develops, and that there were features of Mrs Rich's previous obstetric history which enhanced the risk. He also agreed with the proposition that if a patient presented with escalating pain which appeared to stem from the uterine scar, then at some stage there would be little option but to deliver. I would add: hence the importance on 8th June of seeking to ascertain whether Mrs Rich's pain was emanating from the uterine scar. Mr Porter did not accept that there was clear evidence of escalating pain on 25th May, but he did agree that the pain was not resolving.

88. Mr Porter agreed with Mr Maskrey that steroids should be actively considered by a clinician where there was a real or enhanced risk of pre-term delivery within 34 weeks. I set out the following sequence of questions and answers arising during Mr Maskrey's cross-examination:

> "Q. Leave these considerations aside (i.e. obstetric emergency] you should actively consider as part of your management plan (1) the fact that if she delivers without steroids you have lost the opportunity of reducing RDS by 50%, but (2) if you are using only one dose, and she does not deliver within the next 7 days, possibly 14, I have lost that opportunity?
>
> A. I agree, but there should be some indication that what you are confronting is not just a theoretical possibility.
>
> Q. Is an indication a clinical sign without which you don't act?
>
> A. That is rather too global; but in relation to the specifics of this case, the answer is yes.
>
> Q. Are you saying, that you must be confident that a woman is going into labour, or an obstetric emergency will arise leading to an immediate need for CS?
>
> A. I take issue with "confident". We don't need that. What one has to have is some evidence, some individual findings, that lead you to believe that a potential risk has now crystallised into something more real. "More likely than not" is not what obstetricians think."

89. Aside from the fact that Professor Purdie's witness statements had made clear that the test he applied is "more likely than not", and that Mr Porter should have made adverse comment upon that, I consider that in the cited passage his expert evidence reached the level of quality and specificity one might reasonably expect from a Consultant of his experience giving independent advice to the Court. In my view, assuming that I accept it, this evidence suggests that the difference between foreground and background factors, and active and non-active considerations, is somewhat artificial. When Mr Porter agreed with Mr Maskrey that corticosteroids should be actively considered, he was accepting the generality of the proposition. However, the trigger or spur to recommend these drugs, and active consideration being given to the topic, would not arise in the particular case unless evidence existed to engender a clinical suspicion.

90. Mr Porter was also asked a series of detailed questions about the clinical records in the instant case. He said that the fact that scar dehiscence or fresh placental abruption was not discovered during CS surgery in the two previous pregnancies was a factor which would render an obstetrician less likely to intervene simply on the basis of pain in the index pregnancy. I do not dispute that this might well be so in certain cases, but I do not consider that the argument can possibly succeed in the instant case. Professor Purdie did not put it forward, and the notes make clear that someone thought that Helen Rich would need a CS at 37 or 35 weeks owing to pain.

91. That aside, Mr Porter did not agree that Mrs Rich was presenting with escalating pain on 25th May and 8th June 1993. He did accept that scar dehiscence could not be ruled out, but observed that it would rarely be possible to prove a negative in these circumstances. A clinician has to make a judgment of whether the uterine scar was the cause of the underlying presentation. The diagnosis of RIF pain on 8th June meant that one could not necessarily assume that it emanated from the uterus (I infer from this that Mr Porter was accepting that this was at least a possible inference). He added:

> "The wound extends through several layers … people can have abdominal pain because of multiple surgeries and not because of the uterine scar … there are two conflicting interpretations of the presentation on 8th June. First, Mr Hare's – the pain was referable to the uterine scar, although there was no sign of dehiscence. Secondly, consider the totality of the scar, i.e. from the skin down to the uterine muscle. There was no sign of dehiscence. This was not a surprising observation."

The Rival Contentions on Breach of Duty and Factual Causation

92. Mr Havers' basic contention on the issue of breach of duty is that the Claimant has failed to identify a precise test for intervention about which there was such a degree of consensus that adherence to it was, in effect, mandatory. Mr Hare was adumbrating his own test, rather than any standard practice. The 1992 documents did not lay down any algorithm for intervention beyond stating in very general terms that the use of corticosteroids should be considered if delivery is likely within 34 weeks. In particular, the 1992 documents did not specify the level of risk and the triggers for intervention (c.f. the 1996 RCOG guidelines which were more specific). Moreover, Mr Hare has singularly failed to demonstrate that the practice at Hull was impermissible in Bolam terms. If I were unable to say what Professor Purdie's practice in fact was, owing to the elapse of time and the nature of his witness statements, I should at the very least infer that his practice was likely to have been the same as Mr Tyrrell's, i.e. the standard practice at this particular unit. This was Mr Havers' realistic fall-back submission; his primary submission was that I should rely on Professor Purdie's oral evidence.

93. Mr Havers further submitted that, regardless of Professor Purdie's thought-processes, I must – at the breach of duty and not at the factual causation stage of the analysis – consider whether maternal steroids were mandatory on 8th June, having regard to Mrs Rich's presenting signs on that occasion. I could not fairly and properly conclude that they were. Ultimately, this reduces to a question of clinical judgment which I should

be very slow to criticise in Bolam terms. Professor Purdie was an experienced, senior Consultant who examined his patient and concluded that there was no sign of uterine pain, still less of scar dehiscence. The pain on 8th June was recurrent; it was not described as severe (and Mrs Rich's witness statement states that the pain on 17th June was more acute); irrespective of whether it had moved since 25th May (and the clinical note is unclear), the pain appeared to be or remain where it had been on 18th May; there is no evidence one way or another whether Mrs Rich was still taking Tylex; and, the pain could, on Mr Porter's evidence, very plausibly be linked to the abdominal and not the uterine tissues.

94. Finally, Mr Havers stressed that the difficulty that Mr Hare was having in alighting on a specific date for intervention underscored the point that the prescription of corticosteroids was not mandatory on any one occasion. For forensic reasons Mr Maskrey wishes to focus on 8th June because his case on medical causation could not succeed in relation to any earlier date, but Mr Hare criticisms went back to 19th and 25th May. The inherent temporal elusiveness of Melissa's case is, so Mr Havers submits, telling.

95. In his closing arguments on breach of duty and factual causation, Mr Maskrey emphasised that it was incumbent on Professor Purdie on 8th June to pose to himself two sequential questions, viz. (1) is there, in this case, an increased risk of pre-term delivery before 34 weeks? and (2) if so, does the risk of the mother delivering within the next 1-7 days justify giving steroids when there must also be a chance that she does not deliver within 1-7 days?

96. Mr Maskrey submitted that Professor Purdie did not adopt that sequence of thought processes, and that I should not accept those parts of his oral evidence which suggested (cf. his witness statements) that he applied his mind at least to the first question. This is the breach of duty on which Mr Maskrey relies.

97. In my view, Mr Maskrey's written argument did not sufficiently clearly differentiate between matters which on his analysis go to the issue of breach of duty and those which were relevant to the separate issue of factual causation. In practice, these questions have a tendency towards elision, but they are conceptually distinct. Counsels' written arguments were prepared under some pressure of time, and in Mr Maskrey's oral argument the position became much clearer, and proceeded along the following lines:

    (1) the breach of duty was the failure to consider the prescription of steroids given the increased risk. This is a pure Bolam question, and Melissa's case is established on the expert evidence (provided, I would add, I reject Professor Purdie's unheralded oral elaborations of his witness statements).

    (2) the next issue which arises is that of factual causation. Here, the governing law is set out in Bolitho v City and Hackney HA [1998] AC 232. In that context, two questions arise: first, what would have happened had Professor Purdie not been in breach of duty; secondly, having answered the first question on the evidence, would that have been substandard practice [at 240G, per Lord Browne-Wilkinson]. Although I do not disagree with Mr Maskrey's formulation, the way in which I would prefer to express the Bolitho two-stage test is to ask first of all, what *would* have happened?; and then to ask, what *should* have happened?

(3) at the first stage of Bolitho, Mr Maskrey submitted that if I were satisfied on the balance of probabilities that this unit would have prescribed corticosteroids on 25th May, then I must proceed to the second stage and find that the practice at Hull was Bolam negligent.

(4) having so found, I must then ask myself the further second-stage question of what should have happened in this unit. If there is a range of clinical opinion as to what should have happened on 25th May, particularly given unit policy to prescribe only one course, I should move to 8th June and consider whether the prescription of corticosteroids was mandatory on that occasion, given Helen Rich's clinical presentation.

98. Accordingly, Mr Maskrey adroitly draws me into focusing on the events of 8th June, not 19th or 25th May. He well knows that those dates are too early for the purposes of his case on medical causation. The fact that I am appreciative of the deft and seductive manner of their presentation has not led to any judicial mindset predisposing me to be wary of succumbing to Mr Maskrey's submissions. Succumb I will, provided that they are right.

99. Bringing to the focus of scrutiny to 8th June, Mr Maskrey submitted that Helen Rich's condition had by then deteriorated; that in any event there was nothing to suppose to her pain had lessened; and, that the only fair reading of his clinical note and GP's letter on 8th June is that the pain had not moved, and that it continued to relate to the uterine scar. Furthermore, if one waited for clear clinical evidence of dehiscence, the clinician would in practice be excluding the possibility of prescribing corticosteroids because he would be confronting an obstetric emergency.

100. Finally, I should make explicit Mr Maskrey's reasons for submitting that policy at this unit in Hull constituted sub-standard practice. Insistence on the identification of a specific clinical indication leaves out of account the gestation of the foetus. As one approaches 34 weeks' gestation, the window of opportunity begins to close in any event. Accordingly, the clinician should deploy a very much lower threshold at, say, 32 weeks than at 28 weeks. There is an inherent illogicality in doing otherwise: a proper balancing of the risks entails taking the period of gestation into account. Furthermore, and this is to reiterate a point already made, insistence on the presence of a clinical indication in a case such as Mrs Rich's is tantamount to waiting until delivery becomes too urgent for corticosteroids to have any useful effect.

My Conclusions

The First Question: Professor Purdie's Practice in 1993

101. My point of departure must be to reach a final conclusion on Professor Purdie's evidence. Should I give him the benefit of the doubt, particularly given the twin facts that he has now retired and that these events occurred so long ago? Further, although Professor Purdie's evidence evolved as he was giving it, is not the simple explanation for this that the mental effort entailed in answering Mr Maskrey's searching questions accurately served to bring to mind matters and occurrences, such as the informal

Consultants' meeting, which had previously lain interred under 20+ intervening years of life's experiences and recollections?

102. Plainly, a simple answer to these questions cannot be found. I have been persuaded by Mr Havers that I should not seek to answer my questions by taking much account of Professor Purdie's tone and demeanour as he was being cross-examined. He did not enjoy the experience, but then who would. This was Professor Purdie's first time in the witness box, and I am not sure that he had prepared himself to be subjected to the depth and penetration of questioning which came from Mr Maskrey. Professor Purdie came across as somewhat "old school", slightly patrician and – if he would excuse me for saying so – a bit remote. I suspect that his instincts as a clinician tended towards the paternalistic rather than the participative.

103. My mind has wavered on this issue. Professor Purdie struck me as being an honest witness, and when his recollection permitted him to give straightforward 'yes' or 'no' answers to the questions posed in cross-examination, he gave them. Ultimately, however, I remain troubled by the frankly poor quality of Professor Purdie's witness statements, and the rather massive gulf which now exists between what he was prepared to vouch as true when he signed those statements and his current position. I am not prepared to give a professional man the very considerable latitude which Mr Havers, by implication, urges upon me. In my judgment, Professor Purdie has unwittingly permitted himself to reconstruct events in his mind and to present these to me in his oral evidence as accurate recollection, when they are not. The truth as to Professor Purdie's practice lies much closer to what he said in his second witness statement than during the course of his oral evidence.

104. I have no doubt but that there were occasions on which Professor Purdie prescribed maternal corticosteroids before May 1993, but I believe that these were few and far between. The paradigm case of prescription, as Professor Purdie expressly stated, was when there were incipient or prodromal signs of premature labour. There may have been cases in Professor Purdie's practice where the mother's presentation was such that she could be booked into the ward for an elective CS in a few days' time. However, I do not accept that the prescription of corticosteroids was even a background consideration on 8th June. Professor Purdie's overriding concern was to keep Helen Rich going as long as possible in the probable knowledge that there would come a time when delivery became, as he put it in his second statement, *"imminent"*. This adjective means, "about to happen". It does not mean, "likely to happen within 1-7 days". Although the point is somewhat academic, I cannot accept Professor Purdie's evidence that he would have prescribed corticosteroids even when delivery did become imminent; it would have been too late, as indeed was the case on 17th June when another Consultant was confronted by an obstetric emergency.

105. Should I infer, as Mr Havers strongly submitted that I should, that Professor Purdie's practice was substantially similar to his colleagues' in this unit, including in particular Mr Tyrrell's? I might have been prepared to draw such an inference if I had reached the conclusion that Professor Purdie really had no recollection of what his practice was in 1993. In his second witness statement, Professor Purdie has told me what his practice was, and I have reached the conclusion that this statement is closer to the reality than his oral evidence. Moreover, I am not convinced that this is the sort of situation where I should be drawing the sort of inference that Mr Havers urges upon me. The general practice of the unit is capable of throwing light, perhaps substantial

light, on Professor Purdie's individual practice, but it cannot be determinative; it is just one relevant factor in the evidential jigsaw. I incline to the view that different clinicians interpreted the concept of risk, and the need for clinical signs being present, in slightly different ways, with different thresholds for intervention. There was no formal protocol, and it is also clear to me that Mr Tyrrell, having worked under Professor Lilford in Leeds, had the most developed thinking within the unit on this issue.

106. In short, I find that Professor Purdie would only have considered prescribing corticosteroids if pre-term delivery was imminent.

The Second Question: what was the content of Professor Purdie's duty to Melissa on 8th June 1993?

107. If Mr Maskrey's analysis is correct (see paragraphs 95-97 above), he would say that I have already answered this second question because Professor Purdie gave no consideration to the prescription of corticosteroids on 8th June.

108. In my judgment, Mr Maskrey's analysis is incorrect; or, more precisely, it misses out an essential step. Before deciding whether Professor Purdie was in breach of duty, I must determine the *content* of the duty he owed to Melissa on that date. Was there a duty to consider the prescription of corticosteroids if the risk of pre-term delivery should have been assessed as being above Mr Hare's 10% or Professor Lilford's 5-20%, or did the duty arise only if there was in addition evidence justifying a clinical suspicion of pre-term delivery within the next 1-7 days?

109. The sequential questions posed by Mr Maskrey at paragraph 95 above seek to define the duty by reference to the test outlined by Mr Hare, but the validity of that test is the very issue I must address; and I must do so before dealing with any matters relevant to the issue of factual causation. Both Mr Tyrrell and Mr Porter made clear that the existence of a 5-20% risk of pre-term delivery within the next 1-7 days was insufficient (they were content to accept the Lilford band, although these figures cannot be regarded as fixed). What was required in addition was the existence of evidence justifying a clinical suspicion of delivery within that timeframe. Further, Mr Maskrey's sequential questions serve artificially to sever one question into two. The temporal qualification, according to Mr Maskrey, arises only at the second stage, and not at the first. Forensically, this makes it much easier for him to contend that Professor Purdie was in breach of duty in failing to consider the general, non time-specific question. In my judgment, however, there is only one question, not two, and the temporal qualification is an integral part of it.

110. I must therefore decide whether the Tyrrell/Porter view is correct; or, more precisely, whether it represents reasonable practice in Bolam terms. This, in my view, brings into consideration the Bolam reasonableness of the policy and practice of this unit.

111. I see the logical force of the argument that to insist on the advent of a specific clinical indication has the tendency to place the bar for intervention at such a level that the chance of maternal corticosteroids having any significant practical effect is close to nugatory. In other words, to require the presence of a specific clinical indication may

arguably raise the index of suspicion for delivery within 7 days way in excess of 20% and probably above 50%. I have also referred to Mr Maskrey's forceful submission that the window of opportunity begins to close as the pregnancy progresses, from which it should be deduced that the threshold for intervention must be lower at 32 or 33 weeks than at 28. To my mind, there is also force in the argument that a balanced, holistic approach seems more defensible than one which is more hidebound.

112. Notwithstanding the apparently compelling logic of Mr Maskrey's objections to the Hull policy, ultimately I have concluded that he is seeking to persuade me to apply an overly tight intellectual framework to what can only be properly expressed as a matter of clinical judgment. Here, I need to return to Mr Tyrrell's evidence, which in my judgment was very impressive. Mr Tyrrell made clear, and I accept, that a decision whether or not to administer maternal corticosteroids at any given time is complex, and involves the balancing of a number of factors in an exercise of clinical judgment. Mr Tyrrell said that he was aware of Professor Lilford's Bayesian approach, entailing the allocation of numerals to the risk, but he made clear in re-examination that in his opinion the issue was *"not just a number"*. The whole clinical picture needed to be considered, and within that context there had to be some evidence – amounting to a clinical suspicion - that delivery was likely within the next few days. As he explained at the end of his cross-examination*, "you need a clinical suspicion to give you cause to give that number* [by which he meant, 5-20%]*"*. It seems to me that Mr Tyrrell's evidence, viewed as a whole, answers the point I tentatively made under paragraph 49 above that to insist on the identification of a clinical suspicion must lift the risk significantly in excess of 20%. It does not. That point, in common with many of Mr Maskrey's logical arguments under this heading, was overly analytical.

113. It was not put to Mr Tyrrell that his view, which became the policy at this unit, was dissimilar to Professor Lilford's, nor was the latter asked about any need for a clinical suspicion. What Professor Lilford did say was that if only one dose was to be administered, then the clinician would want to be more cautious.

114. Further, Mr Tyrrell did not exclude from account the sort of considerations Mr Maskrey prayed in aid. He said this:

> "One tries to balance the benefits against the chances of the delivery not happening. This is a very difficult clinical decision, influenced by the clinical findings, the period of gestation, and other factors. I would be more inclined to give them at 28 weeks, when they might be inappropriate, then at 33 weeks, when the risk of RDS is lower."

Mr Tyrrell was not cross-examined further on this answer, in particular the last part of it. I accept that cross-examination was becoming difficult for Mr Maskrey because he was asking questions over the video-link to Queensland, Australia, and by this stage the picture had frozen. Nonetheless, the valid point that Mr Tyrrell was making was that the narrowing of the window of opportunity is paralleled, if not outstripped, by the diminution in the risk of RDS. This point was put in a slightly different way by the RCOG in its 1996 guideline (*"as pregnancy advances, the number of women that will have to be treated with corticosteroids to prevent a single case of RDS increases"*).

115. The ultimate question for me is whether the policy of this unit, as reflected in Mr Tyrrell's practice, was Bolam negligent. Mr Porter said that it was a reasonable policy, explained why, and I accept this part of his evidence. I am not to be understood as holding that Mr Hare's "test" was Bolam negligent; far from it. A solid body of reasonably competent clinical opinion would have supported it. However, I am rejecting his evidence that this was the only permissible approach. I also accept Mr Havers' submission that the Hare "test" is not derived from the 1992 documents or from Professor Lilford's evidence, and that its fluidity and imprecision gives rise to obvious difficulties in fixing the clinician with an obligation to intervene on any particular date.

116. In my judgment, the duty that Professor Purdie owed to Melissa on 8th June 1993 should be expressed in these terms: he should have considered prescribing maternal corticosteroids if he had, or ought to have had, a clinical suspicion that Mrs Rich might or would deliver within the next 1-7 days.

The Third Question: was Professor Purdie in breach of his duty to Melissa on 8th June 1993?

117. In answering my first question, I have found that Professor Purdie gave no consideration to the prescription of corticosteroids on 8th June, and would only have done so had he believed that pre-term delivery was imminent. Implicit in my finding is that Professor Purdie did not pursue the policy of his unit, and that he took a more restrictive view. However, it does not follow that because Professor Purdie's clinical practice was circumscribed, and that his reasoning process was overly narrow, that he acted in breach of duty on this occasion. In line with my conclusion on the second question, a finding of breach should only be made if Professor Purdie had, or ought to have had, a clinical suspicion that Mrs Rich would or might have delivered within the next 1-7 days.

118. Given that I have found that Professor Purdie would only have considered prescribing maternal corticosteroids if he thought that delivery was imminent, I need to exercise care as to the inferences which may be fairly drawn from his brief clinical note and slightly more expansive GP's letter. Obviously, it would certainly be safe to draw the inference that he did not believe that delivery was imminent, because had he done he would have admitted Mrs Rich. In my judgment, the essential question I need to ask myself at this stage is whether Professor Purdie ought to have had a clinical indication that Mrs Rich would or might deliver within the next 1-7 days. Posing the question in these terms does not ignore what I have found to be flawed aspects of his reasoning process, but nonetheless focuses on the content of the duty as I have specified it to be. Further, I agree with Mr Havers that it is impossible to discard considerations of clinical judgment: ultimately, Professor Purdie was exercising such a judgment (whether or not he was applying the right test) when he saw Mrs Rich on 8th June.

119. It is clear that there had been a deterioration on 19th May, but the acute pain soon settled with the administration of Tylex. It is not clear whether Mrs Rich continued to take Tylex after 20th May; and, if so, for how long. It is apparent from the clinical note dated 25th May that Mrs Rich's mobility was severely restricted, but I agree with Mr Havers that it would be unsafe to infer that she was still in acute pain. Had she been, she would have been admitted on that date. The degree and extent of Mrs Rich's pain,

or discomfort, is unclear. However, Mrs Rich did not return to the unit, or visit her GP, between 25th May and 8th June, and I cannot infer that her pain was acute or severe. Overall, I accept Mr Havers' submission that there is no reliable evidence to the effect that the pain worsened between 18th May and 8th June – save, of course, for the events of 19th May.

120. The evidence relating to the location of the pain is difficult to pinpoint. From Professor Purdie's perspective, Helen Rich was experiencing right-sided pain both on 18th May and 8th June. Whether the pain was under the right side of the scar, or to the right of the scar, remains unclear, but I am far from convinced that this matters. In my view, Professor Purdie did not suspect that the pain of which Mrs Rich had complained on 18th May related to the uterine scar, otherwise he would have acted differently. He did, though, organise a scan in order to make sure. On 8th June I agree with Mr Maskrey that Professor Purdie could not exclude scar dehiscence, but in my judgment to require him to do so for Bolam purposes represents a step too far. The real issue is whether Professor Purdie had a rational basis for his clinical suspicion that there was no scar dehiscence and/or Mrs Rich's pain did not relate to her uterus.

121. I cannot agree with Mr Maskrey that much may be gained by subjecting the terms of Professor Purdie's letter to close textual analysis. In his oral evidence, which on this issue I accept, Professor Purdie said that he was not making a presumed diagnosis of scar dehiscence. Although unhappily worded, and Professor Purdie is ordinarily a precise user of language, I have reached the conclusion that the clinical notes and GP's letter, read together, indicate that he suspected that Helen Rich's pain was referable to her old abdominal scar and to the tissues below it, excluding the uterine scar. Whatever else I might think of Professor Purdie's practice, I cannot accept that he would have done nothing had he suspected Helen Rich's uterine scar to be exhibiting signs of dehiscence.

122. In my judgment, Mr Maskrey has failed to establish that Professor Purdie should, in Bolam terms, have concluded that there was a specific clinical indication that Mrs Rich would or might deliver within the next 7 days.

123. It is convenient at this stage to address Mr Hare's contention (see paragraph 76 above) that there were a number of factors which, taken cumulatively, indicated that Mrs Rich's risk of delivery within the next 7 days was in the region of 10%. On my understanding, Mr Hare was not saying that there was any specific clinical indication which pointed to a risk of delivery within the relevant time-frame; rather, he was asking me to consider the case more broadly. In my judgment, there are two effective ripostes to Mr Hare's thinking. First, on the footing that the policy at Hull was not Bolam negligent, it seems to me that what was required was indeed the presence of a specific clinical indication. Secondly, even if a broader approach may be required, I cannot accept that Professor Purdie would have been Bolam negligent. Integral to Mr Hare's analysis is the premise that Mrs Rich's pain emanated from the tissues of her uterus and not her abdomen, and I have already acquitted Professor Purdie of negligence in that respect. Further, what Mr Hare is really saying is that Professor Purdie carried out an inadequate examination of his patient on 8th June, and that this omission was the effective cause of his omission to diagnose uterine pain on that occasion. For the avoidance of any doubt, I must roundly reject this part of Mr Hare's evidence. I have been severely critical of aspects of Professor Purdie's practice, but I cannot accept that he carried out an incompetent examination.

124. Mr Hare accepted that the 8th June materials, taken at their face value, did not betray any specific clinical indication of delivery within the next 7 days. Even if the epithet "specific" is excluded from account, and a broader approach is required, I consider that it is very difficult to say that the only reasonable judgment would or should have been to the effect that there was now a significant risk of pre-term delivery within 1-7 days. Many clinicians might have so concluded, but there must be room for a reasonable range of opinion. In truth, Mr Maskrey's second question (*"if so, does the risk of the mother delivering within the next 1-7 days justify giving steroids when there must also be a chance that she does not deliver within 1-7 days?"*) may give Melissa's case a much better chance of succeeding on the issue of breach of duty, but it is not directly to be derived from Mr Hare's final formulation.

125. For all these reasons, and even on the premise that Mr Hare's test should apply, Professor Purdie was not in breach of duty to Melissa on 8th June.

The Fourth Question: Counterfactuals

126. This finding is sufficient to defeat Melissa's claim, but in the event that this case goes further I should make additional findings on alternative premises.

127. The first issue I should address under this rubric is whether the obstetricians, clinicians and health professionals who cared for Helen Rich on 19th and 25th May 1993 acted in breach of duty in failing to consider the prescription of corticosteroid drugs on those occasions. This is relevant to the issue of medical causation, because a finding that they were in breach of duty would be fatal to her case under that head. For completeness, I should address this issue both on the basis that the practice at Hull was not Bolam negligent, and on the alternative basis that it was.

128. I make no criticism of the Defendant for failing to adduce evidence from any of those who treated Mrs Rich on 19th May, and the identity of the obstetrician who saw her on 25th May cannot be ascertained. Mr Hare believed that the individual in question was Mr Guthrie, then a Senior Registrar, but I have seen no satisfactory evidence to reach that conclusion. In these circumstances, I have no witness statement or oral evidence to evaluate. In my judgment, I have to fall back onto the contemporaneous records and the practice of this unit. I have said that there probably were divergences in individual clinical practice within the unit, but in the absence of any other evidence I am obliged to have regard to Mr Tyrrell's evidence as to that practice, which evidence was in my view was both reliable and compelling.

129. At this juncture, the analysis becomes complex and somewhat artificial. We know that corticosteroid drugs were not administered on 19th or 25th May, but we do not know why not. Contrary, I believe, to Mr Maskrey's preferred approach, at this stage I am required to ask and answer the pure Bolam question of whether the failure to prescribe maternal corticosteroids on these dates was sub-standard practice, rather than any Bolitho questions pertinent to the issue of factual causation. The odd feature of this exercise is that Mr Maskrey would no doubt prefer me to *reject* Mr Hare's view that the omissions on these dates amounted to sub-standard practice than to accept it. Maybe for that reason he did not advance his case in quite the way he did, and he was hoping that I would come to the point unaided.

130. In fact, I am able to reject Mr Hare's evidence in relation to the events of 19th and 25th May. There are no references to corticosteroids in the clinical or midwifery notes for these dates, but on this issue I accept Professor Purdie's evidence that an omission of this sort is not surprising. In my judgment, it is reasonable to infer, in the absence of other evidence or indications to the contrary, that the relevant clinicians and health professionals responsible for Helen Rich's care on 19th and 25th May sought to apply the practice of this unit, as explained to me by Mr Tyrrell. His evidence was the best evidence of that practice. Accordingly, these individuals were giving consideration to the possible need to administer corticosteroids, but concluded that there was no clinical indication that Mrs Rich might deliver within the next 1-7 days. The acute pain which led to her admission on 19th May soon settled, and although there may be arguments about whether the use of Tylex may have masked Mrs Rich's pain on 25th May it seems clear that it was not so severe that she required immediate admission. It follows that I reject Mr Hare's evidence in relation to these two dates. Assuming that the unit's practice was not Bolam negligent, the right analysis in my view is that a reasonable body of clinical opinion would have supported the decision not to use maternal corticosteroids.

131. Even on the alternative premise that those caring for Mrs Rich on 19th and 25th May ought to have adopted a broader approach, I consider that the failure to prescribe maternal corticosteroids was not sub-standard practice. Given that I have so concluded in relation to 8th June, the position must be *a fortiori* as regards these earlier dates.

132. The second issue I should address is on the premise that I have been wrong to reject Mr Maskrey's submission that Professor Purdie was in breach of duty because he failed to consider the prescription of maternal corticosteroids. On Mr Maskrey's approach, I should be addressing a range of questions under the overall rubric of factual causation (see paragraph 97 above). In my judgment, however, if that were the correct analysis, the outcome would be exactly the same. Ultimately, I would still have to address the Bolam reasonableness of the policy at this unit, and whether a clinical indication existed for delivery within the next 1-7 days. Given my findings, Melissa's case would fail on factual causation but not on breach.

133. I propose to address the issue of medical causation in as much detail as I would have done had it been critical to the eventual outcome of this case. I should make clear that I will do so on the assumed basis that maternal corticosteroids should have been prescribed on 8th June and not before.

Medical Causation

Introduction

134. The issues between the parties have narrowed as the trial progressed. Now, it seems to me that there are two key, closely related issues for my resolution. The first is whether I can be satisfied on all the available evidence to the requisite standard that, had maternal corticosteroids been administered following Professor Purdie's consultation on 8th June 1993, Melissa's RDS would have been avoided. It is common ground between the parties that if I can be, then medical causation has been established in this

case because Melissa would not have suffered her PVL. The second is whether, if I am dissatisfied about the first question, I can nonetheless be satisfied that had maternal corticosteroids been administered Melissa's RDS would have been ameliorated to such an extent that *either* her PVL would have been prevented *or* it would have been lessened to a degree that in law I must regard the failure to intervene as a material cause of her PVL. The parties are not in agreement as to the proper approach in fact and in law to this second issue.

135. The forensic contest on these two issues was between Dr Anthony Emmerson F.R.C.P., Consultant Neonatologist at St Mary's Hospital, Manchester since 1993, and Dr Janet Rennie F.R.C.P., Consultant and Senior Lecturer at UCH since 2004. Both experts are highly qualified (I have not included all of their Fellowships) and in my view both were impressive witnesses. Looking carefully at their *cv*s, it is apparent that Dr Rennie's principal research interest has been and remains in the area of neonatal brain injury, precisely the focus of the present case. In that regard, she slightly outguns Dr Emmerson in terms of depth of expertise. I also felt that she was better than he was in explaining complex medical concepts and in giving direct answers to Counsels' questions. Dr Rennie exhibited an intellectual rigour and clarity of expression which were highly impressive.

136. These general observations aside, it is clear that Dr Rennie has changed her mind about one important issue. She is not to be criticised for having done so. Yet I do have to consider whether her change of mind might undermine other parts of her evidence. Further, I have to guard against the temptation of succumbing to the fallacy that merely because Dr Rennie impressed me slightly more as an expert, it must follow that she is right.

137. Dr Rennie's change of mind relates to the operative causal mechanism of Melissa's PVL. Her original opinion, as expressed in her report and continued into the Joint Experts' Meeting, was that Melissa's RDS did not contribute to her PVL. As Dr Rennie frankly explained in answer to Mr Maskrey's questions, she had approached the case on the premise that RDS is not associated with PVL unless there is evidence of hypocarbia. It flowed from that premise that Dr Rennie believed that the cause(s) of Melissa's PVL could not be explained, but whatever it was, this was not related to her RDS. Dr Rennie told me that as time has gone on, she has come to the view that *in this particular case* there is no other likely explanation for Melissa's PVL, notwithstanding her reluctance to pinpoint any specific cause.

138. Dr Rennie now agrees with Dr Emmerson that a combination of Melissa's RDS and the need for her to be ventilated led to her periventricular or white matter injury. Other unidentified factors might still be implicated, but on my understanding of her evidence Dr Rennie now discounts these as probable causes. As she elegantly put it:

> "I have accepted that the RDS is the conduit through which the brain injury occurred here."

139. Dr Rennie accepted in her report that maternal corticosteroids would have ameliorated Melissa's RDS. However, the following series of questions and answers in Mr Maskrey's cross-examination of Dr Rennie demonstrates that there remains an important issue between the parties:

> "A. Because her RDS was severe, it is not probable that it would have been ameliorated sufficiently to prevent the associated brain injury. We have looked at the Cochrane review and the sub-analyses. Because her RDS was severe, and it was the conduit through which the brain injury occurred, that illness would have had to have been avoided to avoid her brain injury … I don't accept the premise that severity of RDS affects the cerebral blood flow.
>
> Q. If you reduce the severity of the RDS, you are likely to reduce the severity of the blood flow [in the watershed areas of the brain]?
>
> A. There is no scientific basis for that. I agree that RDS gives rise to hypoxia-ischaemia by virtue of changes in cerebral blood flow. But there is no scientific basis for the premise that the disturbances in cerebral blood flow are more severe when the RDS is more severe. This is not a justified inference, given the lack of data in any event which shows the link between RDS and PVL. Relatively mild RDS in a ventilated baby can cause blood flow changes."

140. I will need to examine this strand of evidence very carefully in the light of Dr Emmerson's evidence and Dr Rennie's concession.

The First Question: Would Maternal Corticosteroids have Avoided Melissa's RDS?

Epidemiology

141. The issue for me at this stage is whether on the balance of probabilities the administration of maternal corticosteroids following Professor Purdie's consultation on 8th June 1993 would have avoided Melissa's RDS. Mr Maskrey seeks to prove his client's case by persuading me on all the available evidence that had corticosteroids been administered the risk of RDS would probably have been more than halved. Put in epidemiological terms, the relative risk ("RR") would have been 0.49 or less.

142. The application of epidemiological concepts to legal situations is controversial, but I am able to deal with the relevant jurisprudence quite briefly. In Sienkiewicz v Grief (UK) Ltd [2011] 2 AC 229 a number of Justices of the Supreme Court, Lord Rodger in particular, doubted the accuracy and value of an epidemiological approach to legal proof, observing for example that all that statistics can achieve is proof of a probability, not proof of a fact in issue. This judgment is not the right occasion to enter into the debate, because for present purposes (albeit reserving his position in a higher court) Mr Havers concedes that if on the epidemiological evidence the true RR is 0.49 or lower, then Melissa succeeds on the issue of medical causation.

143. Accordingly, I propose to follow the approach outlined by Mackay J in XYZ v Schering Health Care Ltd [2002] EWHC 1420 (QB). In Heneghan v Manchester Dry Docks Ltd [2014] EWHC 4190 (QB) I indicated that I was more than content to

follow that approach, and I have not since changed my mind. Indeed, in my recent lecture to the Personal Injuries' Bar Association, I sought to explain the juridical basis for my opinion.

144. I have already said that the evaluation of the true RR must be made in a legal context on all the available evidence. In theory, such evidence could not necessarily exclude clinical opinion and experience, but the experts were not in agreement as to how this should be weighed against the epidemiological evidence.

145. Turning to the epidemiological evidence, Dr Emmerson and Dr Rennie are in complete agreement that the most authoritative and valuable source is the Cochrane meta-analysis, Antenatal Corticosteroids for Accelerating Foetal Lung Maturation for Women at Risk of Pre-term Birth (Review), authored by Roberts and Dalziel in 2007. In the present context of RDS, this reviewed 21 prospective, randomised studies involving 4,038 infants. The overall conclusion was that treatment with antenatal steroids was associated with an overall reduction of RDS of one-thirds: in other words, the RR was 0.66 within a 95% confidence interval of 0.59 to 0.73. If this is the correct figure, it is obvious that Melissa's case should fail on the issue of medical causation.

146. For the purposes of the Joint Experts' Meeting, Dr Emmerson and Dr Rennie were asked a series of questions seeking to ascertain whether they were in agreement as to the reduction in likelihood of Melissa developing RDS in the event that maternal corticosteroids had been delivered less than 24 hours before delivery, 1-7 days before delivery, and over the first 7-14 days. The experts were in agreement both as to the answers and their source. The experts relied on the Cochrane Review and not on clinical judgment. The answers they gave were, they thought, solely derived from Cochrane and nowhere else.

147. Unfortunately, both experts misread the Cochrane meta-analysis in two important respects. First, they said that the RR in the period 1-7 days before delivery was 0.66, when in fact that was the figure for the entire cohort, regardless of the drug to delivery interval. The experts were not asked to address the global figure, they were only asked to address sub-groups. Secondly, the experts failed to note that one of the questions was wrongly formulated. The third period was not 7-14 days (about which Cochrane is silent) but rather the more open-ended "more than 7 days".

148. On the premise that maternal corticosteroids ought to have been administered after Professor Purdie's consultation on 8th June, and not before (in line with my previous findings), I agree with Mr Maskrey that Helen Rich would have returned to hospital for treatment on 9th June. An emergency situation had not arisen, and it is reasonable to infer that child-care arrangements would have had to have been made. This leaves a drug to delivery interval of 8 days.

149. One of Mr Havers' headline submissions was that an agreed position had been reached at the Joint Experts' Meeting, and that I should not depart from it. Alternatively, he submits that Dr Emmerson's late attempt to undertake a different analysis of the Cochrane data should not be countenanced because no good reason has been advanced for his *volte-face*.

150. I cannot accept Mr Havers' submission in either of its formulations. The experts were answering the specific questions put to them, as it happens the questions framed by the Claimant's legal team. In two important respects, the answers they gave were incorrect. The experts were not asked to address the global figure for all 4,038 babies, and the reference to 0.66 is incorrect (that figure may be derived from "outcome or subgroup title 7" on page 48 of the Cochrane review). In any event, the parties are not bound by any agreement reached at a Joint Experts' Meeting, and Mr Maskrey is quite entitled to draw my attention to the Cochrane meta-analysis as a whole.

151. Adopting that approach, I do not intend to refer to each and every Cochrane finding, merely to those relied on by Counsel in their closing arguments. I need to preface my summary of the findings with three important observations. First, the Cochrane review only considered dichotomous outcomes, i.e. RDS against no RDS. The review does not directly address continuous outcomes, namely the issue of whether maternal corticosteroids reduce the severity of RDS. Of course, at this stage of my judgment I am addressing Mr Maskrey's primary case that such an intervention would have prevented Melissa's RDS altogether. Secondly, what in my judgment Mr Maskrey is seeking to do is to have regard to what the Cochrane authors call "subgroup analysis". There is an important issue between the parties as to the value to be accorded to such an approach. Thirdly, at this juncture I am merely setting out the data, not the inferences, if any, to be drawn from them.

152. Looking at gestational age at delivery, the RRs are 0.67 for babies born before 30 weeks, 0.56 before 32 weeks, and 0.58 before 34 weeks (the review also deals with less than 28 weeks and before 36 weeks). The before 32 week figure includes the before 30 week figure, and so on. All these findings are statistically significant.

153. Looking at entry to delivery interval (this means, entry to the relevant study, but I take it to be the same as drug to delivery interval), the RRs are 0.63 for less than 48 hours and 0.46 for between 1 and 7 days after the first dose. These are statistically significant findings. The RRs at less than 24 hours (0.87) and more than 7 days (0.82) are not statistically significant.

154. Looking at type of corticosteroid, betamethasone treatment resulted in a greater reduction in the incidence of RDS than dexamethasone (RRs of 0.56 versus 0.80).

155. Looking at the decade of recruitment to the study, the RRs are 0.55 for the 1970s, 0.71 for the 1980s and 0.69 for the 1990s.

156. Looking at one specific outcome, namely "moderate/severe RDS" (see group 8), the RR for all 1,686 babies in respect of whom that outcome may be derived from the relevant studies is 0.55. For babies born 1-7 days after the first dose, the RR is 0.37 (based on one study only). For babies born more than 7 days after the first dose, the RR is 1.83 (one study only, and the result not statistically significant).

157. Looking at the need for post-natal ventilation, the RR is 0.7 although, as the experts have pointed out, the numbers available for analysis are small.

158. The Cochrane reviewers conclude that their findings support the continued use of maternal corticosteroids. For clinical purposes, the findings show substantial benefit,

and it goes without saying that doctors and researchers have no interest in any legal standard of proof, which is an inflexible and artificial construct.

159. In the accompanying narrative, the Cochrane reviewers place a significant "health warning" against the betamethasone versus dexamethasone finding, and in those circumstances I can attach only minimal weight to it. I can also attach minimal weight to the findings relating to "decade of recruitment to study". Melissa's is an actual case, not a piece of research.

160. The following two passages in the narrative merit comment:

> "Reduction in RDS is seen in infants born up to 7 days after the first dose. This review has not shown any benefit in primary outcomes for infants delivered greater than 7 days after treatment with antenatal corticosteroids. In fact, birthweight is reduced in this subgroup. This lack of benefit is not a new finding, and in the past has lead [sic] to the practice of repeating courses of antenatal corticosteroid weekly if women remain undelivered."

and

> "We have included the results of the subgroup analysis in this update because we recognise that clinicians will want to see this information for its practical implications, and also because it has been the subject of much conjecture following the first review. Caution, must however, be expressed in the interpretation of the subgroup analyses conducted in this review. There is the possibility of Type 1 error due to the number of analyses conducted [i.e. because the numbers involved become quite small, the risk of wrongly concluding that the null hypothesis has been falsified increases]. Furthermore, the subgroups of gestational age at delivery, length of premature rupture of the membrane and entry to delivery interval, involve post-randomisation variables. Conducting subgroup analysis based on post-randomisation variables is liable to considerable bias as the variable on which the subgroup is based may be affected by the intervention that occurs at randomisation. The clinician should therefore not draw too many conclusions from the results of the sub-group analyses."

161. As for the first of these passages, Dr Emmerson contended that no statistical proof of benefit is not synonymous with proof of no benefit, and as a general proposition I would agree. The authors of the Cochrane handbook, not specifically referred to me but available online, make that very point. On my understanding of his evidence, Dr Emmerson makes two points germane to this particular case. His first point (see paragraph 89 of his report) was that a close analysis of the Cochrane data showed that most infants in the 7+ day sub-group did not get RDS, which explains why the findings did not reach statistical significance. He added that it was highly likely that most of the mothers in this sub-group did not in fact deliver for many weeks. His

second point was that there is no pharmacological or physiological reason why the benefit of corticosteroids should not endure beyond the seventh day.

162. In my judgment, his second point has vastly more force than the first. It is entirely speculative to opine that the mothers in the 7+ day sub-group probably did not deliver for many weeks. We simply do not know. Further, although the numbers involved may have been too small to enable statistical significance to be achieved, this is not a factor which may avail Melissa's case. It is, at best, neutral, but the fact remains that a RR of 0.82 is unhelpful to her. Dr Emmerson's argument would have greater force if the RR were below 0.5 and not statistically significant. As for the second point, it was common ground between the experts that the beneficial effect would not suddenly cease at the end of the seventh day. In my judgment, it would continue, but at what strength or value is unclear. The only reasonable inference is that the RR on day 8 would be lower, perhaps only slightly lower, than the RR between days 1-7.

163. As for the second of the observations made by the Cochrane reviewers in the narrative section of their meta-analysis, there was clear disagreement between Dr Emmerson and Dr Rennie about the value, if any, to be placed on sub-analysis. Dr Rennie was astute to underscore the inherent limitations: the *post hoc* nature of the methodology; the fact that the studies were not designed to answer the specific questions germane to the sub-group carve-outs; the risk of Type 1 statistical errors. Dr Rennie also added that neither herself nor Dr Emmerson had the statistical expertise to synthesise the various sub-group analyses to the extent necessary to attain a valid RR figure for the notional sub-group into which Melissa falls. Dr Emmerson, on the other hand, opined that the exercise had utility because it enables the Court to attain greater specificity (by which I think he means sensitivity) as to the data applicable to Melissa's particular case. Although he embraced the point very late in the day, Dr Emmerson placed particular reliance on the group 8 data (see paragraph 156 above) which yield a headline figure for RR of 0.55. He sought to persuade me that group 8 was not a "sub-group" at all because it was based on an outcome measure which did not require any analysis: the relevant data were patently derivable from the six studies involved.

164. During the course of his oral evidence, Dr Emmerson referred me to three papers, namely Antenatal Glucocorticoid Treatment and Cystic PVL in Very Premature Infants by Baud et al [*N Engl J Med 1999*; 341: 1190-6]; Antenatal Steroids are Associated with a Reduction in the Incidence of Cerebral White Matter Lesions in Very Low Birthweight Infants by Agarwal et al [*Arch Dis Child Fetal Neonatal Ed 2002* 86: F96-F101]; and Antenatal Steroids and Neonatal PVL by Canterino et al [*Obstet Gynecol 2001*; 97: 135-9]. Dr Emmerson had referred only to the Baud et al paper in his written evidence. Counsel spent some time analysing these papers but in my view they are of little or no assistance. These studies were not designed to examine RDS, and none of them was of sufficient quality to meet the Cochrane inclusionary criteria, even had they been relevant. I note that, insofar as any data for RDS may be extracted from the Baud et al paper, the benefits of corticosteroids appeared to be rather modest – much less impressive than the Cochrane figures derived from higher quality studies.

165. Mr Maskrey's final submissions to me proceeded along the following pathway. First, he submitted that the correct group for Melissa is group 8 in the Cochrane review with a RR of 0.55. Dr Rennie advanced no compelling reason for removing Melissa from that cohort and placing her in the much wider cohort of "all babies", with a RR of 0.66. Secondly, he submitted that the statistical trends derivable from other salient sub-groups brought Melissa's RR to a point below 0.55 albeit not, on the balance of probabilities, to 0.49. Thirdly, he submitted that if the drug to delivery interval is 8 days (as I have inferred it would have been), I should allow a combination of the statistical evidence and clinical judgment to bring this case to the magic figure of 0.49 or below. 8 days is only just outside 7 days, and the maximal effect is seen in the 1-7 day sub-group. The RRs for the 1-7 day sub-group are 0.46 for "all babies" and 0.37 for moderate/severe RDS, into which latter category Melissa falls. Clinical judgment, coupled with basic pharmacology and physiology, clearly indicates that the effect would not have ceased on day 8. It therefore follows that I can properly find on the balance of probabilities that had a study been conducted with babies sharing as many of Melissa's characteristics as possible, the probable result of such a study would have been a RR of less than 0.5 with probable statistical significance. Finally, Mr Maskrey strongly submitted that I should be very wary of any outcome which effectively meant that the Defendant's breach of duty (I must assume for present purposes that Melissa has won the first issue) is "empty", in the sense that it cannot sound in damages because the epidemiological evidence is deficient.

Analysis and Conclusions

166. It is unnecessary for me to set out Mr Havers' submissions on this issue. Unsurprisingly, he urged me to adopt what he submitted was a conventional approach, and to conclude that Melissa's case had not been proved on the balance of probabilities.

167. It seems to me that the first issue I have to decide is the true RR figure for Melissa on the basis of the Cochrane review data.

168. Contrary to Mr Maskrey's suggested approach, my starting-point is the 0.66 figure for the entire group of 4,038 babies. These are the babies within the 21 relevant studies, in respect of whose mothers some received corticosteroids and others (the control group) did not. Plainly, not all the cases and controls developed RDS.

169. I must then move on to consider the extent to which it is legitimate to adjust that headline figure to reflect sub-group analysis. In my judgment, and without prejudice to the validity of this approach, there are three sub-groups which are potentially relevant. First, the moderate/severe RDS sub-group where the RR figure is 0.55. I should add that within that sub-group there are two sub-sub-groups in respect of which the evidence is by no means robust, because it derives from a singleton study. In babies born within 1-7 days of the first dose, the RR figure was 0.37; for babies born more than 7 days after the first dose, the comparable figure is 1.83, but without statistical significance. Secondly, there is the gestational age of delivery sub-group – Melissa was born at 32[1] weeks' gestation, but this figure is not precise and in my view it is right to take the RR figure for the less than 32 week sub-group, which is 0.56. It does not in fact make much difference if Melissa were deemed to fall within the less

than 34 week sub-group. Thirdly, there is the drug to delivery interval sub-group where the relevant RR values are 0.46 for 1-7 days and 0.82 for 7+ days (albeit without statistical significance). I propose to ignore all the other sub-groups because they are, at best, of marginal relevance.

170. Is it appropriate as a matter of principle for me to hone or adjust the initial figure for RR of 0.66 to reflect sub-group analysis, or is this a judicial step too far?

171. In XYZ v Schering Health Care Ltd Mackay J examined all the available epidemiological evidence, including evidence bearing on the remaining eight Bradford-Hill factors, before concluding that the RR was 1.7 (the claims failed because this was a doubling of the risk case). At paragraphs 41-43 of his judgment, Mackay J observed that epidemiology was the starting-point for his assessment of what he called the "true" relative risk but not the end-point. There came a time, he said, when he would be constrained, or permitted, to part company with the epidemiology and come to an overall, broader assessment of the issue applying traditional common-law standards.

172. In that case, Mr Justice Mackay received evidence from epidemiologists and statisticians to enable him to ascertain his starting-point figure. He could not reach his end-point without first analysing the epidemiology. I have received no evidence from experts in these disciplines, and without such evidence I would be drawing inferential conclusions on the basis of speculation, guesswork and personal impression. In my judgment, this is not an exercise which I may properly undertake, nor is it akin to the analysis that Mackay J undertook.

173. Whereas I do not accept Dr Emmerson's point that group 8 does not represent a sub-group (it is located in a series of "groups" which, on analysis, are clearly sub-groups, because they are described as such by Cochrane and are all carve-outs from wider studies), I see the force of the contention that the ascertainment of outcome does not run the risk of bias from the application of post-randomisation variables. The problem here is Type 1 error. Mr Maskrey submitted that the numbers participating in the relevant studies were large, but I do not have the expertise either to agree to disagree with him: I simply do not know.

174. In any event, there is no sub-group into which Melissa obviously fits. Mr Maskrey submitted that group 8 was clearly applicable, but I remain doubtful. Group 8 covers "moderate/severe" cases. This might be a combination of two groups, being separately "moderate" and "severe", or it might be a composite group where it was not possible to differentiate further. We do not know. The experts do not agree about the severity of Melissa's RDS. Dr Emmerson's opinion expressed in cross-examination was that Melissa's RDS was *"moderate to severe, not the most severe"*. Plainly, this answer is open to interpretation, and no follow-up questions were put. Given that Melissa was ventilated for 5 days and had to be paralysed, I prefer Dr Rennie's evidence that her RDS was severe. In such circumstances, Melissa may or may not fall within group 8; it is not possible to say, without knowing more about the parameters for inclusion. A comparison between Cochrane groups 7 and 8 reveals that corticosteroids appear to be more effective in cases of moderate/severe RDS than in cases of mild RDS (an examination of the data shows, contrary to Dr Rennie's initial view, that all cases which are not "moderate/severe" are by definition mild RDS). It might be argued that the inclusion of the moderate cases works to Melissa's

disadvantage because they serve, if anything, to increase the RR figure rather than reduce it. However, that is no better than an informed guess, and the data permitting the appropriate analysis are not available. The expertise of an epidemiologist might have assisted, but there is none.

175. If I were to stick out my judicial neck, the farthest extent to which I would be prepared to go in Melissa's favour would be to say that the RR for her is 0.55. I should emphasise that this is not my preferred approach, because it is not evidence-based, but it indicates the outer limit of what might be appropriate.

176. The issue becomes starker when consideration is given to other competing variables. Within the RR 0.55 figure lie the 1-7 day babies (RR 0.37) and the post 7 day babies (RR 1.83, without statistical significance). How should I be adjusting the 0.55 figure to reflect this additional information? If drug to delivery interval is viewed not within the context of the moderate/severe RDS sub-group but more generally, the competing figures for RR are 0.46 and 0.82 (without statistical significance). How should I be adjusting the 0.66 figure to reflect this additional information, and then fuse the adjusted figure with my adjusted 0.55 figure? In my judgment, it is impossible for a court to undertake this exercise on the information currently available. At the very least, an epidemiologist would need to explain how the smaller studies should be weighted for these purposes (given the Cochrane concerns about sub-group analyses), and how, if at all, the studies which have failed to attain statistical significance should be evaluated.

177. I invited Mr Maskrey to draw to my attention an example of a decided case where the Court has performed the exercise he urges on me in this case. He could not find a previous instance of a Court being prepared, without expert evidence, to adjust or hone RR values from X to Y on this sort of quasi-Bayesian basis. Instead, Mr Maskrey drew my attention to a case where the court was prepared to amalgamate epidemiological evidence with clinical judgment/experience in order to avail a Claimant. Although the case was not precisely in point, I need to examine it carefully for the purposes of Mr Maskrey's submission.

178. In Carter v Basildon & Thurrock University Hospitals NHS Foundation Trust [2007] EWHC 1882 (QB) (Mr David Foskett Q.C. sitting as a DHCJ) a Cochrane Collaboration paper showed that the relevant pooled RR was 0.33 with a 95% confidence interval of 0.08 to 1.21. Although, therefore, the RR figure appeared to be very propitious from the Claimant's perspective, the 'p' value was more than 0.05 (because the upper value exceeded 1) and the result was not statistically significant. Further, the confidence interval was extremely wide.

179. At paragraph 95 of his judgment, Mr Foskett Q.C., as he then was, said this:

> "The authors of the paper do comment that although "the estimated pooled risk reductions did not reach statistical significance, patients and doctors may be reluctant to embark on a new trial that involves a placebo group." Translating that observation into the realities of scientific life means that it is very unlikely that there will ever be a research series carried out prospectively that will reveal to the 95% confidence limit whether Heparin does alter the outcome in these cases. As I

> observed during the hearing, given the perceived benefit of Heparin administration, it would almost certainly be regarded as unethical to develop a trial that involved some patients receiving Heparin and others not receiving it for the purposes of trying to see whether death or dependency is prevented by the administration of Heparin. The consequence of this is that if a court felt itself bound to act on evidence that demanded statistical cogency to a 95% confidence limit, no claimant negligently deprived of Heparin could ever succeed at the causation stage."

180. In reaching his inferential conclusion that the deceased would probably have survived if Heparin had been administered, Mr Foskett Q.C. took into account the fact that the law imposes a lower standard of proof than epidemiology, and that on the particular features of the case before him there were good clinical reasons to suppose that the deceased would have been a beneficiary.

181. I do not agree with Mr Foskett Q.C. that the unavailability of epidemiological evidence is, without more, a factor which a claimant may pray in aid – assuming that paragraph 95 of his judgment should be read in that manner. If epidemiological evidence reaching statistical evidence is required in a particular case to satisfy the legal burden of proof, then the fact that it does not exist is, to my mind, irrelevant - given that it has not been suggested that the Fairchild exception applies. Judges must do the best they can with the available evidence, and if – for whatever reason – it is lacking, there is nothing that can be done about it *in this context*. The law softens the harshness of this position in a different context: see paragraphs 189ff below.

182. In my judgment, whether the legal standard of proof is lower than that implied by a confidence interval of 95% raises a separate question. A confidence interval of 95% does not approximate to proof of a fact in issue to a 95% level of probability. As I said extra-judicially, given that the 'p' value is calculated on the assumption that the null hypothesis is true, it cannot be a direct measure of the probability that it is false. There is, in fact and in principle, no ready, mathematical means of ascertaining that probability.

183. On the facts of the case before him, Mr Foskett Q.C. was faced by epidemiological evidence which in terms of the figure for the RR was very helpful to the Claimant but was not robust, because it lacked statistical significance, and by clinical evidence which quite strongly indicated that the deceased would have fallen within the favourable group. The inference which Mr Foskett Q.C. felt able to draw, on all the available evidence, was that the correct RR figure for the deceased was lower than 0.50. He did not say that the 0.33 figure could be supported on a balance of probabilities approach, nor did he say what any honed or adjusted figure might be in the light of the clinical evidence which he accepted.

184. I have my doubts whether this decision was rightly decided, but I understand that it was not appealed. If it was correctly decided, the principle it exemplifies is that clinical judgment/experience relating to the specific features of an individual case may be used to fortify epidemiological evidence which tends to support the claimant's case. The case is not authority for the proposition that the Court may adjust RR figures to reflect its understanding of the epidemiological evidence, read as a whole,

or that clinical judgment/experience may avail a claimant where the RR figure is 0.50 or above.

185. Even if clinical judgment/experience is capable of being relied on, it does not help me in the circumstances of the instant case. It was not Mr Maskrey's contention that any specific clinical features of Melissa's case work in her favour (c.f. the approach in Carter); his argument is that she benefits from an epidemiological approach based on sub-group analysis. Dr Emmerson gave some quite general anecdotal evidence of how in his experience the number of babies with severe RDS requiring ventilation has diminished significantly over the years of his practice. He also told me that the cases he now sees tend to be much less severe, but this to my mind relates only to Mr Maskrey's amelioration argument. It was Dr Emmerson's clinical judgment, as I understood his evidence, that had maternal corticosteroids been administered Melissa would not on the balance of probabilities have developed RDS and/or would not have developed PVL. In my judgment, evidence of this nature is not capable of being quantitative; it is no more than anecdotal and impressionistic. If, on the available epidemiological evidence, I cannot give a proper adjusted figure for RR before having regard to clinical judgment/experience, it must follow that Dr Emmerson's qualitative, as opposed to quantitative, evidence takes my inquiry no further.

186. Mr Maskrey made the less ambitious submission that clinical judgment is valuable to the extent that it supports the conclusion that there is no pharmacological or physiological reason for the beneficial effect suddenly dissipating at the onset of day 8. Dr Rennie agreed with that, and so do I. However, this does not advance his argument sufficiently far. I am still confronted by the difficulty that I cannot quantify all the competing variables, including this one. I cannot properly say what the adjusted RR figure should be on day 8.

187. It does not matter for present purposes whether one takes a RR figure of 0.66 or 0.55, or whether other sub-groups show a tendency towards benefit. The bottom-line is that I cannot quantify that benefit, I cannot reach a proper conclusion as to the RR on day 8, I cannot draw any quantitative assistance from clinical judgment/experience, and I cannot properly evaluate all the available evidence to reach a sound probabilistic conclusion.

188. My overall conclusion on this limb of Melissa's case is that she cannot prove to the requisite standard that the RR of maternal corticosteroids avoiding her RDS was lower than 0.5.

The Second Question: Would Maternal Corticosteroids have Ameliorated Melissa's RDS to the extent that she would not have developed PVL, alternatively did the Failure to Administer Maternal Corticosteroids Materially Contribute towards Melissa's RDS such that the Defendant is liable for all her Loss?

189. As has already been pointed out, the Cochrane review examined dichotomous, not continuous, outcomes, and amelioration in RDS was excluded from account. However, unsurprisingly Dr Rennie agrees that maternal corticosteroids would have materially ameliorated Melissa's RDS. What she cannot say, nor can Dr Emmerson, is

to what extent. In any event, the logic of my conclusion on the first of the medical causation issues is that the extent cannot properly be ascertained.

190. It is not Mr Maskrey's case that I should undertake an apportionment exercise; that would not be possible. He submits that it is sufficient for his purposes that I should find that the failure to administer maternal corticosteroids made a material contribution to the "overall package" of Melissa's RDS. He further submits that *"the duration and severity of the RDS made a material contribution to the PVL and causation is established"* (see paragraph 35(ix) of his closing argument).

191. In answer to Mr Maskrey's submission, Mr Havers invited me to accept Dr Rennie's evidence that Melissa would have suffered her PVL even had maternal corticosteroids being administered. As he put the matter in his closing argument, *"Dr Rennie's expert opinion is that because the Claimant developed severe RDS amelioration would have had to reduce her RDS to virtually zero in order to prevent brain injury"* (see paragraph 38). Mr Havers also submitted, on my understanding, that the present case is not a cumulative damage case which may benefit from a modified "but for" test.

192. I have encapsulated Dr Rennie's evidence at paragraph 139 above. Dr Emmerson's evidence has always been that PVL results from instability and reduction in brain perfusion in the watershed areas. Small changes in cerebral perfusion cause periventricular changes due to ischaemia. Although there is no research evidence to this effect, an amelioration of RDS can be expected to result in less blood flow fluctuation.

193. I have to decide which body of expert evidence I should prefer. Ordinarily, I would reach my conclusion on the basis of factors such as the relative expertise of the experts in the particular topic, and the clarity and force of their explanations (my list is not exhaustive). If those axioms were applied to the instant case, I would prefer Dr Rennie's evidence to Dr Emmerson's. However, re-examining Dr Rennie's reasoning, it is clear to me that an important part of it comes too close to resurrecting a plank of argument which I thought that she had abandoned in conceding causation. In particular:

> "But there is no scientific basis for the premise that the disturbances in cerebral blood flow are more severe when the RDS is more severe. *This is not a justified inference, given the lack of data in any event which shows the link between RDS and PVL.*" [my emphasis]

194. This is not the sort of situation where epidemiological evidence is required; in any event, it is not available, nor is there any research evidence which may prove Melissa's case. Both experts agree that the issue should be resolved on the basis of scientific inferences from the available material, including what is known about neonatal neurophysiology. Dr Rennie's concession that RDS caused Melissa's PVL is founded on such an inference. I prefer Dr Emmerson's evidence on this issue. In my judgment, the link between corticosteroids lessening the severity of RDS thereby lessening the blood flow fluctuations is, to my mind, sufficiently well founded on his evidence to be acceptable.

195. The nexus between lesser blood flow fluctuations and either no or less severe PVL still needs to be examined. Mr Maskrey submitted that whether or not Melissa was ventilated she could be expected to have had better cerebral blood flow and therefore to have escaped repeated ischaemic insults. I have reviewed Dr Emmerson's written evidence, both in his report and the note of the Joint Meeting, and his oral evidence, and nowhere does he say in terms that less severe RDS would, via the mechanism of lesser blood flow fluctuations, have led to less severe PVL. Should I draw that inference? In my judgment, I should. Dr Rennie's argument was that there was no scientific link between severity of RDS and the severity of blood flow fluctuations. I have rejected that argument. She did not raise the separate argument that there is no link between severity of blood flow fluctuations and severity of PVL, and in my judgment she would have done if it were a good point. On my understanding of the operative neuro-physiological mechanisms, if there is less ischaemia there is less damage to the white matter in the brain.

196. I do not claim that the point is an easy one, and it perhaps merited greater exploration with the experts, particularly in cross-examination. I have to do the best I can on the available evidence, against the backdrop of Dr Rennie's concession. Accordingly, I must proceed on the basis that the failure to administer maternal corticosteroids materially contributed to the severity of Melissa's RDS which, in turn, materially contributed to the severity of Melissa's PVL. Her PVL was in fact caused by her RDS, which was severe, but had her RDS been less severe, then her PVL would, equally, have been less severe. However, the extent of the diminution in severity of both Melissa's RDS and PVL cannot be quantified on any existing medical and scientific evidence.

197. Next, the question arises of whether, as a matter of law, this finding is sufficient for Melissa's purposes.

198. Mr Maskrey relied on the decision of the Court of Appeal in Bailey v MoD [2009] 1 W.L.R. 1052. The facts of that case are quite complicated, and the critical part of Waller LJ's judgment is brief. It has been subject to heavy criticism by Professor Jane Stapleton (see Unnecessary Causes, reprinted from the L.Q.R., January 2013), but I have to extract the proposition for which it is authority.

199. In Bailey, the Claimant/Respondent suffered brain damage when she aspirated her vomit leading to a cardiac arrest. She aspirated her vomit because she had been severely weakened by an operative procedure, and a consequent negligent failure to resuscitate her and carry out appropriate interventions. Had these negligent omissions not occurred, she would have developed serious conditions in any event (pancreatitis and renal failure), but they would have been less severe and she would have been in a much fitter state to combat them. The event which led to her brain damage, the aspiration of vomit, was the consequence of her severely weakened condition, but it was not possible to say that "but for" the negligence this event would not have occurred. However, on the available evidence, it was possible to say that the negligent and non-negligent causes materially contributed to the overall weakness.

200. At first instance, Foskett J ([2007] EWHC 2913 (QB)) held that there were two components to the Claimant's weakness: for conceptual purposes, these could be arranged under the headings of "negligent" and "non-negligent". Each of these was material, and together they combined to create the mechanism which led to the

cardiac arrest and brain injury. Given that Hotson was inapplicable because it could not be established on the balance of probabilities that the cardiac arrest would not have occurred without the negligent causes, it was sufficient for the Claimant's purposes that the negligence had contributed to the indivisible outcome. Foskett J made no mention of the "but for" test needing to be modified. Professor Stapleton welcomed this approach.

201. The Court of Appeal adopted a slightly different approach and held that the "but for" test is modified in these circumstances. This was not a case where there were other competing, candidate causes (c.f. Wilsher v Essex AHA [1988] 1 AC 1074). In Waller L.J.'s view, the instant case was governed by Bonnington Castings v Wardlaw [1956] AC 613 and (possibly) McGhee v NCB [1973] 1 WLR 1. There were not different, distinct causes in play (each operating in a different way), but cumulative causes which operated in the same way to bring about the Claimant's weakness. Thus, the instant case was precisely analogous to the industrial disease cases of "innocent" and "guilty" dust. In Waller L.J.'s view:

> "… one cannot draw a distinction between medical negligence cases and others. I would summarise the position in relation to cumulative cause cases as follows … In a case where medical science cannot establish the probability that 'but for' an act of negligence the injury would not have happened but can establish that the contribution of the negligent cause was more than negligible, the 'but for' test is modified, and the claimant will succeed." [paragraph 46]

202. This passage in particular has attracted Professor Stapleton's excoriating criticisms. Her contention is that it is unnecessary to require any modification of the "but for" test at all; it is sufficient in this type of case for the Claimant to prove that the Defendant's negligence contributed to the mechanism which led to her indivisible injury. This is the principle which may be derived from logic and jurisprudence.

203. I agree with Professor Stapleton that the characterisation of the negligent mechanism of injury in Bailey as being one of several cumulative causes is uncomfortable, given that the analogy of silica dust (with each grain of dust causing a separate physical insult) appears incomplete. The "cumulative cause" analysis works much better in a case of divisible injury.

204. The Courts have had difficulty in differentiating between divisible and indivisible injuries in less straightforward cases: see the valuable analysis of Swift J in Jones v Secretary of State for Energy and Climate Change [2012] EWHC 2936 (QB) (at paragraph 6.49), and Smith L.J.'s differing approaches in Dickens v O2 Plc [2008] EWCA Civ 1144 and B v MoD [2010] EWCA Civ 1317. The difficulties arise because one case of indivisible injury (e.g. cancer) can be less severe than another.

205. If the injuries in Bailey are seen as her cardiac arrest and then her brain damage (the first led inexorably to the second), each (or both in combination) should properly be characterised as indivisible. This is Swift J's preferred analysis, and it is mine too. However, Waller L.J. appears to have analysed the matter slightly differently, preferring to focus on Ms Bailey's weakened state. In that regard, the "cumulative cause" approach is still not free from difficulty, as Professor Stapleton has pointed

out. The "weakened state" is not the relevant injury, and there is an artificiality in postulating that its causes were cumulative. It would be much neater, and more attractive, to say that the Defendant's tort contributed to the mechanism which led to Ms Bailey's indivisible injury or injuries. However, neatness and attractiveness are not necessarily the relevant touchstones.

206. In the instant case, Melissa's RDS and PVL were indivisible injuries. I have already found that they would have been less severe had her mother been prescribed corticosteroids on 8th June for administration on 9th June. Professor Stapleton would, therefore, urge me to find in her favour on the straightforward basis that the Defendant's negligence made a material contribution to the mechanism which led to her brain injury.

207. I propose to decline Professor Stapleton's invitation, because I consider that I am constrained by authority to follow a different approach. Implicit in Professor Stapleton's reasoning is the contention that the House of Lords erred in Hotson v East Berks AHA [1987] A.C. 750. That was a case where the causative potency of the two potentially relevant matters was quantifiable, and the non-negligent component exceeded 50%. Professor Stapleton's objection is that the ability to quantify causative potency should not make a difference to the applicable legal test. But whether or not I agree with her analysis, I am operating in different conditions and under different constraints.

208. Yet although my approach differs, the outcome is the same.

209. I can see that the instant case is less clear than Bailey to the extent that there it was possible to identify temporally sequential acts and omissions which combined to create the composite condition – her weakened state – which led to the catastrophic injury. It made complete sense for Foskett J to describe the operative causes as negligent and non-negligent "components". In the instant case, that term feels less apt, because the operative causes worked simultaneously and in an identical manner to bring about a composite outcome, viz. severe RDS. Its negligent and non-negligent aspects are "components" only in the sense that they may be valid legal attributions.

210. However, I have concluded that this does not alter the analysis. What matters for present purposes is the conceptual attribution and the identification of a condition which would have been less severe had the postulated omission not occurred. The fact that these causes operated simultaneously, not sequentially, is an evidential, not a legal, feature. What also matters for present purposes is that we have a continuous pathological process, involving the destruction of cells, and not a situation where the Defendant's tort merely contributes to the risk of injury (see my analysis in Heneghan). Finally, what matters for present purposes is that medical science does not permit one to say what would have happened, on the balance of probabilities, but for the Defendant's tort. This is the crucial point of distinction with Hotson.

211. In these circumstances, I conclude that the ratio of the decision of the Court of Appeal in Bailey applies, and that I must find that the Defendant's tort materially contributed to Melissa's damage such that she recovers in full (or, would have recovered in full had I found in her favour on the breach of duty issue).

212. There are, I accept, issues which may be worthy of further consideration, but in my view that should occur at appellate level. These include: (i) whether some wider principle should now be applied to these cases (per Professor Stapleton), (ii) whether a material contribution analysis is truly apt to this sort of case, (iii) whether the McGhee extension to Bonnington Castings embodies the true governing principle, in which circumstances the present case may find itself within the hornet's nest Mr Maskrey analogises and is rather keen to avoid; and, (iv) whether the time has come to clear out the hornets altogether. This list is not exhaustive. Again, in Heneghan I discussed a number of the possible difficulties, and the extremely fine, almost casuistical, distinctions which fall to be drawn in this domain.

Conclusion

213. I have found that Professor Purdie did not act in breach of duty to Melissa in failing to consider the prescription of maternal corticosteroids on 8th June 1993. Although there were flaws in his practice and the reasoning which supported it, he was entitled to conclude that there was no clinical indication that Mrs Rich would deliver within the following 1-7 days.

214. On the premise that maternal corticosteroids should have been administered on 9th June 1993, I have found that the breach materially contributed to Melissa's RDS and to her PVL, and that her claim would have succeeded in full. I have rejected Melissa's case that her RDS, and therefore her PVL, would on the balance of probabilities have been avoided altogether.

215. I understand that both parties are reserving their respective positions on aspects of the law applicable to this case. As for Mr Havers, he reserves his position as to the use to which epidemiological evidence may be put, and on the decision of the Court of Appeal in Bailey. As for Mr Maskrey, he reserves his position as to whether or not proof that the defendant's negligence made a material contribution to the mechanism which led to Melissa's brain injury suffices for the purposes of proof of causation, and thus as to whether Hotson v East Berks AHA was correctly decided.

216. These cases cannot be decided on the basis of judicial sympathy for Melissa and her mother. The latter attended the trial and acted with great dignity throughout.

217. I record my gratitude for the clarity, economy and insightfulness of Counsels' submissions. As I indicated at a very late stage in these proceedings, I found the case to be finely balanced. In the end, I have come to firm conclusions, but the route to them has been windy and challenging.

218. There must be Judgment for the Defendant.