Neutral citation number: [2024] EWHC 1478 (KB)

Claim No: G90MA272

IN THE HIGH COURT OF JUSTICE

KING'S BENCH DIVISION

MANCHESTER DISTRICT REGISTRY

BETWEEN:

LN

(a Child, by his Litigation Friend, MC)

Claimant

-and-

BLACKPOOL TEACHING HOSPITALS NHS FOUNDATION TRUST

Defendant

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Mr CHRISTOPHER MELTON KC instructed by **FLETCHERS** for the **Claimant**

Mr JONATHAN HOLL-ALLEN KC instructed by **HEMPSONS** for the **Defendant**

Hearing dates: 29 and 30 April 2024, 1, 2, 3 and 7 May 2024

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JUDGMENT

His Honour Judge Pearce sitting as a Judge of the High Court

This judgment was handed down remotely at 10:30 am on 18 June 2024 by circulation to the parties or their representatives by e-mail and by release to the National Archives.

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INTRODUCTION

1. The Claimant was born at Blackpool Victoria Hospital (for which the Defendant is responsible) on 16 September 2016 and is therefore now 7 years old. He brings this claim for damages for personal injuries and consequential losses allegedly sustained as a result of the negligent management of his mother's labour and birth. His mother is his Litigation Friend.

2. This matter proceeded to trial before me on the preliminary issue of liability. Whilst the Defendant admits breach of duty in the management of the labour, it denies that any of the Claimant's injuries are a consequence thereof.

THE CLAIMANT'S INJURIES

3. At the beginning of the trial, I made an anonymity order. The result of that order is that the Claimant and his mother are to be known by the initials at the head of this judgment. During the course of this judgment, I shall refer to him as "the Claimant" and his mother as "MC". This may make the judgment seem somewhat impersonal. However, the Claimant, his mother and his family more generally are of course at the centre of this claim. Regardless of the consequences of his injuries, the Claimant no doubt has the usual characteristics of 7 year old children, with their ability to bring pleasure and frustration to their families in almost equal measure. Insofar as he suffers challenges because of his condition (whether as a result of the negligence of the Defendant or otherwise), his parents, siblings and wider family have my sympathy for having to deal with those issues and my admiration for what they do for him.

4. For the purpose of the trial on the preliminary issue, it has not been necessary for the parties to adduce detailed evidence of the Claimant's current condition, although his condition at birth and in the earlier neonatal period is significant, as is the general picture of his ongoing visual and developmental issues. As a result, I only have a general picture of his condition.

5. The Claimant's developmental condition as of 19 December 2019 (when he was 3) is described in a report from Dr Stephen Rose, consultant paediatrician, dated 1 January 2020 which can be summarised as follows:

    a. The Claimant has visual impairment.

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b. His gross motor skills are delayed. Whilst he can walk and run competently, he tends to trip over objects.

c. His fine motor skills, hearing and speech are also delayed.

d. His social skills are delayed and his visual impairment can cause problems in crowded areas.

6. Dr Rose later examined the Claimant at age 5 years nine months (in or around June 2022). His report following that examination (which was for the purpose of the liability trial only) does not deal with his condition in any great detail. In the joint statement, he summarises the Claimant's condition as follows:

a. Microcephaly;

b. Problems with fine motor skills;

c. Problems with gross motor skills in particular in that he cannot skip and climbs stairs one at a time;

d. Speech and language problems, including that his speech can be monotonous and repetitive and it is likely that at the very least his inability to skip is due to delay in receptive language skills;

e. Delayed social skills and problems with sleep disturbance and toileting.

7. Dr Agrawal, the paediatric neurologist instructed by the Defendant, saw the Claimant in January 2022 prior to his report, again prepared for the purpose of determining liability issues. He notes the following features of the Claimant's condition in the joint statement:

a. Microcephaly;

b. Fluent conversation, but with a monotonous voice and erratic eye contact;

c. Slightly poor fine coordination;

d. Progressive visual impairment.

8. Broadly these experts are in agreement though Dr Agrawal doubts that the Claimant truly has issues with gross motor skills, suggesting that the problems identified by Dr Rose may be due to visual impairment.

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9. The Claimant's condition more recently is reported upon by Dr Simcox, neuropsychologist instructed by the Claimant, who saw him in February 2023, and Dr Hunt, neuropsychologist instructed by the Defendant, who saw him in September 2022. Dr Simcox speaks of him having executive, attentional and social communication difficulties, leading to some areas of weakness particularly with regard to daily living skills. Dr Hunt summarised the Claimant's neuropsychological functioning at paragraph 10.7 of her report, describing the pattern as being "*low average verbal ability, low average adaptive functioning, subtle difficulties with executive functioning and intact memory*."

10. In their joint statement, Dr Simcox and Dr Hunt agreed that there is evidence of delayed development, with some persisting problems, and a record of visual problems and speech and language difficulties. They agree that there are some executive functioning problems and that the Claimant has weaknesses in daily living skills.

THE COURSE OF THE LABOUR AND DELIVERY OF THE CLAIMANT

11. MC's pregnancy with Claimant was booked on 16 February 2016, with an expected date of delivery of 3 September 2016.

12. Her pregnancy proceeded routinely and there were no concerns about fetal growth.

13. On 16 September 2016 (at 41 weeks + 2 days' gestation) MC was admitted to the delivery suite with a history of antepartum haemorrhage at home. She was contracting 1 in 4. Thereafter, the labour proceeded in summary as follows:

    a. A cardiotocograph trace[1] ("CTG") was commenced, and she had some observations which were all normal.

    b. At 09:40 a vaginal examination was performed by the midwife, and she was found to be 3cm dilated and the membranes were ruptured.

    c. At 09:55, MC was given pethidine.

    d. At 10:15, a vaginal examination was carried out to apply a fetal scalp electrode. MC was 4cm dilated at this stage.

[1] The cardiotocograph trace measures fetal heartbeat and uterine contractions during pregnancy and labour.

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e. At 10:30, MC was reviewed by Dr Gho who made a plan to commence a Syntocinon[2] infusion to increase her contractions. MC requested an epidural for pain relief which delayed the commencement of the Syntocinon. The epidural was sited by the anaesthetist at 13:20. Syntocinon commenced at 13:45 at 3ml per hour.

f. At 14.45, Syntocinon was increased to 12ml per hour.

g. At 14.50, MC was noted to be having 5-6 short contractions per 10 minutes and Syntocinon was decreased to 6ml per hour.

h. At 15.15, contractions were noted to be 6-7 in 10 minutes and therefore Syntocinon was reduced to 3ml per hour.

i. At 16:00, MC was reviewed by the obstetric registrar. The CTG was reported to show occasional episodes of shallow decelerations. She was contracting 4-5 in ten, but they were not very long lasting. A plan was made to continue the CTG and review after 20 minutes.

j. At 17:00, it was documented that the Syntocinon infusion was stopped as MC was hyper-stimulating with 6-7 short contractions in ten minutes.

k. At 17:05 a vaginal examination showed MC was fully dilated. A plan was made to commence pushing, and the doctor was to be informed if the CTG was abnormal after 30 minutes.

l. Active pushing commenced at 17:15. At this time the obstetric registrar was asked to see her because of a maternal tachycardia[3]. The CTG was reported to be normal and on examination by the midwife the vertex was almost visible on parting the labia. The plan was to continue CTG, commence active pushing, and to commence antibiotics if she remained persistently tachycardic.

m. At 17:25, MC was contracting 6-7 in ten minutes. Syntocinon was recommenced at 3ml per hour shortly after this.

n. At 17:35 a further examination was carried out by the doctor. The fetal head was now visible between contractions although contractions were short lasting.

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[2] Syntocinon is a synthetic oxytocin, used to induce or to accelerate contractions during labour.

[3] An unusually fast heart rate.

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o. At 17:40, the Syntocinon was increased to 6ml per hour with a plan to review after fifteen minutes.

p. At 17:50, it was recorded that oxytocin was causing hyperstimulation with 6-7 contractions in 10 minutes.

q. At 18:04 normal vaginal delivery occurred following which the placenta delivered. The Claimant was described as having a good heart rate of greater than 100 beats per minute ("bpm"). However he was not breathing. A Guedel airway was inserted and the paediatric team were called.

r. Cord blood pH[4] readings were recorded at birth, with an arterial pH of 6.92 and a venous pH of 7.23.

14. The Claimant's immediate post natal course:

a. The paediatric team arrived at 18:14, 10 minutes after birth. At the time, the Claimant was on the resuscitaire and was described as blue and floppy but with a heart rate >100bpm. Five inflation breaths were repeated three times, but no chest movement was seen.

b. The Claimant's Apgar[5] scores were stated to be 2 at 1 minute, 5 at 5 minutes and 5 at 10 minutes. The first of these was based on a score of 2 for heart rate with no other scoring. The second and third were based on 2 for heart rate, 1 for response to stimuli and 2 for skin colour.

c. At 18.19, irregular gasps were observed and more regular respiratory effort was seen by 20 minutes of age. He remained limp but was said to be improving. The oxygen saturation was recorded as 94% in 30% oxygen and some continuous positive airways pressure was given. No pneumothorax was seen.

d. At 18.39, the Claimant was intubated. This was successful as indicated by a colour change on the colorimetric capnography and the oxygen saturation was 94% in 30% oxygen. He was now becoming more active, and was transferred to the transport incubator to move to the neonatal unit. The endotracheal tube required repositioning due to a decrease in saturation.

[4] pH is of course a measure of acidity; the lower the figure, the more acidic.

[5] The Apgar score, named after an American physician, Dr Virginia Apgar, is a measure of the health of a newborn baby. It scores each of heart rate, respiratory effort, muscle tone, response to stimuli and skin colour out of 2, giving a total score of between 0 and 10.

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e. He arrived on the neonatal unit at 60 minutes of age. The temperature on admission was 37.6ºC.

f. Passive cooling was commenced at 18:30.

g. Initial ventilation was with pressures of 20/5 and a rate of 30 breaths per minute. He required 45% oxygen initially and was described as very active. The initial blood gas, recorded at 19.40, was pH 7.05, CO2 10, BE -11.8, lactate 8.8mmol/L.

h. Following this he was given 10ml/kg of normal saline although the indication for this was not clear.

i. At 20:00, a chest x-ray was performed which revealed a large right sided tension pneumothorax with mediastinal shift. A bolus of fentanyl and morphine infusion[6] were given but, as the Claimant remained very active, Atracurium[7] was also given.

j. Following the administration of Atracurium, the oxygen saturations decreased to 85% and as a result the endotracheal tube was removed, and replacement was attempted by Dr Hopewell the Paediatric Consultant. This was unsuccessful therefore the right side of the chest was needled whilst mask ventilation was used. Naloxone was given to reverse the effects of the drugs already given.

k. A chest drain was inserted at 21:35 and the Claimant was intubated again by Dr Cable at 21:45 following which the oxygen saturations increased to 90%. After insertion, the chest drain bubbled initially in the under-water sealed drain indicating it had been successful in draining the air.

l. A repeat blood gas at 22:00 showed pH 7.28, CO2 6.6, BE -4 and lactate 5.3mmol/L. He was ventilated on 50 breaths per minute and 85% oxygen. The temperature at 22:30 was 34.9ºC with passive cooling.

m. At this point transfer to a neonatal intensive care unit with facilities for whole body cooling treatment was requested.

n. The transport consultant suggested giving Atracurium again, repeating the chest

x-ray, IV antibiotics, surfactant and to commence passive cooling.

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[6] Both fentanyl and morphine are drugs that have a sedative effect.

[7] A muscle relaxant.

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o. At 23:16 the pH was 7.28, CO2 6.6, BE -4 and the lactate concentration was 5.3
mmol/L.

p. Following this, the Claimant was noted to be having possible seizures[8] and a dose of phenobarbitone[9] was given at 01:25. He was given 84 mg of phenobarbitone (instead of the intended dose of 66 g). Surfactant[10] was also given. Insertion of an umbilical venous catheter was attempted but failed.

q. The Claimant was admitted to St Mary's Hospital, Manchester at 04:45. At the time of transfer the temperature was 34°C.

r. On arrival at St Mary's Hospital, he remained ventilated on low ventilator settings and had a normal blood gas (pH 7.32, CO2 6.57, BE-0.4). He was on 60ml/kg of 10% dextrose and his glucose concentration was 7.6mmol/L. The cerebral function monitor (CFM) was recording and showed a normal background activity and no evidence of any seizures.

s. A head MRI scan was performed on 22.9.16 and reported on the next day. The findings were reported as follows:

> "*There is a focal round and linear area of T1 signal abnormality in the left lentiform nucleus which retains high signal on FLAIR. This does not cause signal drop out on DWi and may still represent an area of haemorrhage or calcification. Otherwise, normal appearances of the deep grey matter nuclei.*
> *Normal appearances of the white matter. Myelination is appropriate.*
> *Normal appearances of the cortex. No cortical migratory abnormality.*
> *Normal anterior and posterior pituitary. Normal corpus callosum.*
> *Normal appearances of the ventricular system, posterior fossa, brainstem and craniocervical junction."*

The report goes on:

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[8] There was some issue during the trial as to whether possible seizure activity was seen on just this one occasion or on others. Mr Melton KC conceded that all references to seizure activity appeared to be to this one incident. Having considered matters, Mr Holl-Allen KC agreed that this was correct.

[9] A drug used to treat seizures.

[10] Surfactants reduce surface tension. They are used amongst other things to treat new borns with respiratory problems.

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"*Impression:*

No MR evidence of hypoxic ischaemic encephalopathy changes.

Focal linear area of T1/FLAIR high signal abnormality in the left lentiform nucleus. Exact nature is uncertain[11]*, but may represent calcification or haemorrhage, a follow up ultrasound is recommended in a few weeks time to review this area."*

t. The Claimant was discharged from neonatal care at St Mary's on 25 September 2016. His principal problems were stated to include "*HIV grade 2 – moderate neonatal encephalopathy*."

THE PARTIES' CASES IN SUMMARY

15. The Claimant's case, in summary, is that the Defendant's admitted breach of duty managing MC's delivery of him, namely the administration of Syntocinon caused or materially contributed to him developing a hypoxic ischaemic encephalopathy[12] ("HIE"). The HIE in turn was either the sole cause of or was a material contributor to the Claimant's current developmental delay.

16. The Defendant contends that either the Claimant did not suffer HIE, but rather other neonatal insult or that, even if he did suffer HIE, that was caused not by the admitted breach of duty but by other factors. In any event, the Defendant denies that the Claimant's current developmental delay can be attributed to HIE.

17. It can therefore be seen that the central issues in the case are:

a. Whether the Claimant suffered HIE;

b. If he did suffer HIE, whether that was caused by the Defendant's admitted negligence;

c. If he suffered HIE caused by the Defendant's admitted negligence, whether that was a cause of his developmental delay and/or any other injury for which damages are recoverable.

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[11] As will be seen, one potential explanation for this abnormality is neonatal stroke, which, if present may be significant in explaining the Claimant's condition at birth.

[12] HIE can defined in the neonate as clinical evidence of brain dysfunction manifest as an abnormality in tone and conscious level sometimes accompanied by seizures (encephalopathy) caused by lack of oxygen (hypoxia) and reduced blood flow (ischaemia) to the brain around the time of birth.

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18. In considering these issues, it is necessary to look at various factors during the course of the labour and delivery, as well as the Claimant's condition at and since birth. In large part, these questions do not involve consideration of factual issues, but rather expert opinion as to the nature and cause of various features of the Claimant's condition.

COMMON GROUND

19. The parties agree the following features of MC's labour:

    a. The Claimant's mother was administered Syntocinon from 13.45.

    b. The administration of Syntocinon is admitted to have been negligent. The matter is put thus in the Defence at [7]:

        "*By 1345 there was evidence of tachysystole (excessive uterine activity), potentially attributable to the antepartum haemorrhage and/or a small placental abruption. It is accepted that in the circumstances: a further review, including a vaginal examination to assess progress in labour, ought to have been carried out before commencing Syntocinon; such a review would have identified that there had been significant progress in labour since the previous vaginal examination at 1115; and the appropriate decision would therefore have been not to commence Syntocinon, but to maintain observations.*"

        The Defendant does not suggest that Syntocinon would have been administered later. It follows that, but for the admitted negligence, the Claimant's mother would not have received Syntocinon at all.

    c. The result of the administration of the Syntocinon was that the Claimant's mother suffered uterine hyperstimulation and the Claimant at times suffered tachycardia in consequence.

    d. During labour, the Claimant's Cardiotocograph ("CTG") trace was abnormal, though the degree of abnormality is in issue.

    e. The Claimant was born with the umbilical cord around his neck, a so-called nuchal cord.

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f. The nuchal cord led to the cord being compressed shortly before delivery, causing acute hypoxia[13] with an associated fetal bradycardia;

g. At birth, there was evidence of metabolic acidosis, as demonstrated by the arterial cord gas pH reading of 6.92.

h. The acidosis was at least partially explicable by acute hypoxia caused by the nuchal cord;

i. The Claimant may have developed hypoxic ischaemic encephalopathy[14] ("HIE");

j. If he did so, the HIE was of mild to moderate severity;

20. It is common ground in respect of the Claimant's condition since birth that:

a. He is microcephalic;

b. He suffers symptoms of developmental delay and executive functioning problems, as described by Dr Simcox and Dr Hunt.

c. He has speech and language problems, problems with fine motor skills and possibly problems with gross motor skills, as described and analysed by Dr Rose and Dr Agrawal.

d. He suffers retinal dystrophy. This is described by the ophthalmologists and agreed by them and the geneticists to be of genetic cause, unrelated to the management of the labour and delivery.

AREAS OF DISAGREEMENT

21. In terms of the differing opinions of the experts in the trial the following areas are of particular significance:

a. Whether the Claimant's CTG is suggestive of chronic hypoxia rather than another cause such as neonatal stroke;

b. Whether the Claimant's condition at birth was suggestive of chronic hypoxia;

[13] Lack of oxygen

[14] HIE can be defined in the neonate as clinical evidence of brain dysfunction manifest as an abnormality in tone and conscious level sometimes accompanied by seizures (encephalopathy), caused by lack of oxygen (hypoxia) and reduced blood flow (ischaemia) to the brain around the time of birth.

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c. The significance of the cord gas pH readings;

d. Whether the MRI scan was consistent with chronic hypoxia;

e. Whether the apparent seizure was indicative of chronic hypoxia;

f. Whether the claimant's condition since birth supports the conclusion that he suffered injury due to HIE rather than any other cause.

22. Taking these factors together, the Claimant argues that the court can be satisfied on the balance of probabilities that, whilst a period of acute hypoxia may have contributed to his neonatal condition, there was an earlier period of chronic hypoxia, consequent on uterine hyperstimulation, which contributed to the Claimant's condition at birth and his subsequent developmental problems.

23. The Defendant argues that the progress of the labour as evidenced by the CTG trace, the Claimant's condition at birth and his subsequent constellation of difficulties are not consistent with the mechanism of chronic hypoxia but rather have other explanations. In particular, the Claimant's neonatal condition was suggestive of stroke; and his retinal dystrophy and microcephaly are suggestive of a genetic syndrome which, whilst as yet unrecognised, is the probable cause of some or all of the Claimant's difficulties.

THE TRIAL

24. The following witnesses were called at trial:

a. For the Claimant:

i. Mr Duncan Irons, Consultant Obstetrician and Gynaecologist, whose report is dated February 2024;

ii. Dr Stephen Wardle, Consultant Neonatologist, whose report is dated February 2024;

iii. Dr Shivaram Avula, Consultant Radiologist, whose report is dated February 2024;

iv. Dr Stephen Rose, Consultant Paediatrician, whose Condition and Prognosis report is dated 1 January 2020 and whose liability report is dated February 2024;

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v. Professor Jane Ashworth, Consultant Paediatric Ophthalmologist and Ophthalmic Surgeon, whose report is dated February 2024;

b. For the Defendant:

i. Mr James Penny, Consultant Obstetrician and Gynaecologist, whose report is dated August 2022;

ii. Dr Nimish Subhedar, Consultant Neonatologist, whose report is dated August 2022, with supplemental letter dated 20 December 2023;

iii. Dr Daniel Connolly, Consultant Radiologist, whose report is dated August 2022, with supplemental letter dated 27 November 2023;

iv. Dr Shakti Agrawal, Consultant Paediatric Neurologist, whose report is dated August 2022, with supplemental letter dated November 2023;

v. Mr John Elston, Consultant ophthalmic Surgeon, whose report is dated August 2022, with supplemental letter dated November 2023;

25. The parties also relied on the written evidence of two other experts:

a. For the Claimant:

i. Dr Angela Simcox, Consultant Paediatric Neuropsychologist, whose report is dated February 2024;

ii. Professor Dhavendra Kumar, Consultant in Clinical Genetics and Genomic Medicine, whose report is dated February 2024.

b. For the Defendant:

i. Dr Katie Hunt, Consultant Clinical Psychologist and Paediatric Clinical Neuropsychologist, whose report is dated March 2023, with supplemental letterer dated 24 January 2024;

ii. Professor Andrea Nemeth, Consultant in Clinical and Neurogenetics, whose report is dated November 2023.

26. Each of these experts contributed to joint statements as follows:

a. Irons and Penny (obstetrics), dated 12 March 2024

b. Wardle and Subhedar (neonatology), dated 12 March 2024;

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c. Avula and Connolly (neuroradiology), dated 12 March 2024;

d. Rose and Agrawal (paediatrics/paediatric neurology[15]), dated 13 March 2024;

e. Ashworth and Elston (ophthalmology), undated following discussion on 11 March 2024;

f. Simcox and Hunt (neuropsychology), dated 14 March 2024;

g. Kumar and Nemeth (genetics), dated 14 March 2024.

THE EXPERT EVIDENCE

27. In drawing the threads of the expert evidence together, it is clear that the course of the Claimant's delivery, his neonatal state, his longer term outcome and the available objective signs do not hang neatly together to produce a coherent picture. The various experts have each sought to address the issue from their own viewpoints, looking insofar as they felt able to at the opinions of experts from other disciplines to inform their opinions. Nonetheless, no expert has been able to come up with a formulation which is consistent with all of the evidence before the court.

28. In that circumstance, one potential route to determining the issues on the balance of probabilities is to look at the quality of expert evidence on the various issues, preferring that opinion which, overall, is more internally consistent. To that extent, an assessment of the quality of the expert evidence is particularly important.

29. During the course of the cross-examination of expert witnesses, each conceded on at least one occasion that his or her original report did not fully reflect their opinion and that it was only in the joint statement that their complete reasoning could be seen. In a complex clinical negligence case of this kind, where there is varying evidence as to the Claimant's condition and differing opinions as to the significance of that evidence, it is not surprising that an expert's opinion is only fully fleshed out in the joint statement or even in the witness box. Having heard the experts here, I conclude that, in general, they have each sought to assist the court but that the complex and changing clinical picture as well as the development and thinking by experts instructed for the opposing party in general terms explains certain inconsistencies in their evidence.

[15] For the purpose of simplicity within this judgment, I shall refer to Dr Rose and Dr Agrawal collectively as "the paediatricians". In so doing, I do not mean to diminish either Dr Agrawal's particular specialty as a paediatric neurologist, nor the potential significance of that in this case, a point dealt with further below.

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Setting aside for the moment the evidence of the paediatricians, I do not consider that those inconsistencies significantly undermine the force of the expert evidence.

30. The paediatricians were however subject to rather more vigorous criticism. This is perhaps not surprising. In the case of this nature, it is necessary for anyone opining on the cause of the Claimant's condition to consider a series of factors, some within their expertise, some outside of it. The paediatricians have doubtless been asked to consider the evidence from experts of other disciplines and, at least in their initial reports, will not have known in any great detail what the other side's experts were going to say. They are undoubtedly dependent on those other experts for significant parts of the material that they have to bear in mind in seeking to reach an overall conclusion as to the cause of the claimant's condition. Further, whilst the role of the expert is not to act as an advocate for any particular explanation of the condition, it is easy to see in a case where many factors are to be brought into the equation that an expert might be somewhat tempted to make assumptions that support their overall conclusion.

31. In his cross-examination of Dr Rose and his closing submissions, Mr Holl-Allen KC made a number of points critical of Dr Rose:

    a. He appeared, at least in his initial condition and prognosis report, to have assumed that the Claimant had suffered HIE rather than to have analysed the evidence before coming to that position. Given that whether the Claimant in fact suffered HIE at all is a central issue in the case, it was suggested that Dr Rose had assumed that which needed to be proved.

    b. During the course of cross-examination, Dr Rose withdrew his suggestion at [7] of the section headed Case History in his liability report the effect that the Claimant had suffered "*some 15 to 20 minutes of acute catastrophic hypoxia.*" Again, it was suggested that this was an example of his making assumptions about the Claimant's condition.

    c. Whilst he had not expressly attributed the Claimant's microcephaly to a period of hypoxia in either of his reports or the joint statement, in oral evidence he expressed the view that it probably was so attributable

32. On behalf of the Claimant, Mr Melton KC was critical of the evidence of Dr Agrawal for several reasons:

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a. His opinion that the Claimant suffered neonatal stroke is one that he alone holds and is not shared by other experts instructed for the Defendant;

b. He failed to explain how the alleged neonatal stroke was consistent with neonatal acidosis;

c. Notwithstanding his opinion that the Claimant had not suffered HIE, he applied the Sarnat classification to assess the Claimant's condition.

d. He copied sections from Dr Rose's report as to the Claimant's clinical history, in particular the misinterpretation of the note of December 2016, without independently verifying that history.

e. Whilst he relies upon the absence of neuroradiology evidence as supportive of his opinion, he did not, within his reports, mention the benign effect of cooling on neurological appearances and clinical signs and symptoms.

33. In fact, as with experts more generally, I did not consider that either paediatrician was an inherently unreliable witness or was doing anything other than seeking to assist the court. As regards Dr Rose, whilst I accept that the condition and prognosis report reads as though he assumed the Claimant had suffered HIE, I found his oral evidence to have been given carefully with consideration of the various factors. He was willing to defer to the geneticists on the significance of the genetic cause of the retinal dystrophy and, perhaps more significantly, to Dr Agrawal on the argument that the Claimant's retinal dystrophy might have contributed to some of the other features of the Claimant's condition. I did not consider this to be suggestive of an expert witness who had a closed mind.

34. As regards Dr Agrawal, I consider his opinion of perinatal stroke as a cause of the Claimant's encephalopathy to be out on a limb, a factor dealt with below. But his use of the Sarnat Classification to assess the severity of the Claimant's Condition was understandable in a context where Dr Rose himself was using it. His error as to the note of December 2016 is of course unfortunate but insofar as it flows from him accepting Dr Rose's account without independently verifying it, does not suggest that he is in some way seeking to bend the evidence in a way unfavourable to the Claimant's case or to Dr Rose's opinion.

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35. It follows from the above that I do not consider it possible in this case to simply favour the opinion of one expert over another as being more obviously persuasive on the central issues. There are strengths and weaknesses to the position of each expert and it is necessary to look, issue by issue, at the varying positions and to reach conclusions on the central points in the light of that material.

THE DISPUTED MATTERS

(1) Is the Claimant's CTG suggestive of chronic hypoxia?

36. At paragraph 7 in their joint statement, the obstetricians agree that contractions of the uterus cause placental blood flow to be impaired. When the uterus is hyper stimulated and over contracting, the placental blood flow may deteriorate causing the oxygen supply to the fetus to become impaired and the fetus to become hypoxic. This hypoxia may in turn lead to acidosis since, as Mr Irons explained in his evidence, the reduced supply of oxygen prevents the fetus obtaining energy from that source and may lead to anaerobic metabolism, where lactic acid is released as a byproduct.

37. The obstetricians agree that hyperstimulation was present from around 2.15pm. They also agree that the CTG trace shows shallow decelerations[16] with reduced variability[17]. Overall, they agree in answer to question 10 in the joint statement that the CTG shows some accelerative episodes but over 90% of the trace shows reduced variability. From 17.15, active pushing commenced. There was initially a tachycardia (which was a fetal response to being stressed or hypoxic) and thereafter there was a fall in baseline heart rate[18] followed by a final bradycardia[19].

38. Mr Irons, the obstetrician instructed on behalf of the Claimant, considered that the shallow decelerations and reduced variability were due to hypoxia secondary to hyperstimulation and that the late drop in baseline heart rate was a consequence of acute on chronic hypoxia. He accepted that the CTG trace showed some accelerations which were an indicator that the fetus was not acidotic until very late on in the labour, with episodes at 14.40 and 15.25. He was doubtful that the trace at 16.40 was

[16] Reductions in fetal heart rate

[17] Variation in the fetal heart rate between one beat and the next. Variability is a sign of the fetus' ability to respond to its environment so reduced variability suggests that the baby is responding poorly and may be due to hypoxia.

[18] The average heart rate of the fetus measured over a period of time.

[19] An unusually slow heart rate

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accelerative, thinking it more likely that the word "semi-recumbent" written on the trace indicated that this was a change in trace consequent upon the mother moving. In any event it followed only 6 minutes of possible slight improved variability from which it was no possible to draw any firm conclusion. He also doubted that the trace at 17.00 showed an acceleration but considered it to be so unclear that he could not draw any conclusion about what was happening. His opinion was that the period of chronic hypoxia had caused the Claimant to be exhausted as labour moved into the second stage.

39. In cross-examination, Mr Irons accepted that not all cases of uterine hyperstimulation lead to fetal hypoxia and where it does so, the hypoxia will be relative not total.

40. In contrast, Mr Penny, instructed on behalf of the Defendant, considered the "*dramatic"* change in the baseline rate and the final bradycardia to be a consequence of the cord compression as the cord tightened. He considered that the presence of accelerations shows that the fetus was able to respond and was generally an indication that the baby was not hypoxic. He accepted that there was reduced variability for 90% of the trace, with it sometimes being less than 5bpm. He accepted this was due to hyperstimulation of the uterus.

41. Mr Penny considered there to be particularly significant points showing a reactive episode at around 14.40, and an accelerative episode between 15.20 and 15.30. The latter was preceded by 20 minutes of normal CTG trace, a reassuring feature. Further, at around 16.40 and 17.00 he considered that there were further accelerative episodes.

42. When asked about Mr Irons' theory that the Claimant was exhausted at the end of the first stage, he said that he did not accept that the CTG showed this and in particular that the accelerative change at 17.00 in particular suggested he was doing well at that stage. He did not think that second stage trace showed decompensation from earlier chronic hypoxia.

43. During cross examination, Mr Penny accepted that there were periods when the administration of Syntocinon was reduced or stopped but that it was restarted. In particular, he accepted that his reference at paragraph 5.19 of his report to Syntocinon being "finally stopped" at 17.00 was mistaken because in fact it was restarted at 17.15.

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44. In the second stage of labour, the trace did not, in Mr Penny's opinion follow a classic pattern, he considered the deceleration to be due to interruption of the blood flow on account of cord compression, but he said it was difficult to explain the fact that the fetal heart rate returned to a lower baseline.

45. The neonatologists agreed that uterine hyperstimulation can cause fetal hypoxia and consequent brain injury. They said that shallow decelerations and sustained tachycardia may be signs of this but they deferred to the obstetricians as to their significance.

46. The neonatologists also agree that the Claimant suffered a terminal bradycardia from 18.01 until 18.04, consistent with acute cord compression secondary to a nuchal cord.

(2) Was the Claimant's condition at birth suggestive of chronic hypoxia?

47. The obstetricians agree that the Claimant was born at term and that there was no evidence of growth restriction. In consequence, he would not be prone to be born in poor condition. However, at birth, he was suffering an apparent encephalopathy, was not spontaneously breathing and needed resuscitation.

48. I have dealt above with Mr Irons' explanation of the CTG trace and his theory that the fetus was exhausted at the beginning of the second stage as a result of chronic hypoxia during the first stage. In examination in chief, he compared the position of the Claimant who was exhausted at the end of the first stage with a person who was being asked to take part in a 100m sprint after running 400m at full speed. The runner would not have the reserves to run a further 100m and therefore would finish the 100m in far worse condition than another runner doing the 100m who had not just taken part in the 400m. The latter would finish the 100m exhausted. The former would finish not just exhausted but hypoxic and probably acidotic. The corresponding result in the Claimant's case was that, at the end of the first stage, he did not have the reserves to deal with the acute hypoxia of the second stage of labour and the outcome at birth was his depressed condition.

49. In oral evidence, Mr Irons said that a cord around the fetal neck is a common experience in labour. The typical position with such a child is that they would be floppy for the first five minutes. Indeed, he noted that the Claimant was delivered within four minutes of the bradycardia being recorded. This suggested that the period

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of acute cord compression was only very short and that, correspondingly, one would expect only mild symptoms in the newborn with a rapid recovery. However, the Claimant's condition was very different from that, with low Apgar scores, abnormal blood gases, and the need for prolonged resuscitation and cooling.

50. Mr Penny accepted in the joint statement (and did not retreat from this position in oral evidence) that there was evidence of chronic hypoxia. However that was only mild. The wide arterial venous difference suggests that there was a much more significant acute insult prior to delivery.

51. The neonatologists noted in their respective reports that the Claimant had signs of encephalopathy and that he was treated for this. Neither disputed this diagnosis and they agreed that the Claimant's encephalopathy, taken together with his subsequent evidence of possible seizure and the evidence of cord pH acidosis (see below), led to the conclusion that the cause of the encephalopathy was hypoxic ischaemia. However, they agreed that it was unusual to find on the one hand a heart rate of above 100, with on the other hand very severely depressed Apgar scores for respiration, muscle tone and response to stimuli, as was the case here. They considered the HIE to be in the mild-moderate range of the Sarnat classification, based on neurological signs and the possibility that he had suffered one or more seizure.

52. Dr Wardle, instructed by the Claimant, accepted the plausibility of Mr Irons' theory that the Claimant was exhausted by a period of chronic hypoxia consequent upon uterine hyperstimulation and was the less able to deal with the late acute hypoxia caused by cord compression. He considered that the condition of the Claimant at birth, with a fast improvement in heart rate, was suggestive of a terminal event, but that the cord gas pH, the resuscitation needed and the delay in onset of breathing suggested more than simply three minutes of hypoxia and consequent bradycardia from cord compression. In particular, Dr Wardle stated that a period of bradycardia for 3 to 4 minutes may have led to the requirement for some resuscitation but not of the degree that was necessary here. He also accepted that the cord compression in the second stage was a substantial contributor to the Claimant's encephalopathy but that he had not stated this to be the case in his report at [5.1]. He maintained that he had had this mechanism in mind and that he had expanded upon it in the joint statement.

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53. When pressed further on the significance of the period of chronic hypoxia that he said was consequent upon uterine hyperstimulation, Dr Wardle said that, but for the cord compression and acute hypoxia, the Claimant would still have required resuscitation but would have responded more quickly. Mr Holl–Allen KC explored whether Mr Wardlaw was saying that the pneumothorax would have been avoided with lesser resuscitation. Dr Wardle replied that he could not say that this was so.

54. Dr Subhedar stated that he considered the Claimant's condition at birth to be explained by the period of terminal bradycardia and acute hypoxia consequent upon cord compression. During cross-examination, he agreed with Mr Melton KC that both he and Dr Wardle considered that the Claimant had suffered HIE but that the difference between his opinion and that of Dr Wardlaw was whether the sole cause of that was the terminal bradycardia consequent upon cord compression or whether the earlier chronic hypoxia contributed to some extent.

55. Dr Subhedar agreed during cross-examination that not all children who had suffered four minutes of bradycardia would need cooling or would display evidence of encephalopathy. Indeed he agreed that the vast majority would not.

56. Whilst Dr Wardle and Dr Subhedar are agreed that the evidence suggested that the claimant had suffered neonatal encephalopathy due to hypoxic ischaemia, Dr Agrawal considered that the cause of his condition was more likely a perforator artery stroke. His opinion that the Claimant suffered a perforator artery stroke was based upon the evidence of Dr Connolly as to the abnormalities visible in the left hemisphere of the MRI scan.

57. He described the course of such neonatal stroke as follows in his report at [4.16]: "*Most commonly the babies evince impaired consciousness, breathing problems, and seizures. In cases of acute stroke, it is typical for the newborn to initially appear relatively healthy, reflected by high Apgar scores, and then deteriorate*."

(3) What is the significance of the cord gas pH readings?

58. Cord gas pH readings are measures of fetal acid levels taken just after delivery by measuring the pH of blood in the umbilical cord. Both arterial and venous blood is conventionally measured.

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59. It is common ground that cord gas pH readings are of value in measuring acidosis[20]. Acidosis itself may be a consequence of respiratory factors that lead to increased levels of carbon dioxide in the blood and consequent higher acidic levels but may also be due to a shortage of oxygen. In the case of cord pH, one is not concerned with a respiratory contribution (prior to birth the fetus is not breathing) but rather a metabolic acidosis due to hypoxia.

60. As noted above, Mr Irons for the Claimant accepts that the arterial cord blood pH reading was very low as a result of cord compression from the cord being around the neck. However, he considered the mildly abnormal venous cord blood pH to be indicative of hypoxia due to hyperstimulation. He stated that, in the umbilical cord, there were two arteries and the vein. The arteries are more muscular and consequently the vein will be compressed much more readily. Thus it comes as no surprise that the acidosis apparent in the arterial blood is not apparent in the venous blood, the blood flow having been compromised. This mild acidotic level in the venous blood is therefore indicative of a more long-standing hypoxia.

61. He put his opinion thus in the joint statement:

> "*The gases show very clearly that when cord was fully compressed an already mildly acidotic fetus from the labour due to chronic hypoxia was then subjected to additional effect of the acute insult giving the typical difference in venous and arterial values. So unequivocal evidence of two separate injuries namely chronic with acute on top at end*."

62. In cross-examination, Mr Irons accepted that, with hypoxia secondary to uterine hyperstimulation over a substantial period prior to delivery, one might have significant acidosis in the venous cord blood pH result, though the reading will depend on how long the hypoxia had lasted. In this case, the Syntocinon was reduced at one point and was stopped at another. In both cases, the CTG trace improved, suggesting that the hypoxia had reduced. The venous pH of 7.23 was, he agreed, not to be described as more than mildly acidotic.

63. Mr Irons was pushed on how it might be that, given the duration and extent of chronic hypoxia that he said was present, the venous result was not lower. He referred to

[20] Increased acidity in the blood.

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research originally from Singapore to the effect that a term fetus that is normally grown can generally withstand the stresses of labour for several hours before becoming acidotic due to hypoxia. He did not accept that, if his theory that there had been a significant period of chronic hypoxia for about four hours prior to delivery was correct, one would expect the venous and arterial blood gases to be equally low. In this respect, he returned to his metaphor of the runner who would finish the 100m sprint in much worse state if it immediately followed a 400m run. As he put it at one point "*if you have been hypoxic for a while, it does not take much to tip you into acidosis.*"

64. Mr Holl–Allen KC put to Mr Irons that the more obvious explanation for the discrepancy between the arterial and venous blood pH results was that the nuchal cord led to a terminal bradycardia with complete cessation of blood supply. Mr Irons responded that this explanation did not take into account the evidence of hyperstimulation and what he described as "a textbook CTG" for hypoxia. He accepted that a baby born with a cord around the neck would be likely to show some degree of insult but he considered that the CTG showed that this insult had been worsened in the Claimant's case. As he put it, the evidence of hyperstimulation coupled with a fetal heart trace showing reduced variability would generally indicate that the fetus was hypoxic.

65. It should be noted that Mr Irons also commented in the joint statement on the raised lactate level apparent at 19.40, stating that it "*strongly*" supported his thesis of acute on chronic hypoxia. Dr Wardle, the neonatologist instructed on behalf of the Claimant, did not agree with this opinion. There is little doubt that a neonatologist is better placed to advise on the relevance of raised lactate levels in the newborn baby and Mr Irons said in cross examination that he would defer to neonatologists on this issue.. Unsurprisingly, given the opinion of Dr Wardle, the Claimant did not adopt this aspect of Mr Irons' opinion in closing submissions. Mr Holl–Allen KC for the Defendant relies upon this as evidence of Mr Irons moving outside of his area of expertise and asserts that he was a less persuasive expert than Mr Penny.

66. In contrast, Mr Penny says:

> *"The arterial cord pH is low. The venous pH was marginally low. This wide difference was due to the critical cord compression at delivery. There are no*

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readily available normal ranges for venous cord pH. However a level of 7.23 is very mildly reduced and commonly seen. It suggests that the baby was coping well with the excessive uterine activity. In my opinion, the arterial cord gas was very low as a result of the cord compression from the cord around the neck."

67. But for the cord compression, Mr Penny considered that the arterial cord blood pH would have been marginally low, probably less than 7.23 but not as significantly reduced as was in fact the case. In re-examination he suggested that the probable reading would have been around 7.2 and made the point that many babies are born with cord blood readings of around this level yet do not have a poor outcome.

68. Mr Penny accepted that he had stated at [5.26] of his report that "*the relatively normal venous cord pH would suggest that the prolonged hyperstimulation was not the predominant and certainly not the only cause of the final acidosis in the arterial sample."* He accepted that this might be taken to indicate that he considered the hyperstimulation to be a significant rather than merely minor cause of the acidosis. His explanation for this use of language was that the terms hypoxia and acidosis tend to be used interchangeably by obstetricians and that, though his language was lax, his point was that the hyperstimulation caused hypoxia, not acidosis.

69. I have some difficulty with this explanation. It is not easy to take the reference to "*the final acidosis in the arterial sample"* to be a reference simply to hypoxia which might in turn cause acidosis (but did not significantly do so here),. The cord pH is a direct measure of acidosis not hypoxia, even if the hypoxia might cause that acidosis. It follows that Mr Penny must have meant in [5.26] of the report that the hyperstimulation was one of the causes the ultimate acidosis.

70. Mr Penny rejected the argument advanced by Mr Irons that there was hypoxia in the first stage of labour that led to the mildly acidotic pH reading in the venous blood gas. He considered that the fetus was not hypoxic at all until the second stage (as demonstrated by the acceleration at 17.00) but that the chronic hypoxia which he accepted had occurred as a result of uterine hyperstimulation contributed to a small degree to hypoxia in the second stage.

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71. The neonatologists agree that the there was significant difference in paired blood gas pH as between the arterial and venous bloods, suggesting that the Claimant was exposed to a relatively short period of hypoxia-ischaemia towards the end of labour.

72. Dr Subhedar considered that the most plausible explanation of this was the nuchal cord. Whilst the uterine hyperstimulation prior to this may have caused recurrent episodes of partial hypoxia-ischaemia, he thought this unlikely because one would have expected greater abnormality in the venous cord gas pH in that case. In cross examination, he indicated that he disagreed with Mr Penny's opinion that chronic hypoxia made some contribution to the acidosis.

73. Dr Subhedar considered the venous cord gas reading to be only mildly abnormal. He referred to a Swedish study of 15,000 healthy pregnancies and deliveries in which a pH of 7.23 was on the 5th percentile, which would be considered on the borderline of normality. The relative normality of the venous cord gas pH was a strong argument against chronic hypoxia. Ultimately, he considered that, if one were persuaded that the Claimant suffered HIE, it was more likely to be due to the terminal bradycardia consequent upon the cord compression. Any earlier chronic hypoxia probably had no effect.

74. Dr Wardle on the other hand considered that this was probably a case where there was some hypoxia due to uterine hyperstimulation, followed but a period of acute cord compression secondary to the nuchal cord causing hypoxia-ischaemia. As I have noted, he accepted the thesis that this earlier chronic hypoxia may have led to the fetus being more vulnerable to a shorter period of acute profound hypoxia. He stated that the arterial pH was lower than he would have expected for a period of terminal bradycardia as short as 3 to 4 minutes and considered that a probable explanation of this was a preceding period of chronic hypoxia. He accepted that, but for the period of cord compression shortly before birth, the arterial and venous pH readings would have been closer together.

75. It was put to Dr Wardle that, had there been a period of chronic hypoxia sufficient to lead to the Claimant's encephalopathy, one would have expected lower venous pH. He accepted that one might have expected a lower venous pH in that circumstance, but noted that hypoxia does not necessarily cause severe acidosis.

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(4) Was the MRI scan consistent with chronic hypoxia?

76. The neuroradiologists found no evidence of congenital brain malformation in the scan of 22 September 2016. They agreed further that there was no evidence of hypoxic ischaemic damage, whether caused through an acute profound route or through a chronic partial route. Dr Avula said that, absent the therapeutic effect of cooling, in a case of moderate or more severe hypoxic injury which had carried on for 10 minutes or more, one would normally expect to see radiological changes on an MRI scan at six days. On the other hand, if the damage was only mild, it might not be visible. Dr Connolly said, during his oral evidence, to similar effect that, prior to the use of therapeutic cooling, one would expect to see evidence on MRI scan if ischaemia was going to cause long-term damage but that in the era in which therapeutic cooling is used, children or seen with minor neurodisability notwithstanding having had normal scans.

77. Dr Connolly accepted that he had not referred to the therapeutic effect of hypothermia in the course of his report. He pointed out that it was referred to in the joint statement.

78. It was further common ground between the neuroradiologists that there is evidence of abnormalities in the left caudate nucleus and the left globus pallidus. Dr Avula also thought that there was evidence of abnormality in the adjacent anterior limb of the internal capsule.

79. Dr Avula for the Claimant thought that the abnormalities were probably due to thrombus but Dr Connolly for the Defendant thought they were probably evidence of arterial stroke.

80. Dr Avula accepted that the MRI scan was not typical either for thrombus or arterial stroke, but considered the appearances on the MRI scan to be more probably that of thrombus of the left thalamostriate vein, in particular because

    a. Bright areas in the T1 sequence were suggestive of haemorrhage, consistent with thrombus;

    b. The veins looked more prominent on the left side of the brain, suggesting congestion of the vein.

However, he accepted that perforator artery stroke was a possibility, albeit less likely.

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81. During cross-examination, Dr Avula was taken to his report at [1.1] where he deals with his findings on the MRI scan. He accepted that there were bright areas on the right-hand image (the left-hand side of the brain) in the perforator artery territory but he said that the images did not show the typical wedge shape which one finds in large arterial stroke. Given the closeness of the structures, he was not persuaded that this probably showed perforator artery stroke. During the course of cross examination, Dr Avula was willing to consider an alternative cause to his favoured opinion, but was ultimately not persuaded that the radiological changes were probably due to stroke rather than thrombus.

82. Dr Connolly considered that the radiological changes were more consistent with perforator arterial stroke. He said so because:

    a. There is no evidence of acute infarction on the MR scan;

    b. There is no evidence of haemorrhage on b0 diffusion weighted imaging;

    c. The distribution of high T1 signal abnormality is in an arterial perforator distribution.

    When asked about venous thrombus as a possible cause, Dr Connolly stated that he did not agree that the fact that abnormality was unilateral made this more probable. He stated that venous infarction can be bilateral whereas perforator artery stroke can be unilateral.

83. Dr Avula and Dr Connolly agreed that perinatal asphyxia has associations both with perforator artery stroke and venous thrombosis.

84. There was a slight difference between Dr Avula and Dr Connolly on the timing of the radiological changes that are suggested to be either stroke or thrombus. Dr Avula considers that, given the lack of diffusion visible on the scan, if this were a perforator artery stroke it probably occurred 5 to 7 days before the scan on 22 September 2016. In coming to this opinion, Dr Avula acknowledged that the absence of diffusion weighted changes is normally taken as an indication that the stroke occurred at least seven days earlier. However he stated that this was a small lesion and, if a stroke, he considered that the period might be slightly shorter. This would be consistent with a neonatal insult.

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85. Dr Connolly considered that the lack of abnormality on diffusion weighted imaging suggested that the stroke occurred at least 7 days before the MR scan. In the joint statement, he used the word "strongly" to describe his opinion on this point. In oral evidence, he was less strong in his opinion and acknowledged some force in Dr Avula's argument that the period might be a little shorter than 7 days.

86. I have noted above that Dr Agrawal attributed the Claimant's neonatal encephalopathy to perforator artery stroke rather than hypoxia ischaemia. In considering the circumstances of this injury, he considered that the timing of this would probably be around the time of birth, probably due to the cord occlusion consequent upon the nuchal cord, therefore slightly less than the 7 days favoured by Dr Connolly.

87. The neuroradiologists agreed that there was no evidence of optic nerve atrophy on the MRI scan. If it had been present then, it would been present since before birth. However, if it was caused by HIE, it would not have been apparent on scan until later.

88. As with the neuroradiologists, it is common ground between the neonatologists that, in term or near-term babies, therapeutic hypothermia[21] is associated with reduced risk of death and disability. It also (disproportionately to its reduction of the risk of disability) reduces the chance of neuroradiological abnormalities. It follows from this that cooling of the Claimant may explain why he suffered chronic hypoxia without having either the pattern of injuries which is typical of chronic hypoxia or any radiological abnormalities visible on brain scan.

89. As Dr Wardle puts it, there are three possible explanations of the absence of evidence of neuroradiological damage:

    a. There was no period of hypoxic ischaemia sufficient to cause damage;

    b. There was hypoxic ischaemia sufficient to cause damage, but that damage was avoided because of the therapeutic cooling;

    c. There was hypoxic ischaemia that caused the damage, but it is not visible on then neuroradiological evidence because of the therapeutic cooling.

90. The neonatologists noted the suggestions from the radiologists that the MRI scan showed either perinatal arterial stroke or venous thrombus. They accepted that either of these conditions may be associated with hypoxia-ischaemia but stated that there

[21] Cooling

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was no known causative link between the two. Dr Wardle agreed in cross examination that one could not attribute either a perinatal arterial stroke or venous thrombus to the period of hypoxia ischaemia that he felt the Claimant had been exposed to as a result of uterine hyperstimulation.

91. The neuroradiologists agreed with the neonatologists that it is well recognised that therapeutic hypothermia decreases the risk of brain tissue injury in infancy as a result of HIE. Accordingly, a normal neonatal MRI following therapeutic hypothermia would not exclude brain injury secondary to hypoxic ischaemic brain injury.

92. During cross-examination, Dr Subhedar conceded that he had not mentioned therapeutic cooling and its potential effect on neuroradiology in the course of his report, though this had been mentioned in the joint statement. He denied that his failure to mention this in his own report was an indication that he was not being even handed. He accepted that, in retrospect, he should have dealt with this issue, since it is known that the absence of neuroradiological evidence may be neutral on whether neurological damage had actually been caused.

93. Dr Connolly made the specific point that it is recognised in population studies that hypothermia has a modulating effect both on the extent of neurological impairment and developmental delay and on the extent of abnormalities visible on MRI scanning. However, the extent of that modulation cannot be quantified in the individual case.

(5) Was the apparent seizure indicative of chronic hypoxia?

94. As Mr Melton KC pointed out during closing submission, it is necessary for the court to consider whether it is able on the balance of probabilities to make a finding in respect of whether the episode described shortly after 23.16 on 16 September 2016 was in fact a seizure. The medical evidence is postulated on this being a possible seizure (see for example the neonatologists at [7] in the joint report). That description reflects the uncertainty as to whether in fact the Claimant suffered a seizure but factors the possibility that he did into the grading to the HIE and therefore the material that may be used to predict the possible range of outcome.

95. During the course of re-examination, Dr Wardle appeared to be stating that it was necessary for the court to determine whether the Claimant had in fact suffered a seizure in order to determine whether his HIE fell into the mild or moderate category

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of the Sarnat classification. This however is not how I had understood his earlier evidence, whether in the joint statement or in his oral testimony in chief and in cross examination. Rather, he appeared to be stating that the possibility that there were seizures on this one occasion (but noting that there was no evidence of seizures on any other occasion) had left the experts in a position where they simply could not be confident whether or not the Claimant had suffered seizures, hence putting him on the cusp of the mild and moderate categories.

96. On any version of events, the presence of a seizure would be consistent with a period of chronic hypoxia but would not be diagnostic.

(6) Does the Claimant's condition since birth support the conclusion that he suffered injury due to HIE rather than any other cause?

97. I have set out in the Introduction section above, a summary of the opinions of the paediatricians on the Claimant's current condition, to the effect that he suffers microcephaly, some problems with fine motor skills, possibly problems with gross motor skills and speech and language problems, with some evidence of developmental delay.

98. His visual difficulties flow from reduced visual acuity due to retinal dystrophy which is agreed by all experts to have been a genetic cause and not a consequence of the admitted breach of duty. The ophthalmologists agree that the Claimant also has evidence of optic nerve atrophy. There was no early neuroradiological evidence of this, indicating that the atrophy was not congenital. The ophthalmologists agree that optic nerve atrophy could be a consequence of a period of hypoxia or could be secondary to the retinal dystrophy.

99. Professor Ashworth stated in her oral evidence that it was probable that the claimant had suffered cerebral visual impairment as a result of HIE that was a cause of some of his current problems, though she considered it difficult to identify to what extent his visual problems had that cause rather than being a consequence of retinal dystrophy. She agreed that the retinal dystrophy was towards the severe end of the spectrum, placing the Claimant in the partially sighted category. She accepted that, in coming to her opinion as to the cause of visual problems, she was assuming that the Claimant had in fact suffered HIE. She accepted that the Claimant's optic nerve atrophy is as

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capable of being explained by the retinal dystrophy as it is of being due to some other cause relating to cerebral visual impairment. She also accepted that the evidence of the neuropsychologists pointed against the Claimant suffering a visual processing problem. Ultimately, she accepted that it was difficult to say whether the presumed HIE had contributed at all to the Claimant's visual impairment.

100. Dr Elston, instructed by the Defendant, essentially agreed with this final comment. He was not able to identify whether cerebral visual impairment was a contributor to the Claimant's ongoing visual problems.

101. However the geneticists agree that the claimant now has evidence of retinal dystrophy which is probably of a genetic cause. Not all genetic mutations are identified and as yet that is the case with the Claimant. They also stated the following in the joint statement:

> "*We agreed to propose a list of potential causes of Noel's clinical features:*
>
> *1. a single genetic cause to explain the retinal dystrophy and the other features (a syndromic retinal dystrophy).*
>
> *2. a single genetic cause to explain the retinal dystrophy with secondary effects on learning and behaviour due to the visual impairment, or other factors, although this would not explain the microcephaly.*
>
> *3. a genetic cause to explain the retinal dystrophy and additional genetic causes to explain other features (these could include additional gene mutations or predisposing genetic factors)*
>
> *4. a genetic cause for the retinal dystrophy and acquired cause(s), (specifically HIE) for the other features.*
>
> *We agreed that since the retinal dystrophy is genetic, the most parsimonious explanation is a genetic cause for all the clinical features."*

102. The neuroradiologists agreed that the MRI scan appearances did not explain the Claimant's apparent microcephaly. They were each asked about the possibility that hypoxic ischaemia could have caused microcephaly. Each expected that, if this were so, one would see evidence of hypoxic ischaemic damage on MRI scanning at 6 days.

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Dr Connolly was asked whether this would still be the case where therapeutic cooling had occurred. He stated that he was not aware of any literature on this issue.

103. In his report at [4.12], Dr Wardle refers to the microcephaly being consistent with a perinatal brain insult but he did not identify the encephalopathy as an insult that could lead to such a consequence. In their joint statement, he and Dr Subhedar deferred to the paediatric neurologists for the cause of the Claimant's microcephaly.

104. The geneticists agree that, whether the Claimant's microcephaly is classified as primary (that is to say evident at birth) or secondary (not evident until later), the microcephaly might be either acquired or genetic in origin. Equally, the paediatricians note in their joint statement that microcephaly can be caused by birth asphyxia but note that it also has a range of other aetiologies, including genetic, environmental and infectious factors.

105. In oral evidence, though not in the earlier joint statement, Dr Rose positively attributed the microcephaly to a period of hypoxia. He accepted that this is not referred to in his breach of duty report where, it can be noted, he lists 8 clinical features of perinatal hypoxia without mentioning microcephaly. In oral evidence he attributed the microcephaly to the presumed period of hypoxia, stating that this opinion was implicitly recorded in his condition and prognosis report where he listed microcephaly as part of the Claimant's condition. However, this is not a connection that is expressly made. Further, when he dealt with this issue in the joint statement, Dr Rose deferred to the neonatologists on the point.

106. Dr Agrawal considers that where, as here, there is no radiological evidence of damage attributable to birth asphyxia and there is separately evidence of progressive rod and cone dystrophy, birth asphyxia is unlikely but genetic or inherited aetiology or more likely, possibly a genetic syndrome that has caused both the microcephaly and the retinal dystrophy. He notes that whole genome sequencing ("WGS") has not identified a genetic syndrome. However, not all genetic abnormalities are picked up by WGS.

107. The neonatologists refer to Chalak 2018[22] in support of the general comment that mild hypoxic ischaemic encephalopathy has been reported to lead to long-term

[22] Prospective Research in Infants with Mild Encephalopathy Identified in the First Six Hours of Life: Neurodevelopmental Outcomes at 18-22 months, Chalak and others, Pediatr Res/ 2018 December

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neurodevelopmental problems in approximately 16% of cases. They defer to paediatric neurologists[23] on the issue. Dr Agrawal acknowledged this report and this incidence of neurodevelopmental delay in those who have suffered HIE, but pointed out that the population from whom that figure was drawn was one who had encephalopathy diagnosed by CFM. That was not the case here.

108. Dealing with the Claimant's developmental delay and associated communication and other issues, the paediatricians were invited in their joint statement to comment on the most likely explanation for the Claimant's problems that they had identified. Dr Rose considers that having regard to the known low Apgar score at five minutes of age, cord blood gases consistent with perinatal hypoxia and a neonatal HIE, the present difficulties are more likely than not caused in whole or in part by perinatal hypoxia, the more severe clinical and neuroradiological effects of which were ameliorated by therapeutic cooling. Given the absence of any genetic explanation of his present difficulties, Dr Rose considers that to be a less likely cause. He accepted that the Claimant's visual impairment might have affected his development but he considered that HIE played a greater part. For example, he had described certain features of the Claimant's use of crayons in his report at [82]. This might be consistent with visual impairment. However he also noted at [83] that the Claimant could pick up crayons individually but could not turn single pages. Dr Rose considered this to be suggestive more of an issue of brain function than a visual impairment.

109. Dr Agrawal considers the Claimant's condition, with language development, communication and behavioural problems in the presence of progressive visual impairment due to retinal dystrophy to suggest that the visual impairment is causing or contributing to the Claimant's problems. He states that vision is essential to the acquisition of language and communication skills, as well as the ability to interact with others effectively because of its effect on a child's ability to observe and interact with others. Further, difficulties consequent upon visual impairment can lead to problems with accessing educational materials which in turn affects academic performance and language development. It can cause feelings of isolation, frustration and anxiety which may lead to behavioural problems and impact on communication

[23] I have referred above to the distinction between Dr Agrawal, instructed by the Defendant, who is a paediatric neurologist and his counterpart instructed by the Claimant, Dr Rose, who is a paediatrician.

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skills. All of these problems lead to a need for a child to adapt which may affect language and behaviour.

110. Dr Agrawal considers that the Claimant's clinical presentation is not typical of acute profound hypoxia. On the other hand, the retinal dystrophy which is probably of genetic origin suggest that an as yet undiagnosed genetic mutation is a more likely explanation of the Claimant's condition – he points out that the geneticists' evidence indicates that the majority of genetic mutations have not as yet been identified through genetic testing, including genome sequencing.

(7) What was the cause of the Claimant's pneumothorax?

111. The neonatologists agree that the Claimant's pneumothorax was caused by resuscitation. As I have indicated above, Dr Wardle indicated that it was not possible to say that the pneumothorax would have been avoided with the lesser resuscitation that would be necessary on his argument that part of the cause of the Claimant's encephalopathy and depressed state at birth was chronic hypoxia due to uterine hyperstimulation.

112. There is no other evidence to support the conclusion that the pneumothorax would have been avoided but for the Defendant's breach of duty.

ISSUE 1 – DID THE CLAIMANT SUFFER HIE?

113. Both neonatologists, together with the paediatrician instructed by the Claimant, Dr Rose, consider that the Claimant suffered HIE. The only expert to doubt this diagnosis is Dr Agrawal.

114. Certain major features of the reasons for the majority conclusion are uncontroversial:

a. The Claimant was born in a depressed state, requiring resuscitation;

b. Those treating the Claimant shortly after birth gave very low Apgar scores and considered he was a suitable candidate for therapeutic cooling;

c. The Claimant was thought to have encephalopathy, as noted in several ward rounds and recorded in the discharge summary

d. The Claimant suffers evidence of developmental delay of a kind which may flow from hypoxic ischaemic encephalopathy.

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115. On the other hand, other aspects of the evidence could be said to be no more than neutral on the issue:

a. For reasons that are identified in respect of issue two below, the evidence does not support the conclusion that the Claimant suffered significant acidosis because of chronic hypoxia.

b. The period of acute hypoxia consequent upon the nuchal cord was far too short to be suggestive of acidosis that would lead to encephalopathy.

c. There is positive evidence in the form of accelerative episodes, at the very least at 15.25 and 16.40, even if the trace at 17.00 is artefactual, to suggest positively that the Claimant was not acidotic at those points;

d. The Claimant's early MRI scan shows no neuroradiological evidence of encephalopathy consequent upon chronic hypoxia;

e. On the other hand, the scan does show evidence that is arguably consistent with the Claimant having suffered perinatal stroke;

f. The Claimant's ultimate condition has features that are not consistent with encephalopathy and are not suggested to be caused by it, in particular microcephaly and retinal dystrophy.

116. As to the possibility that the Claimant suffered a seizure shortly after 23.16 on 16 September 2016, it is neither appropriate nor necessary that I make a finding of fact on that issue for the following reasons:

a. The various medics who have examined the records have been unable to reach a definitive conclusion on the issue. Whilst of course I would be determining the issue on the balance of probabilities, I am less well-placed than they are to reach a conclusion on the medical points.

b. Notwithstanding Dr Wardle's comment in re-examination that this was a matter that the court needed to decide, my understanding of his previous evidence and the position taken by the neonatologists in their joint statement was that, since it was not possible to be confident on whether the Claimant had had a seizure, they would treat him as being on the borderline of mild and

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moderate in the Sarnat Classification. If I now treat him as being either mild or moderate, I am usurping that judgment.

117. The other potential confounding factor in this case is the fact that the Claimant underwent therapeutic cooling. All relevant experts agreed that this had a positive effect both on neurodevelopmental outcome and neuroradiological abnormality. This provides a neat explanation for why the Claimant's outcome is not as severe as one might have expected in clinical terms and why no radiological abnormalities have been identified. But caution is required in placing too much reliance upon this particular issue. As Dr Agrawal pointed out, the evidence of the therapeutic effect of cooling is based on populations that are known to have evidence of encephalopathy on CFM. Abnormalities of CFM are themselves diagnostic of encephalopathy and therefore one can be confident that one is dealing with patients who have suffered encephalopathy when one is looking at the results from the various studies. In contrast, in this case one is looking at an overall picture to try to assess whether the Claimant suffered an encephalopathy. To assume that the evidence of encephalopathy is either weakened (in terms of neurodevelopmental outcome) or missing altogether (as in the case of the neuroradiological evidence) raises a significant risk of presupposing that which one seeks to determine, namely whether there was an encephalopathy. Thus, whilst the therapeutic effect of cooling goes some way to neutralising the effect of the absence of neuroradiological evidence and the relatively mild neurodevelopment outcome, it cannot provide positive support for the hypothesis that the Claimant suffered HIE.

118. Taking this evidence together, the fact that the majority of experts who dealt with the issue favoured the conclusion that the Claimant suffered HIE points in favour of that conclusion being correct. I have considered whether nevertheless Dr Agrawal's opinion of an alternative cause should be favoured. On the balance of probabilities I reject that for the following reasons:

    a. The conclusion of those treating the Claimant in the neonatal period that he was suffering encephalopathy is powerful evidence. They observed him and were well placed to conclude whether his condition was most likely due to encephalopathy.

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b. The Claimant's condition at birth was profoundly depressed in a way that is accepted to be consistent with encephalopathy; on the other hand, his condition was not consistent with the description at [4.16] of Dr Agrawal's report of a baby who is "*relatively healthy.*"

c. There is no suggestion from the neonatologists that the pattern of the Claimant's encephalopathy in the neonatal period was consistent with a perinatal stroke rather than a HIE;

d. As Dr Agrawal accepted, there is no feature of his long term condition suggestive of injury caused by stroke – in particular, there is no right sided impairment of a kind which one might expect were a left hemisphere stroke to have occurred and to have caused any long term damage.

119. I conclude that, whilst the evidence is by no means unequivocal on the issue, it is more likely than not that the Claimant's condition at birth reflected the fact that he was suffering a hypoxic ischaemic encephalopathy

ISSUE 2 - DID UTERINE HYPERSTIMULATION CAUSE OR CONTRIBUTE TO THE HIE?

120. There is no dispute that the CTG trace shows evidence of uterine hyperstimulation. However, I accept that it is unlikely that the Claimant was significantly acidotic because of any hypoxia consequent upon the hyperstimulation. Had there been such acidosis, the venous blood gas pH would have been lower and significantly closer to the arterial pH. Whilst I can accept some discrepancy between the two, even with chronic hypoxia leading to acidosis, the discrepancy here is greater than one would have expected had that acidosis been significant.

121. None of the experts was able adequately to explain this discrepancy. Whilst other features of the Claimant's history, such as the lack of evidence of hypoxic injury on MRI scanning or his long term outcome may be explained by the therapeutic effect of cooling, that is not a feature of his condition that could be explained in that way.

122. It is however here that Mr Irons' theory as to the additive effect of a short period of acute hypoxia on a longer period of chronic hypoxia comes into play. It is common ground amongst the experts that a short period of perhaps just 4 minutes of acute hypoxia due to cord compression due to a nuchal cord is unlikely to have led to a

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baby being born in such poor condition as the Claimant was here. But Mr Irons' explanation of the additive effect of the period of acute hypoxia on a background of chronic hypoxia is a neat theory to explain the facts of this case. As Mr Melton KC put it in closing, the absence of reserve due to hyperstimulation leads to a situation in which the baby cannot withstand the final insult. Indeed, Mr Penny in cross examination accepted that hyperstimulation was a contributor to the ultimate condition of the Claimant at birth, albeit that he appeared to consider it to be the minor contributor.

123. Notwithstanding this concession from Mr Penny, there remains the difficulty in explaining the relatively mild derangement of the venous pH at birth (if it was even outside of the normal range). If one supposes a hypoxic ischaemic encephalopathy (and I have indicated above that on the balance of probabilities, I consider that the Claimant indeed suffer this condition), one would anticipate that it is its severity would be measured not just by outcome but also by the objective measure of pH.

124. Dr Subhedar's evidence that the venous cord pH was barely in the abnormal range was persuasive and I accept that evidence. But that still leaves in play Mr Irons' theory as to the effects of chronic hypoxia and the depletion reserves. On balance, I accept this argument since it explains why the Claimant had such a bad outcome from a short terminal bradycardia. That factor affected both Mr Irons and Mr Penny in their ultimate conclusion that the chronic hypoxia that they suppose occurred was a probable factor in the outcome. I accept that to be so. However, the discrepancy between arterial and venous pH is a strong indicator that it must have been the terminal hypoxia that played the greater part in the ultimate acidosis.

125. It is not possible on the medical evidence before me to distinguish in quantitative terms the extent to which the acidosis was due to chronic hypoxia and the extent to which it was due to acute hypoxia. But for the nuchal cord, it is probable that the chronic hypoxia would have had little if any consequence for the Claimant, given the relatively normal venous pH. On the other hand, but for the chronic hypoxia, it is probable that the acute hypoxia would have had little if any consequence for the Claimant, given the evidence that children regularly recover rapidly from a short period of acute hypoxia due to a nuchal cord. On the evidence before me there is simply no basis to distinguish the relative contributions.

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ISSUE 3 – WAS THE SUPPOSED HIE THE CAUSE OF SOME OR ALL OF THE CLAIMANT'S DEVELOPMENTAL DELAY AND/OR OTHER DIFFICULTIES?

126. In order to determine the contribution of the HIE (if any) to the Claimant's condition, I distinguish between the retinal dystrophy and the microcephaly on the one hand and other aspects of his condition on the other.

127. As I have indicated, it is common ground between the parties that the Claimant retinal dystrophy is not caused by HIE and is probably caused by a genetic abnormality.

128. The evidence on the issue of microcephaly is relatively clear. As I have indicated, Dr Rose deferred to Dr Wardle on this issue and Dr Wardle and Dr Subhedar deferred to the paediatric neurologists on this issue. There is only one expert in this case who is a paediatric neurologist, that is Dr Agrawal. His opinion that this is more likely than not part of the same genetic abnormality that has led to the retinal dystrophy has the attraction of seeking to show a united cause for several aspects of the Claimant's presentation. On the other hand, Dr Rose's opinion in oral evidence that the microcephaly was secondary to perinatal hypoxia is both inconsistent with his deferral to Dr Wardle and is not a view that he had previously expressed.

129. On this issue, I consider Dr Rose's evidence to be ambiguous and unpersuasive. Given that he deferred to Dr Wardle on the issue; that Dr Wardle and Dr Subhedar deferred to a paediatric neurologist, that Dr Agrawal is the only paediatric neurologist in the case and that Dr Agrawal's opinion on the point is clearly expressed, I am satisfied that the more likely explanation for microcephaly is that which he advances, namely some genetic syndrome which simultaneously has caused that as well as the retinal dystrophy. I am not persuaded that the microcephaly is a result of any birth asphyxia and consequently am not persuaded that it is secondary to the HIE.

130. In terms of the Claimant's visual impairment, the written evidence of Professor Ashworth had appeared to point to a conclusion that might be relied upon by the Claimant that he suffered CVI consequent upon HIE which contributed to his current visual impairment. By the end of Professor Ashworth's evidence I was satisfied that this was not a finding that I could make. Her evidence did not support the conclusion that on the balance of probabilities the Claimant suffered some kind of visual processing problem that explained his visual impairment. His visual impairment was

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equally capable of being explained entirely by the retinal dystrophy. In those circumstances, the Claimant cannot satisfy the balance of probabilities on showing any contribution from the HIE that I have found him to have suffered.

131. When dealing with the significance of the Claimant's condition in other respects, I bear in mind what the geneticists had to say in their joint statement about a genetic cause for all clinical features being the "most parsimonious" explanation. I take those words to mean simply the explanation that supposes the fewest causes, the so-called principle of parsimony. The principle, often alternatively expressed as Occam's razor, is doubtless a valuable tool of logic when analysing competing arguments about causation. As I have indicated above, the lack of alternative explanation of the Claimant's microcephaly makes the possibility that he suffers a genetic syndrome a real possibility – certainly, it is a more probable explanation of the microcephaly than the presumed HIE and this may be said to be the conclusion to be favoured by applying the principle of parsimony.

132. However, one must be cautious as to the application of the principle in the particular case. The Claimant has at least one objectively discernible feature of his condition, namely the abnormalities on the MRI scan, which it is not suggested to be caused by the retinal dystrophy. Equally, it has not been suggested that any single unifying genetic cause has led to both the abnormal MRI scan and the retinal dystrophy. One might suppose that some unknown genetic syndrome has caused that condition as well as the retinal dystrophy and the Claimant's other developmental and associated issues. But no potential candidate has been identified for that, nor has anyone explained why that genetic syndrome would lead to that particular abnormality on MRI scanning. If the Claimant has at least two concurrent causes of physiological abnormalities which cannot be explained by a single causative factor, there is a potential danger in assuming that one of them is the pointer to explaining his condition more generally.

133. That said, it is common ground that genetic syndromes can have a variety of consequences including causing developmental delay. No witness has positively asserted that the Claimant's developmental delay could not have been caused by a genetic syndrome that is also the cause of his retinal dystrophy. In considering the various features of his presentation, it is striking that his impaired visual acuity is capable of being a factor in a number of them. In particular, I see force in Dr

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Agrawal's opinion that language impairment, social communication difficulties and impairment of gross motor skills may be caused in whole or in part by the retinal dystrophy.

134. In reaching the conclusion that I have on issues one and two, I have not needed to make findings as to the detail of the Claimant's current presentation since to have done so would have been to suppose an outcome which may then be taken to be supportive of a particular causative mechanism. But at this stage in the evidence it is necessary to stand back and ask what precisely is the nature of the Claimant's condition. In this respect, I am struck by the relatively imprecise nature of the material before me. I have no doubt that each of the experts who have addressed the issue, the paediatricians and the neuropsychologists, have sought to identify as clearly as they can the nature of the impairment. But the picture is as yet unclear. I note the comment of the neuropsychologists that, whilst the Claimant's presentation is consistent with the literature on children with HIE, "*influences are multifactorial (visual impairment, brain injury and environment) and assessment of development over time is important to determine greater clarity."* Furthermore, the evidence suggests that there have been improvements in his ability to communicate, as demonstrated by the difference between Dr Rose's comment in his first report that the Claimant's speech "*is generally indistinct*" and that it was "*difficult to understand his sentences*", with his comment in the joint report that he is speech is "*clear and… intelligible."*

135. The Defendant seeks to persuade me that, taking the evidence together, I can be satisfied that the Claimant's continuing problems have a single unifying cause, namely a genetic syndrome leading to retinal dystrophy, microcephaly and developmental delay which in turn affects skills such as communication. I am not satisfied that there is a sufficiently convincing unifying explanation to come to this conclusion. Were a genetic syndrome to have been identified I might of course have reached a different view, but it has not. I have identified above the potential problem in applying the parsimony principle in a case where, on any version of events, there appears to be more than one discrete pathology at play. Taken together, these factors dissuade me from the conclusion that on the balance of probabilities all of the Claimant's continuing problems are due to that single cause.

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136. I accept Dr Agrawal's evidence that the Claimant's visual impairment due to retinal dystrophy is likely to have had some effect on his development and to be a cause in at least part of the cause of the Claimant's continuing problems,. I found this opinion, summarised at paragraph 109 above, to be well reasoned and persuasive.

137. I have considered whether, having found that the Claimant did suffer HIE, I should conclude that it was more likely than not that the HIE has had long-term effect, causing some of the Claimant's continuing problems. Certainly, the literature referred to shows that HIE can have a long-term effect, although as Mr Holl-Allen KC has pointed out on behalf the Defendant, it would appear that a mild or moderate HIE does not have long-term effect in the significant majority of cases. Equally, it could be that the therapeutic effect of cooling has led to a less severe outcome here than might otherwise have been the case, masking the HIE as a cause of the problems.

138. However, I am not persuaded by the Claimant's case that he suffers the continuing consequences of HIE that cause or contribute to the various aspects of his condition described by the paediatricians and neuropsychologists. I cannot exclude the possibility that they have some contribution. But there is insufficiently clear evidence here for the Claimant to satisfy the burden of proof on this issue. Having accepted the common ground that the Claimant's retinal dystrophy is due to a genetic cause, the evidence of Dr Agrawal that the Claimant's visual impairment due to the retinal dystrophy is a cause of at least some of his developmental problems and the evidence of Dr Agrawal that the microcephaly was not caused by HIE, the remaining features of the Claimant's condition, whilst not inconsistent with his having suffered the long terms effects of HIE, are not sufficiently compelling as to show that on the balance of probabilities that is the case.

CONCLUSION

139. It follows from the above that:

    a. Whilst the Claimant shows that he suffered HIE as a result of the Defendants admitted breach of duty, he cannot show on the balance of probabilities that he has suffered anything other than the immediate depression of his condition at birth as a result of the HIE.

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b. Even in respect of his birth condition, he cannot show that the pneumothorax was a consequence of the breach of duty.

140. I have no doubt that this judgment will be a disappointment for the Claimant's family who have fought hard for him. They can be reassured that the evidence in this case has been comprehensively explored by experts and lawyers on their behalf. Even where there has been admitted negligence and where a person has suffered a bad outcome, it is sometimes not possible to show any connection between the negligence and the bad outcome. That is my conclusion here.

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