SECOND DIVISION, INNER HOUSE, COURT OF SESSION

[2017] CSIH 58
A10/07

Lord Justice Clerk
Lord Brodie
Lord Drummond Young

OPINION OF THE COURT

delivered by LADY DORRIAN, the LORD JUSTICE CLERK

in the reclaiming motion

by

AW, as legal representative of LW

Pursuer and reclaimer

against

GREATER GLASGOW HEALTH BOARD

Defender and respondent

Pursuer and Reclaimer: Clancy QC, Drysdale; Drummond Miller LLP
Defender and Respondent: McLean QC, Dawson; NHS Scotland Central Legal Office

29 August 2017

Introduction

[1] The pursuer's date of birth is 2 December 1965. She is the mother of LW, who was born by caesarean section on 8 October 1996. LW suffers from cerebral palsy, with learning and behavioural difficulties. The case is based on the alleged negligence of midwifery staff who spoke to the pursuer on Tuesday 1 and Thursday 3 October and who attended her on 5 October, all 1996.

[2] Certain issues of fact were in dispute at proof, critically: (i) the circumstances of the home visit by the midwives on 5 October; (ii) the information they were given or imparted; (iii) the nature, extent and results of any examination carried out by them; (iv) the circumstances in which the pursuer was given an unscheduled ultrasound on 8 October; and (v) the nature of any concerns about her presentation on that date. Other disputed issues related, amongst other things, to: the nature and significance of any symptoms reported by the pursuer; the accuracy of a diagnosis of pre-eclampsia made on 8 October, and the significance thereof; the timing, extent and nature of any placental abruption; whether LW suffered a hypoxic insult during the period between the ending of a cardiotocograph (CTG) trace immediately prior to the caesarean section and delivery; the interpretation of radiological evidence; and the nature, extent and distribution of LW's disabilities. Negligence and causation were both disputed.

[3] The Lord Ordinary found that negligence was established in relation to the events of 5 October. He concluded:- (1) that no urine test was performed on that date; (2) that there was no recording of blood pressure on that date; and (3) that the pursuer reported symptoms suggestive of pre-eclampsia on that date but that the midwives failed to record them. They also failed to refer the pursuer to hospital. These failures were negligent.

[4] However, the Lord Ordinary held that the action failed on causation. Two causation issues arose. First, the pursuer maintained that but for the midwives' negligence she would have been admitted to hospital earlier than she was, and that LW would have been delivered prior to the event which was said to be the cause of his cerebral palsy. This we shall refer to as the "first causation issue". The Lord Ordinary held that, although the midwives were negligent in not referring the pursuer to hospital on 5 October, that negligence had no causative effect because a referral on that date, or at any time prior to her

actual attendance at the hospital on 8 October, would not have resulted in an earlier caesarean section.

[5] Secondly, the pursuer maintained that LW's cerebral palsy was caused by "an acute hypoxic ischaemic insult ('AHII') which began several minutes before he was born and which ended when he was about six minutes old." The Lord Ordinary was not satisfied that such a cause had been established. We shall refer to this as the "second causation issue".

[6] The grounds of appeal allege serious errors in the Lord Ordinary's analysis of these points, including that he failed to do justice to the evidence led by the pursuer; failed to give adequate or proper reasons for his decision; misunderstood passages of evidence and overlooked others. It is said that the cumulative effect of the errors and omissions satisfies the test for overturning his conclusions on causation.

[7] A cross-appeal has been lodged on the basis that *esto* the Lord Ordinary erred in his assessment of causation, he had previously erred in his assessment of the factual evidence underlying the pursuer's case of negligence. He had erred in finding the pursuer and her husband to be credible and reliable witnesses. In his assessment of negligence he had failed to consider the expert evidence for the defenders in light of the whole factual evidence. His findings that on 5 October there was no urine test, that the pursuer was suffering from symptoms suggestive of pre-eclampsia and that she should have been referred to hospital, are all challenged. It is said that there was no evidential basis for the persistence of symptoms such as would have required earlier admission to hospital.

[8] As ordered by the court, the reclaiming print, appendix, supplementary appendix, parties' respective submissions in the Outer House and notes of arguments in the Inner House, and the transcript of evidence were reproduced in PDF form and the resultant 15,018 pages provided to the court in digital form on memory sticks. Each of these pages is

numbered with the prefix "MS". We shall use the MS numbers to allow parties to identify particular material which we have relied on in framing our opinion. It is our intention, however, that the opinion should be comprehensible without access to the documents.

[9] A feature of the submissions of both parties has been to present the court with a great quantity of material and many lines of arguments. Parties notionally presented oral submissions but in large part what this consisted of was drawing the attention of the court to the detail of the extensive written material and a reiteration of lengthy submissions made to the Lord Ordinary. In this regard the defender's material is particularly lengthy, diffuse and, to use an adjective to which we shall return, rhetorical. Parties, in particular the defender, would appear to have found it impossible to frame their submissions in a way that was both comprehensive and reasonably succinct, or indeed contained in one document. As a result it has been impractical to provide a reasoned response to each point made by each party. We have accordingly confined our detailed consideration to the more salient points and in particular those critical to the resolution of the issues. We have not attempted to summarise the submissions, which should generally become clear from the context of what follows.

[10] Parties should understand that we do not consider their respective arguments to have been taken forward by the various rhetorical flourishes to be found in the submissions and notes of argument. Simply to commend or depreciate a witness by adding an epithet such as "careful" or "unimpressive" is frankly a waste of everyone's time unless accompanied by the reasons why this is said to be so. Similarly, we have gained limited assistance from what are presented as summaries of evidence in the written submissions where they are not accompanied by reference to specific passages in the transcript as identified by MS numbers. What would have been of assistance to the court, namely a joint

glossary of relevant medical terms, was not provided. The opinion which follows is a joint opinion which has been substantially contributed to by all members of the court. For convenience we refer throughout to the "pursuer" and "defender", following the course adopted by counsel in the submissions; and for similar reasons we attribute those submissions to the parties themselves.

Background

[11] The pursuer's first pregnancy was confirmed in April 1996 with an estimated date of delivery of 2 December that year. She was seen at the booking clinic on 21 May at 12+ weeks gestation with no past medical history of significance. Weight, blood pressure and urine tests were all normal. Blood tests showed that the pregnancy was low risk for the conditions of Spina Bifida or Down's Syndrome. Weight, blood pressure and urine were generally tested at regular intervals until 13 September with normal results being reported.

[12] On 20 August at 25+ weeks the pursuer discussed the option of a home birth and opted for a "domino" delivery. "Domino" stands for domiciliary in and out, involving a plan of antenatal care conducted by midwives followed by a brief stay of around 4 hours in hospital at the time of delivery. On 13 September the pursuer's blood pressure was normal, there was no oedema, the urine test was negative and foetal movements were felt. She was reviewed at home on 19 September when again blood pressure was normal, no abnormality was detected in the urine, fundal height was recorded as equal to the gestation and the foetal heart was ticked as having been heard. The abdomen was described as soft. This history is not disputed.

[13] On Saturday 5 October, midwives Isobel Giles and Evelyn Mohammed attended the pursuer's home to review the pursuer, after attending an overnight delivery. What

happened at that review was a matter of dispute. The pursuer stated that she told the midwives that she was feeling unwell; was exhausted and tired; was hot and puffy in her face; had blurring at the edges of her eyes; had been suffering from headaches; had felt reduced movements; and was concerned that her stomach had not increased in size since the previous check-up. She had been so unwell attending a funeral that she had had to lie down. She was tearful. She said that the midwives were dismissive about her symptoms, and told her that a woman's eyesight can change during pregnancy and then revert to normal, and that all babies behaved differently. The midwives examined her and listened to the foetal heart rate for about 15-30 seconds using a Pinard stethoscope. They told her the heart rate was "fine". They took a blood sample but did not test her blood pressure.

[14] The midwives had no real recollection of the events of 5 October. Their evidence that blood, urine, fundal height and foetal heart were all tested and found to be normal essentially relied on usual practice, and to some extent the content of the notes. The notes referred to consisted of a pregnancy care plan ("PCP") provided to the pursuer. The notes indicate that fundal height was thought to be appropriate suggesting that the abdomen was palpated. Under the heading "urine" is an entry reading "CC", said to indicate a reading clear for both sugar and protein. Mrs Giles said "I would not record anything like that if I had not tested the urine". The PCP did not contain a recording for blood pressure. This was a departure from normal practice, notwithstanding that Mrs Giles asserted that she would definitely have taken the blood pressure. In the pleadings, it is specifically denied that the pursuer complained of headaches, blurred vision, a puffy face, exhaustion or reduced foetal movements, with an averment that had she done so the same would have been documented and there would have been no question of omitting to check the blood pressure. This reflected Mrs Giles' final position in evidence.

[15] The pursuer said that she continued to suffer symptoms through 6 and 7 October and that at about midnight on 7 October she experienced a sharp abdominal stabbing pain for a minute or two. She was due to attend an antenatal class the next morning, where a physiotherapist taking the second half of the class, Dot Sorley, informed of her concerns, arranged for an ultrasound scan to be carried out. The pursuer had not mentioned her concerns to the midwife who had conducted the first part of the class as she did not wish to make a fuss. She had, however, formed the impression of Dot Sorley as a sympathetic individual. By the time of proof in the action Dot Sorley had since died, so no light could be shone on the precise circumstances in which the pursuer's scan was arranged. What is clear is that an unscheduled scan was arranged.

[16] The scan was carried out between 1300 and 1311 hours by a consultant obstetrician, Dr Crichton (then Macara), who expected a routine scan which would reassure the patient. In fact she noted from the scan the following: the baby's weight (estimated at 8-900 gms) was below the fifth centile for gestational age of 32 weeks; reduced liquor volume; and reduced "Doppler" readings. There was evidence of the possible presence of a retro-placental clot. Dr Crichton recorded a biophysical score ("BPS") of 0/8. By recording a BPS of 0/8 Dr Crichton was indicating that, on ultrasound examination over some 20 minutes, she could not detect the foetus exhibiting sustained movement, breathing or tone. "Doppler readings" or "Dopplers" are shorthand expressions referring to umbilical artery Doppler assessment, which is what Dr Crichton carried out. Such an assessment is done by ultrasound scanning and is a technique routinely used in the surveillance of foetal well-being in the third trimester of pregnancy. It allows the calculation of a resistive index which is a measure of the impedance in what in LW's case was a single umbilical artery to the flow of blood from the foetus to the placenta (usually a foetus will have two umbilical arteries

and one umbilical vein but LW had only one artery and one vein). The mechanism of the flow of deoxygenated blood from the foetus to the placenta through the umbilical arteries and the consequences of that being impeded were explained by Professor Draycott in the context of his evidence that the reason that LW was so small at delivery was that his intrauterine growth had been restricted by reason of poor placental function (MS 2566 to 2573). Dr Crichton measured LW's Umbilical Artery Resistive Index ("UARI") at 0.9 as opposed to the expected 0.6 or 0.7 for a foetus at 32 weeks gestation (MS 1683). Professor Draycott described that measurement as "quite a reasonably high resistance" (MS 2569). It approaches a UARI of 1.0 which describes the situation where in the diastolic phase of the cardiac cycle (that is when the foetal heart muscle is relaxed and therefore not pushing blood towards the placenta) there is no forward flow. That may be referred to as absent end diastolic flow; the situation where, as Professor Draycott put it, "there's no flow at the end of diastolic, the bit where the heart stops contracting" (MS 2570). Notwithstanding the measurement of UARI at 0.9 which would indicate some blood flow in the diastolic phase, albeit in the face of high resistance, Professor Draycott drew attention to the fact that the ultrasound scan had detected intermittent absent end diastolic flow, in other words instances where there was no blood flow to the placenta, meriting a UARI of 1.0. The professor attributed the Doppler readings recorded by Dr Crichton to a narrowing of LW's umbilical artery. He associated this with a reduced delivery of nutrients and oxygen to the foetus resulting in LW's small size (MS 2571). In Professor Murphy's opinion, the fact that LW had a single umbilical artery as opposed to the usual two, additionally compromised the effective perfusion of oxygen (MS 11395).

[17] Dr Crichton advised immediate admission with cardiotocograph ("CTG") monitoring of the foetal heart and prescribed a cortical steroid to accelerate foetal lung

maturity. Dr Crichton was extremely concerned about the health of the foetus, and thought that delivery should follow within 48 hours. The pursuer was admitted to hospital and her blood pressure recorded as 174/118, causing the midwife, Mrs Ovens, to ask for urgent attendance of the registrar. Mrs Ovens said blood pressure was high and this would have given her concerns about pre-eclampsia. She recorded "asymptomatic" in the notes, meaning that the pursuer had not reported symptoms of headache, blurred vision or abdominal pain. The duty registrar, Dr McLelland, examined the pursuer. Her note records the results of the ultrasound scan as: "IUGR + oligohydramnios - BPP 0/8 - ?RPC …" ("IUGR" refers to intra-uterine growth restriction, "BPP" refers to biophysical profile (otherwise BPS), "RPC" refers to a retroplacental clot). Dr McLelland further recorded that the pursuer was complaining of "…2 episodes of diarrhoea + irregular u/a today – no pv bleeding". She then recorded that on examination the pursuer was "well anxious". There was protein 1+ on testing of the urine.

[18] Dr McLelland's impression was of IUGR and placental abruption. She considered the results of a CTG trace to be "worrying", showing "low to absent variability" with the possibility of decelerations. She transferred the pursuer to the labour suite where she was admitted at 1445. The CTG trace, discontinued during transfer, was recommenced on arrival. The Lord Ordinary notes that Dr McLelland appears to have proceeded on the basis that the pursuer was presenting with pre-eclampsia and she considered that delivery should follow as soon as the pursuer's blood pressure was stabilised.

[19] At 1510 the pursuer was assessed by a consultant obstetrician, Dr Hanretty, who diagnosed IUGR and pre-eclampsia and concluded that the pursuer required immediate delivery. His notes record "IUGR and pre-eclampsia. Foetal assessment concerning." His conclusion was stated thus: "I think delivery is indicated in both the foetal and maternal

interest." Delivery was planned to follow when blood pressure was stable. By 1547 blood pressure was stabilising but the trace remained concerning.

[20] There was a period of about 13 minutes prior to delivery during which there was no CTG measurement and therefore no record of foetal heartbeat. This is what parties have described as "the dark period". The 13-minute duration of the "dark period" is derived from a timing by the midwife, Mrs Mailer (née Main), of 1639 for the end of the CTG and the timing of delivery at 1652. It is to be assumed that all those involved in recording timings will have been endeavouring to be accurate but, timepieces were not synchronised and there is scope for individuals attributing different timings for a particular event. Thus the "dark period" may have been less than 13 minutes. For example, Dr McLean thought that the CTG ran until 1644 (MS 5429) whilst Dr Thorburn's entry in the anaesthetic record indicated that LW was delivered at 1650 (MS 5367).

[21] LW's condition on delivery was assessed as being poor, cyanosed with no respiratory effort and marked bradycardia. His heart rate was less than 60 bpm (Dr Gallagher's note – MS 5495 to 5497, 10464). Apgar scores were 3 at 1 minute, 4 at 5 minutes and 7 at 10 minutes. (An Apgar score is an indication of the physical condition of a newly born baby, obtained by measuring five individual features: appearance (skin colour); pulse rate; grimace (reflex irritability); activity; and respiratory effort, and marking each from 0-2: the maximum score is thus 10). Weight was 920g and there was evidence of a retroplacental clot assessed to take up around 25% of the area of the placenta. He was intubated and given positive pressure ventilation at one minute. A solution of adrenaline was given by endotracheal tube at two minutes and a further dose was given very shortly afterwards. The heart rate increased to 80 bpm at 5 minutes and 100bpm at 10 minutes. LW's condition at birth and its significance is discussed by Dr Coutts at MS 5504 to 5521.

There he rejects the analogy between LW's condition and a stillbirth and notes the distinction between a newborn appearing blue or cyanosed and appearing white (MS 5497). If a newborn is cyanosed that indicates that it still has blood flowing to its extremities, whereas if the newborn is white there has been a shut-down of circulation to the skin. He explains that it can take longer than six minutes to resuscitate a newborn (MS 5520).

[22] LW was transferred to the neonatal unit. He was hypertensive with asymptomatic hypoglycaemia, and suffered several episodes of oxygen desaturation. He suffered respiratory difficulties, requiring intermittent ventilation. He suffered from other health difficulties. The pursuer avers that there was neonatal encephalopathy, (namely, the signs of abnormal neurological function in a neonate, such as is commonly caused by birth asphyxia), which it is said can go unnoticed in premature babies. The Lord Ordinary notes that neither of the treating physicians considered there to be encephalopathy, and the defender maintains that there was not.

[23] The advice on eventual discharge was that LW should be treated as a normal baby and that there were no indications for long term health problems. However, problems did develop over time, with LW failing to meet developmental milestones, and failing to thrive. It is not disputed that LW suffered from IUGR nor that he suffers from cerebral palsy, and that he has significant learning and behavioural difficulties. The pursuer maintained that the growth retardation suffered by LW was asymmetrical, with catch–up growth (in other words disproportionate between the head and the body, but with the disproportionality evening out within about two years). The defender maintained that the retardation was symmetrical. This has some significance in relation to the possible cause of LW's disabilities. The interpretation of ultrasound imaging taken on 9, 14 and 23 October and 8 November

1996, the detail of which is referred to below, was central to the case. As will be seen, the parties were in dispute about the proper interpretation of this evidence.

[24] The pursuer maintained that on 8 October, when she was being admitted to the hospital Mrs Giles removed the PCP from her. Despite the fact that a box thereon records "no complaints" in relation to midwifery care, the pursuer said she had not been asked about this. Mrs Giles had no recollection of meeting the pursuer on 8 October. The Lord Ordinary noted that Mrs Giles had made an entry in the PCP after delivery in which she recorded "EMSCS for IUGR and reduced liquor baby ventilated in paediatric department". The Lord Ordinary notes that the entry is "to some extent, erroneous in that the reason for the Caesarean section was not IUGR but pre-eclampsia" (paragraph 24).

[25] Whilst accepting that Dr Hanretty made a diagnosis of pre-eclampsia the defender maintains that this was only a working diagnosis made on the basis of hypertension and proteinuria +, not on the basis of any reported symptoms, and that the pursuer did not in fact have pre-eclampsia. The level of proteinuria was minimal. The blood indices were normal making it likely that the hypertension was in consequence of an extending small abruption on that day rather than the precipitating cause of the abruption. The consequence it maintains, is that neither blood pressure nor urine testing would have been likely to have shown any abnormality on 5 October.

[26] The defender maintains that the likely indication for an ultrasound on 8 October was lack of foetal growth rather than either reduced foetal movement or maternal symptoms. It is likely that there had been long standing abnormality of the placenta further compromised during the course of the pregnancy by ongoing, multiple small abruptions. Even if proteinuric hypertension had been detected on 5 October and the pursuer referred to hospital for assessment, a scan would not have been likely to have been performed before

Monday 7 October. The BPS score would probably have been normal and steroids would have been prescribed to mature the foetal lungs prior to delivery which is unlikely to have occurred materially sooner. These issues are addressed in our treatment of the first causation issue.

[27] The defender avers that the basal ganglia damage (as demonstrated by imaging of LW's brain) cannot be attributed to an acute hypoxic event occurring just prior to delivery and that at least some of the changes are compatible with events over a period of weeks in a severely growth retarded infant. Features such as the symmetrical nature of the growth retardation, soft dysmorphic features and what the defender maintains is a diplegic rather than quadriplegic presentation, all suggest a prenatal cause. The defender also avers that LW was not in circulatory collapse *in utero* immediately prior to delivery. Circulatory collapse in the context of significant hypoxia *in utero* causing bradycardia followed by hypertension leads to generalised poor tissue perfusion and acidosis, none of which were present. Any hypoxic insult is likely to have occurred after delivery, as the cord was cut and the baby attempted to ventilate independently, and to have continued through resuscitation. Such an insult would have occurred in any event, given LW's condition, even had a caesarean section been carried out earlier following a hospital admission on 5 October. These issues are all addressed at length in our treatment of the second causation issue.

Negligence

The pursuer's case

[28] The pursuer's case on negligence substantially relates to the midwifery staff who attended her on 5 October, and who she maintained had a duty to respond to the symptoms of which she advised, which were indicative of late onset pre-eclampsia, a condition which

can lead to complications for both mother and baby. It is a matter of agreement that the signs of pre-eclampsia include protein in the urine (proteinuria) and hypertension. The symptoms may include headache and visual problems, and the signs usually precede the symptoms. The pursuer maintains that the symptoms which she reported should have led to a more detailed assessment. Given her symptoms, the midwives had a duty to test blood pressure and urine. It is likely that on 5 October testing would have revealed both elevated blood pressure and proteinuria, requiring a referral to hospital. Had these results been repeated at the hospital a scan would have followed, showing a severely growth retarded baby and leading to delivery prior to the placental abruption said to have occurred on 8 October. Had there been no proteinuria the pursuer would have been reviewed on 6 October when both hypertension and proteinuria would have been likely to be present, leading to a scan and earlier delivery of the child.

[29] After admission to hospital, there would by 7 October have been an abnormal scan. CTG on admission and thereafter would have shown by 7 October at the latest intermittent decelerations consistent with cord compression secondary to reduced liquor volume. The pursuer would have been in hospital when she suffered the sharp pain in her abdomen on 7 October. Further assessment would have resulted in immediate delivery.

[30] Had the pursuer been admitted on 5 October her management would have been optimised, such that LW would have been born by caesarean section on the evening of 7 October or at least later that night, and by 1000 on 8 October at the latest.

[31] It is averred that it is well recognised that reduction or cessation in foetal movements may precede death by a few days or more, that such movements are useful in supplementing other tests of wellbeing and should be taken seriously. The pursuer's concern about the growth of the foetus should have resulted in referral for CTG which was

merited in any event given the signs of reduced foetal movement. Such CTG tracing would have been abnormal by 7 October at the latest, leading to delivery by the morning of 8 October at the latest.

The Lord Ordinary's conclusions

[32] The Lord Ordinary concluded that on 5 October no urine test was done by the midwives, and that they failed to take the pursuer's blood pressure. Moreover he found that the pursuer reported to the midwives symptoms "suggestive of pre-eclampsia" which they failed to record or act upon. He concluded that these failings were negligent, and that the pursuer should have been referred to hospital on 5 October.

[33] He went on to find, however, that the pursuer had not established that had she been referred to hospital on that date she would have been admitted. He accepted evidence that the pursuer's blood pressure would then have been likely to be normal, as would a urine test. He rejected the evidence of Professor de Swiet, an expert called for the pursuer, who had, simply put, suggested that the results of blood pressure and urine tests on 5 October could be predicted by working back from the results found on 8 October. The Lord Ordinary concluded that Professor de Swiet's reasoning, however elegant, was entirely theoretical. As to the issue of foetal symptoms, he concluded that, on referral, the pursuer would have been assessed by a midwife who would have measured fundal height. On any view the likelihood was that they would have considered the result reassuring, so on this basis also the pursuer failed to establish that she would have been admitted. She had not established that she would have been admitted before her actual attendance and admission on 8 October. The pursuer therefore could not prove a causative link between the negligence and the injuries sustained.

[34] Although he considered that there was thus no requirement for him to consider the second causation issue the Lord Ordinary went on to do so. In paragraph 119 of his opinion, the Lord Ordinary noted that there was:

> "no dispute that LW had sustained an insult to his brain which was causative of his deficits and which had first been clinically determined by MRI scan performed by Dr Zuberi in September 2002. The issue was not whether the insult to the brain was causative of LW's deficits but whether the cause of the brain insult was an event or events for which the defenders were responsible as a result of negligence on the part of their employees, the midwives."

He repeated this at paragraph 134:

> "The pursuer's case is critically dependent on the cause of LW's disability being a single event, that being an acute ischaemic hypoxic insult commencing at some time immediately prior to delivery and ending on his resuscitation 6 minutes after delivery. The occurrence of such an event is not in doubt, albeit its timing is."

[35] What exactly is meant by these passages of the Lord Ordinary's opinion is somewhat obscure. There was no doubt that LW suffered at birth a period of hypoxia from which he required to be resuscitated. However, the question whether he had sustained an AHII as maintained for the pursuer was very much in dispute. Again, we will address this more fully in due course.

[36] After briefly summarising some of the evidence and submissions relating to the second causation issue, the Lord Ordinary stated his conclusions in paragraphs 134-136:

> "[134] It appears to me that criticisms of the timing made by the defenders have some force. I do not consider that it is established on the basis of the evidence exactly how long the event occurred. So far as the MRI scan and an interpretation thereof is concerned with the exception of the timing of the acute hypoxic event, I am satisfied that I can rely on the evidence of Drs Kendall and Connelly. Both were highly and relevantly experienced. Both gave their evidence clearly and precisely. In my view neither overstated the implications of their evidence, nor importantly did either suggest that their evidence on its own was conclusive of a diagnosis of cerebral palsy caused by any particular factor. Both Dr Kendall and Dr Connelly were clear that the causation of cerebral palsy depended upon teamwork from a number of medical disciplines. When that consideration is taken into account I find it difficult to accept the pursuer's proposition that the single insult theory must be accepted in this case.

> [135] In my view there is a very considerable body of expert opinion, most notably that of Professors Walker and Murphy, that cogently and persuasively suggests the admitted features of severe intra-uterine growth restriction and placental insufficiency cannot be ignored for their potential causative effect so far as LW's condition is concerned. It is significant in my view that the treating clinicians in this case all recognised and accepted that these features were likely to have played a part in the development of LW's deficits. Whilst I recognise that there are other features in LW's case, his prematurity and his difficult neo-natal history that cannot be ignored it appears to me that the majority of the medical opinion I heard was to the effect that intra-uterine growth restriction and placental insufficiency were the most significant features in LW's clinical history.
>
> [136] Having regard to that consideration the present case appears to me to raise issues similar to that faced by the court in *Honisz (supra)*. There is a body of clinical opinion presented by the pursuer which supports a single insult theory as establishing causation for LW's disabilities. Against that there is another body of expert opinion which does not accept that theory and presents an alternative hypothesis to the effect that as a result of intra-uterine growth restriction and placental insufficiency LW's deficits were in existence before 8 October 1996 and would have caused his condition regardless of the date of his delivery. I have no objective reason to reject that second body of evidence. In these circumstances I cannot be satisfied that the pursuer has established her case."

[37] Finally, the Lord Ordinary dealt with what he described as "some incidental matters." He stated that although the defender made detailed criticisms of the pursuer's experts, in the event the issue of conflict between the experts did not figure prominently in the formulation of his views. The issue of difference of opinion was only relevant in respect of the midwifery issues and then only to a minor extent and in the obstetric evidence of Professor de Swiet rebutted by Professors Walker and Murphy. So far as the midwifery issue was concerned, the finding on negligence turned on matters of fact. In relation to the obstetric issue he preferred the evidence of Professors Walker and Murphy. The Lord Ordinary considered that he did not need to address the detailed criticism made (by the defender) of expert witnesses for the pursuer, but in any event he found no reason to criticise any expert in the case.

The approach of an appellate court

General

[38] The present reclaiming motion raises at the outset the extent of the powers that an appellate court has to interfere with decisions at first instance. That issue has been the subject of considerable case law, including the recent decisions in *McGraddie* v *McGraddie,* 2014 SC (UKSC) 12; and *Henderson* v *Foxworth Investments Ltd*, 2014 SC (UKSC) 203; and we were addressed at some length by the parties on what they submitted was the present state of the law.

[39] At the outset, it is useful to consider the categories of first instance decision that may come before an appellate court following a proof before answer. Such decisions may be said to fall into four general categories. The first consists of decisions as to credibility, reliability and the primary facts: what the persons involved actually did or said, or what happened to them. The second consists of inferences of fact drawn from the primary facts. The third comprises the application of the law to the facts, both primary facts and inferences; such questions are frequently referred to as questions of mixed law and fact. What is involved, however, is the application of a legal norm to facts that have been found by the court. The fourth category consists of decisions on pure questions of law, that is to say, the general rules of law in the abstract. We do not suggest that those four categories cover every sort of decision that a judge at first instance may make at a proof. Nor do we suggest that they are mutually exclusive; in practice the categories of decision shade into one another. Nevertheless, we consider that those categories provide a useful framework for analysing what first instance judges do at a proof and what an appeal court may do when it is invited to reverse or interfere with a first instance decision made at proof.

[40] The issues in the reclaiming motion and cross-appeal, at a general level, relate to

three principal matters. The first of these is a challenge to the Lord Ordinary's findings on causation as to the consequences of the negligence that he had found on the part of the two midwives, Mrs Giles and Mrs Mohammed, when they saw the pursuer on Saturday, 5 October 1996. The second issue is a challenge to the Lord Ordinary's findings on the causation, at a general level, of LW's injuries whether those injuries were caused by a condition that could have been successfully treated had proper medical and midwifery advice been obtained immediately before and at the time of his birth. The third, which arises in the cross-appeal, is a challenge to the Lord Ordinary's findings in relation to the negligence of the two midwives. In respect of those three issues, it is the first and second of the categories described in the last paragraph that are primarily relevant: the primary facts and inferences drawn from those primary facts. To some extent the application of legal norms to the facts is relevant, in respect of both causation and want of a duty of care, but the issues are primarily factual in nature.

[41] On issues of primary fact, the traditional approach of the Scottish courts is found in the well-known statement by Lord Thankerton in *Thomas* v *Thomas*, 1947 SC (HL) 45, at 54; 1948 SLT 2:

> "(1) Where a question of fact has been tried by a Judge without a jury, and there is no question of misdirection of himself by the Judge, an appellate Court which is disposed to come to a different conclusion on the printed evidence should not do so unless it is satisfied that any advantage enjoyed by the trial Judge by reason of having seen and heard the witnesses could not be sufficient to explain or justify the trial Judge's conclusion. (2) The appellate Court may take the view that, without having seen or heard the witnesses, it is not in a position to come to any satisfactory conclusion on the printed evidence. (3) The appellate Court, either because the reasons given by the trial Judge are not satisfactory, or because it unmistakably so appears from the evidence, may be satisfied that he has not taken proper advantage of his having seen and heard the witnesses, and the matter will then become at large for the appellate Court. It is obvious that the value and importance of having seen and heard the witnesses will vary according to the class of case, and, it may be, the individual case in question".

In the same case, Lord Macmillan referred, at p 59, to the advantage that the trial judge has in reaching a conclusion that is not available to an appellate court, in particular in observing the demeanour of the witnesses, their candour or their partisanship, and "all the incidental elements so difficult to describe which make up the atmosphere of an actual trial". He continued

> "When this is so … then the decision of the trial Judge… becomes of paramount importance and ought not to be disturbed. This is not an abrogation of the powers of a Court of appeal on questions of fact. The judgment of the trial Judge on the facts may be demonstrated on the printed evidence to be affected by material inconsistencies and inaccuracies, or he may be shown to have failed to appreciate the weight or bearing of circumstances admitted or proved, or otherwise to have gone plainly wrong".

[42] Viscount Simon dissented from the majority, but the test that he applied was similar to that of Lord Thankerton (pp 47-48):

> "If there is no evidence to support a particular conclusion (and this is really a question of law), the appellate Court will not hesitate so to decide. But if the evidence as a whole can reasonably be regarded as justifying the conclusion arrived at at the trial, and especially if that conclusion has been arrived at on conflicting testimony by a tribunal which saw and heard the witnesses, the appellate Court will bear in mind that it has not enjoyed this opportunity and that the view of the trial Judge as to where credibility lies is entitled to great weight. This is not to say that the Judge of first instance can be treated as infallible in determining which side is telling the truth, or is refraining from exaggeration. Like other tribunals, he may go wrong on a question of fact, but it is a cogent circumstance that a Judge of first instance, when estimating the value of verbal testimony, has the advantage (which is denied to Courts of appeal) of having the witnesses before him and observing the manner in which their evidence is given".

The approach of the court in *Thomas* is similar to that adopted in the earlier case of *Clarke* v *Edinburgh and District Tramways Co Ltd*, 1919 SC (HL) 35; 1919 1 SLT 247, and it was followed by the House of Lords in *Thomson* v *Kvaerner Govan Ltd*, [2003] UKHL 45; 2004 SC (HL) 1; 2004 SLT 24. In both of those cases the criterion adopted for interference with the decision of the Lord Ordinary is that the appellate court should be satisfied that the Lord Ordinary's opinion is "plainly wrong": see, for example, *Thomson* at paragraph [18]-[19]. The

importance to be attached to the views of the judge of first instance was emphasised in *McGraddie* at paragraph 28 (see paragraph [77] below). It was also highlighted by Lord Hope in *Thomson* at paragraph 20:

> "… the fact that reliability, not credibility, was the issue does not mean that an appellate court is in as good a position to resolve it as the trial judge. This is because there are various ways of testing a witness's reliability. One way is to see how his account fits in with the other evidence. If that were to be regarded as the only test, it would no doubt be capable of being performed equally well by an appellate court as by the judge who is sitting at first instance. But another way is to examine the witness's demeanour in all its various aspect when he is giving his evidence. If his version of the facts is in conflict with that which is given by another witness whose honesty is not in doubt, the demeanour of that other witness too will also be relevant. … An appellate court should be slow to interfere with the decision based on a view of the reliability of witnesses of whom the Lord Ordinary was able to make a personal assessment."

[43] We should draw attention to two features that are present in all of the speeches in *Thomas*. First, no attempt is made to impose dogmatic or hard-and-fast rules; general guidance is given, rather than a rigid methodology. Secondly, the approach taken by the House of Lords is firmly based on the advantage that a first instance judge has in deciding questions of fact: he or she has actually seen and heard the witnesses. That is why the approach taken is flexible and pragmatic. We would also add that there are cases where an appellate court has interfered with findings of primary fact made by a first instance judge. One example is *Yuill* v *Yuill*, [1945] P 15, where the issue was whether the defendant and the co-respondent had committed adultery on a particular occasion. The view of the judge of first instance as to the credibility and reliability of the witnesses concerned was reversed on appeal. The court in that case followed the earlier decision of the House of Lords in *Hvalfangerselskapet Polaris A/S* v *Unilever Ltd*, (1933) 46 Ll L Rep 29, a case involving a shipbuilding contract, where the view of the judge of first instance as to the credibility and reliability of crucial witnesses was disapproved on appeal.

[44] The issue in *Thomas* v *Thomas* was whether the defender had been guilty of cruelty towards the pursuer such as to warrant decree of divorce, a matter that turned on the primary facts of the case. The ability of an appellate court to interfere with the decision at first instance is greater, however, when the challenge on appeal is to an inference drawn by the Lord Ordinary. This was considered in *Benmax* v *Austin Motor Co Ltd*, [1955] AC 370, where Lord Reid (at pages 375-376) quoted from the speech of Lord Thankerton in *Thomas* and continued

> "*Thomas* v *Thomas* was a consistorial case based on cruelty, and I think that the whole passage which I have quoted refers to cases where the credibility or reliability of one or more witnesses has been in dispute, and where a decision on these matters has led the trial judge to come to his decision on the case as a whole.… But in cases where there is no question of the credibility or reliability of any witness, and in cases where the point in dispute is the proper inference to be drawn from proved facts, an appeal court is generally in as good a position to evaluate the evidence as the trial judge, and ought not to shrink from that task, though it ought, of course, to give weight to his opinion".

He noted that the hearing upon appeal is a rehearing, and there was no presumption that the judgment in the court below was right. That is a distinction drawn between questions of primary fact, where the general rule is one of deference to the judge who heard the proof, and inferences, where an appellate court may be more ready to interfere with the judge's findings. We would add two further points: first, in our opinion the reasoning involved in drawing inferences can be reviewed by an appellate court with greater confidence than findings of primary fact; and secondly, the intellectual process involved in drawing an inference of fact is liable to be less influenced by the judge's immediate perceptions of the witnesses.

[45] A similar approach to that set out by Lord Reid in *Benmax* v *Austin Motor Co Ltd* has been adopted in other cases. In *Powell and Wife* v *Streatham Manor Nursing Home*, [1935] AC 243, a case on the negligence of a nursing sister, the critical question turned on the trial

judge's assessment of the credibility and reliability of witnesses of primary fact. The House of Lords, reversing the Court of Appeal, held that there was no warrant for interfering with the conclusions of the trial judge. Nevertheless, Lord Wright (at pages 266-267) stated

> "I think that it is difficult, if not impossible, to seek to lay down any precise rule to solve the problem with which faces the Court of Appeal when it has to act as a judge of fact on the rehearing, but finds itself 'in a permanent position of disadvantage as against the trial judge.' In truth, it is not desirable, in my opinion, to do more than state… principles which will guide the appellate Court in the majority of such cases. The problem in truth only arises in cases where the judge has found crucial facts on his impression of the witnesses: many, perhaps most cases, turn on inferences from facts which are not in doubt, or documents: in all such cases the appellate Court is in as good a position to decide as the trial judge".

That case was followed shortly afterwards in *Flower* v *Ebbw Vale Steel, Iron and Coal Co Ltd*, [1936] AC 206, where on the question of whether contributory negligence had occurred the House of Lords reversed the decision of the Court of Appeal and restored the decision of the trial judge. Lord Wright once again (at page 220) emphasised that an appeal is a rehearing, so that the appellate court is bound to exercise a judgment of its own. Where the character of witnesses was an essential element in the decision the appellate court should only set aside the decision of the trial judge in the "clearest cases". The case under consideration, however, did not turn on the credibility of witnesses but on a question of the sufficiency of that evidence to establish what the respondents had to prove: the existence of contributory negligence. That is a clear indication that, in the application of a legal test (the third of the categories discussed above at paragraph 2), an appellate court is not constrained from interfering with the decision at first instance.

[46] As we have noted, the powers that an appellate court has to interfere with the decision at first instance have been the subject of fairly recent comment in the UK Supreme Court, in *McGraddie* v *McGraddie, supra*, and *Henderson* v *Foxworth Investments Ltd*, *supra*. In the latter case Lord Reed, delivering an opinion in which the other judges concurred,

referred to *Thomas* v *Thomas* and other cases (at paragraphs [42] et seq), and in particular to the statement by Lord Macmillan in *Thomas* (at page 59) that "an appellate court can interfere where it is satisfied that the trial judge has gone 'plainly wrong'". He continued (at paragraph 62):

> "[T]here may be some value in considering the meaning of that phrase. There is a risk that it may be misunderstood. The adverb 'plainly' does not refer to the degree of confidence felt by the appellate court that it would not have reached the same conclusion as the trial judge. It does not matter, with whatever degree of certainty, that the appellate court considers that it would have reached a different conclusion. What matters is whether the decision under appeal is one that no reasonable judge could have reached".

He subsequently remarked (at paragraph 67):

> "It follows that, in the absence of some other identifiable error, such as (without attempting an exhaustive account) a material error of law, or the making of a critical finding of fact which has no basis in the evidence, or a demonstrable misunderstanding of relevant evidence, or a demonstrable failure to consider relevant evidence, an appellate court will interfere with the findings of fact made by a trial judge only if it is satisfied that his decision cannot reasonably be explained or justified".

[47] The foregoing remarks were founded upon by the defender in the present case in support of an argument that the Lord Ordinary's decision could only be disturbed if this Court were satisfied that he had come to a decision that was "plainly wrong" in the sense that it was a decision that no reasonable judge could have reached. It was only if that threshold were crossed that the Court could embark on the task of reviewing all of the evidence and effectively redecide the case.

[48] In our opinion the defender's argument on this matter involves a misunderstanding of the principles laid down in *Thomas* v *Thomas* and other cases, which we understand to have been reaffirmed in *Henderson* v *Foxworth Investments Ltd*. We consider that the correct approach, following the principles laid down in those cases, is as follows. First, as indicated in Lord Thankerton's primary statement of principle in *Thomas*, an appellate court should

not come to a different conclusion from the trial judge on the basis of the printed evidence unless it is satisfied that any advantage enjoyed by the trial judge through having seen and heard the witnesses could not be sufficient to explain or justify his conclusion. Nevertheless, in certain cases, "either because the reasons given by the trial judge are not satisfactory, or because it unmistakably so appears from the evidence", an appellate court may be satisfied that the judge has not taken proper advantage of his having seen and heard the witnesses, in which case matters are at large.

[49] Secondly, an appellate court may interfere with the findings of fact made by the trial judge more readily in a case where the findings are inferences drawn from the primary facts rather than findings of primary fact based on the credibility and reliability of the witnesses; that is clear from cases such as *Benmax* v *Austin Motor Co Ltd*, *Powell and Wife* v *Streatham Manor Nursing Home* and *Flower* v *Ebbw Vale Steel, Iron and Coal Co Ltd*. In such cases, "an appeal court is generally in as good a position to evaluate the evidence as the trial judge, and ought not to shrink from that task, though it ought, of course, to give weight to his opinion" (Lord Reid in *Benmax* at page 376).

[50] Thirdly, it is apparent from *Henderson* v *Foxworth Investments Ltd* that an appellate court may interfere with the trial judge's decision on issues of fact for a range of reasons. The fact that the trial judge has gone "plainly wrong" in the sense that his decision is one that no reasonable judge could have reached, is not the only criterion for interference with his decision. The expression "plainly wrong" is found in the speech of Lord Macmillan in *Thomas* (at page 59), in the passage quoted above at paragraph [44]. It is plain from that passage that going "plainly wrong" was merely a residual category; other specific grounds for interference with the trial judge on the facts included cases where it could be demonstrated on the printed evidence that he had been affected by "material inconsistencies

and inaccuracies", or where he was shown to have failed to appreciate the weight or bearing of circumstances admitted or proved. This is recognised in *Henderson* (at paragraph 67), where the court accepts that a trial judge's decision may be interfered with on a range of grounds: these include a material error of law, or the making of a critical finding of fact that has no basis in the evidence, or if the trial judge has demonstrably misunderstood relevant evidence or failed to consider relevant evidence. The final ground, that the appellate court is satisfied that the trial judge's decision cannot reasonably be explained or justified, is a residual category, as with Lord Macmillan's reference to the judge's having gone "plainly wrong".

[51] For the foregoing reasons we are of opinion that the flexible approach adopted in the earlier cases is reaffirmed by *Henderson* v *Foxworth Investments Ltd*, a view that accords with the opinion of the then Lord Justice Clerk in *HS* v *FS*, [2015] CSIH 14, at paragraph 22. This has the advantage of preserving the flexible and undogmatic approach that has been adopted in repeated Scottish cases, notably *Thomas*: it is an approach based firmly on the advantage enjoyed by the trial judge of having seen and heard the witnesses. Moreover, the formulation in *Henderson* v *Foxworth Investments Ltd* (at paragraphs of 62 and 67) is confined to findings of fact made by the trial judge. Those observations are made in the context of a discussion of *Thomas* v *Thomas* which, for the reasons explained by Lord Reid in *Benmax* v *Austin Motor Co Ltd*, are confined to findings of primary fact – the credibility or reliability of witnesses – rather than the proper inferences to be drawn from proved facts. For this reason we are of opinion that the formulation in *Henderson*, important as it is, is concerned with findings of primary fact. An appellate court may still reassess the inferences drawn by the trial judge from proven facts. For reasons that we will discuss, inferences are frequently of particular importance when a court has to consider questions of fault and causation.

[52] It is obvious that an appellate court may interfere with the decision of a trial judge on questions of law, including the application of legal principles to the facts of the case. Questions of law can affect the evidence, as the formulation in *Henderson* demonstrates. This may happen in a number of respects. For example, the trial judge may have considered the wrong question, or may have taken account of manifestly irrelevant considerations, or may have ignored evidence that was manifestly relevant. In all these cases we consider that an appellate court may properly interfere with the findings of primary fact made by the trial judge. It can, indeed, be said that the application of the "no reasonable judge" test is an example of an error of law affecting the findings of primary fact.

Evaluation of evidence and expert evidence

[53] In a case such as the present which involves complex factual evidence and substantial amounts of expert evidence, the Lord Ordinary is obliged to evaluate the evidence led, both as to its individual components and as a totality, to determine its relevance to the fundamental issues in the case. Evaluation is essentially a matter of judgment – an evaluation of the evidence that has been led, with a view to drawing necessary inferences of fact and applying the relevant legal standard, whether as to negligence or causation. In some cases it has been said that an appellate court should be slow to interfere with a first instance judge in matters of evaluation: see *George Mitchell (Chesterhall) Ltd* v *Finney Lock Seeds Ltd*, [1983] 2 AC 803, at 815-816 per Lord Bridge of Harwich; *Re Grayan Building Services Ltd*, [1995] Ch 241, at 254-255 per Hoffmann LJ; *Biogen Inc* v *Medeva PLC*, [1997] RPC 1, at 45 per Lord Hoffmann. In the first of those cases, when considering the test of "fair and reasonable" in the Unfair Contract Terms Act 1977, Lord Bridge stated (at 815-816)

> "[T]he court must entertain a whole range of considerations, put them in the scales on one side or the other, and decide at the end of the day on which side the balance comes down. There will sometimes be room for a legitimate difference of judicial opinion as to what the answer should be, where it will be impossible to say that one view is demonstrably wrong and the other demonstrably right. It must follow… that, when asked to review such a decision on appeal, the appellate court should treat the original decision with the utmost respect and refrain from interference with it unless satisfied that it proceeded up on some erroneous principle or was plainly and obviously wrong".

Similar remarks were made by Lord Hoffmann in *Biogen*, in relation to the obviousness of an invention in the law of patents. He stated (at 45)

> "The need for appellate caution in reversing the judge's evaluation of the facts is based upon much more solid grounds than professional courtesy. It is because specific findings of fact, even by the most meticulous judge, are inherently an incomplete statement of the impression which was made upon him by the primary evidence. His expressed findings are always surrounded by a penumbra of imprecision as to emphasis, relative weight, minor qualification and nuance (*as Renan said, la vérité est dans une nuance*), of which time and language do not permit exact expression, but which may play an important part in the judge's overall evaluation".

On that basis Lord Hoffmann thought that the decision in *Benmax* v *Austin Motor Co Ltd*, *supra*, another patent case, should not be taken as permitting a *de novo* evaluation of all the facts in any case in which no question of the credibility of witnesses was involved.

[54] We agree that care must be taken in reversing evaluative decisions made by first instance judges. Nevertheless, in writing an opinion a judge has an opportunity to set out his or her reasoning at length and in detail, and in a complex case that is precisely what should be done. That exercise should make the reasoning process reasonably clear; imprecision should be minimised and any significant factors or qualifications affecting the weight of evidence should be set out. While nuance is no doubt important, we do not think that linguistic scepticism (of the sort propounded by *Renan*) needs to be taken so far that a re-evaluation of the evidence is rendered all but impossible. This is particularly the case

with expert evidence, where reliance is generally placed on reports that are based on either technical literature or practical experience; in both cases the literature or accounts of practical experience can be tested against competing views, and we consider that an appellate court should not shrink from that task.

[55] Expert evidence is never evidence of primary facts; it invariably proceeds on versions of the primary facts, sometimes including matters of inference, that are derived from the evidence of the witnesses of fact. The task of the expert is to assess that primary evidence and come to a view as to certain specific matters that are relevant to the outcome of the litigation: in the present case the alleged negligence of the midwives and two aspects of causation. In doing so, the expert may require to make inferences of fact, and must ultimately express a view on the application of a legal standard; in the case of negligence this is the standard of a midwife of ordinary competence applying ordinary skill and care; in the case of causation, the tests are largely practical and depend on particular circumstances: see paragraph [56] below. In every case, however, the views of expert witnesses as to the application of a legal test are not binding on the court. Instead the judge must evaluate the expert evidence as a whole, decide which parts of that evidence should be accepted or rejected, and on the basis of the parts that are accepted decide whether the relevant legal standard is indeed met. On that question, although respect is obviously due to the views of the first instance judge, an appellate court can properly come to a contrary view; the expert evidence that is relied on is never evidence of primary fact, and the inferential, deductive and analogical processes that are used in that evidence can be assessed by an appellate court just as effectively as by the judge at first instance.

[56] In evaluating expert evidence, both a judge at first instance and an appellate court are in our opinion entitled to apply ordinary standards of logic and common sense. What a

court requires to do in matters of causation, is to look critically at the various explanations advanced by the experts, evaluating them against standards of logic and practical likelihood, and decide which, if any, is a probable explanation for the sequence of events. The competing explanations must be compared with each other, to discover which parts can be reconciled and which cannot; and to the extent that the views of experts are irreconcilable the court must choose between them, applying standards of logic and common sense and comparing the views of the experts with the generally accepted scientific or medical or other professional knowledge in the area under consideration. That will frequently involve consideration of literature in the field, but that literature must itself be scrutinised carefully to discover how soundly it is based and to determine whether it is indeed relevant to the particular case under consideration. That applies with particular force to epidemiological studies, where the underlying basis of the study may be either unsound or irrelevant to the particular case that is under consideration: compare *Dingley* v *Chief Constable, Strathclyde Police*, 1998 SC 548, at 569-571, where epidemiological studies are discussed at length. The same is true of the views advanced by experts based on their practical experience. In this case, for example, the views of Professor de Swiet involved working backwards from the time of delivery rather than working forward from the time when symptoms of eclampsia first became apparent. Professors Walker and Murphy, by contrast, were of opinion that it was not permissible to work backwards, as the time of delivery was essentially arbitrary in relation to the development of eclampsia. In this case we prefer the views of Professors Walker and Murphy because we consider that the logic of their reasoning is preferable. That is an example of comparing the views of experts by using logic and common sense. We address this issue further, in respect of the present case, at paragraphs [137] to [141] below.

[57] On that basis the court should be able to determine whether, as a matter of probability, an explanation for, in this case, LW's condition has been established. It is of course open to a court to decide that none of the competing explanations has been proved on the balance of probabilities, in which case the pursuer as the party bearing the burden of proof must fail; that is the fundamental point decided in *Rhesa Shipping Co SA (The Popi M)*, [1985] 1 WLR 948, discussed in more detail at paragraph [297] below. The processes leading to such a conclusion, however, involve evaluation of the expert evidence in the manner that we have sought to describe. Those are processes that can and should be carried out by an appellate court if it appears that the Lord Ordinary's reasoning is defective or inadequate, either as to the analysis of the expert evidence or as to its practical application.

[58] Finally, we should note the important point made by Lord Prosser in *Dingley* v *Chief Constable, Strathclyde Police*, *supra*, at 604, and repeated by Lord Hodge in *Kennedy* v *Cordia (Services) LLP*, 2016 SC (UKSC) 59, at paragraph 48 (quoted at paragraph [138] below), that in assessing expert evidence courts should have regard to the explanations that are given for the views advanced, rather than to the mere fact that a view is widely held, or the apparent distinction of the person expressing the view. Critical assessment of the reasoning underlying expert evidence, and of the premises on which it is based, is essential.

Powers of an appellate court in the circumstances of the present case

[59] We must now consider how the foregoing principles apply to the circumstances of the present case, with particular reference to the three primary issues that arise in the reclaiming motion and cross-appeal. We will first consider their significance in relation to the Lord Ordinary's findings on the negligence of the midwives, and thereafter his findings on causation: first in relation to the consequences of negligence of the midwives and

secondly on the question of general causation. At the outset, it should be noted that all three issues require the drawing of inferences from the primary facts, and all three require evaluation of the facts, both primary and inferential, against the legal standards applicable to negligence or causation, as the case may be. The expert evidence must be examined critically to discover which version, if any, is preferred. Thereafter the inferences that are drawn must be informed by that version of the expert evidence. Finally, the relevant legal standard must be applied, making use of the explanations given in that preferred version of the expert evidence. All of these are matters that can be subject to reconsideration by an appellate court if it forms the conclusion that the Lord Ordinary's reasoning is defective or inadequate.

The Lord Ordinary's findings on negligence of midwives

[60] The Lord Ordinary's findings on negligence are based largely on a determination of the primary facts, and in particular his findings at paragraphs [100] and [101] as to what happened in the course of the visit made by Mrs Giles and Mrs Mohammed on Saturday, 5 October 1996. At paragraphs [89]-[98] the Lord Ordinary embarks on a detailed analysis of the credibility and in particular the reliability of the pursuer, her husband and the two midwives, and concludes that in cases of doubt the evidence of the pursuer and her husband on the events of that day is to be preferred. That conclusion is central to the Lord Ordinary's preferred version of what transpired on 5 October.

[61] The Lord Ordinary's conclusions on negligence found in large measure on his findings of primary fact. The critical findings are that no sample of urine was taken from the pursuer and tested and the pursuer's blood pressure was not tested, and that symptoms suggestive of pre-eclampsia were reported but not recorded or acted upon. Expert evidence was relevant, in that (paragraph [101]) Mrs Tranter gave evidence that the symptoms

reported by the pursuer, headaches, visual disturbance and blurred vision, merited referral to hospital regardless of the presence of other signs of pre-eclampsia such as hypertension and proteinuria. Dr McConville supported Mrs Tranter in that opinion. On that basis the Lord Ordinary inferred that the procedure that should be followed by a midwife of ordinary competence was that described by Mrs Tranter and Dr McConville; consequently the failure of Mrs Giles and Mrs Mohammed to follow such a procedure in the circumstances reported to them was negligent. While that represents matters of inference and the application of a legal standard, the conclusion reached is heavily dependent on findings of primary fact.

[62] In relation to the negligence of the midwives, therefore, we are of opinion that the principle laid down principally by Lord Thankerton in *Thomas* v *Thomas* (paragraph [41] above) is relevant to the Lord Ordinary's findings of primary fact. These include what transpired at the meeting of 5 October between the pursuer, Mrs Giles and Mrs Mohammed, and in particular what the pursuer told the midwives and what precisely the midwives did in response to that information. That means that we should only interfere with those findings in the event that the Lord Ordinary's reasons are not satisfactory, or because it "unmistakably so appears from the evidence" that he has not taken proper advantage of having seen and heard the witnesses. That is inevitably a demanding test. The conclusion that the midwives were negligent, however, is in a somewhat different position, for the reasons that we have already explained. To the extent that determining the relevance of the expert evidence to the facts depends on the drawing of factual inferences, the approach laid down by Lord Reid in *Benmax* v *Austin Motor Co Ltd* (paragraph [44] above) is applicable: we are accordingly in a position if so advised to revisit the inferences drawn by the Lord Ordinary on the basis of the expert evidence. The final aspect of the application of expert evidence involves the application of a legal standard: essentially the standard that no

midwife of ordinary competence exercising ordinary skill and care would have adopted the course adopted by Mrs Giles and Mrs Mohammed in the circumstances in which they found themselves. An appellate court can interfere with the application of a legal standard. In the present case, in relation to negligence, there is a conflict in the expert evidence between that of Mrs Tranter and Dr McConville on one hand and Dr Sanders on the other hand. We discuss this subsequently, at some length (paragraphs [82]-[94]). While we find that there were inadequacies in the Lord Ordinary's reasoning, his final preference for the evidence of Mrs Tranter and Dr McConville was based on justifiable considerations (paragraphs [95]-[97] below); in particular, Dr Sanders had not considered a number of important factors, and her overall approach was based on individual symptoms rather than the overall clinical picture.

The Lord Ordinary's findings on causation

[63] The analysis of causation in a medical negligence case (and more generally) involves a number of stages, which we have attempted to discuss above. The task is at its most difficult in cases where there is no obvious physical connection between the fault relied on and the ultimate injury. Ultimately, however, the court must apply the legal test for causation. Usually this will be fairly straightforward: did negligent act A cause injury B? In some cases, however, a simple common sense approach to causality is not enough, and considerations of policy intervene to determine whether a person who has been at fault in a specified respect should bear responsibility for a particular result; this arises where, for example, the fault is only one of the causes of the result, or the result might have happened in any event.

[64] We were referred to a number of cases falling into this category, notably *Chappel* v *Hart*, [1999] Lloyd's Rep Med 223, *Chester* v *Afshar*, [2005] 1 AC 134, and *Vaile* v *London*

Borough of Havering, [2011] EWCA Civ 246, [2011] ELR 274. It is not necessary for present purposes to give those cases detailed consideration. What has happened is that the courts have decided as a matter of policy that, for example, failure to warn a patient of a risk inherent in a particular surgical procedure could be held to be causative of the injury caused by that risk, even though the risk was inevitable if the procedure were to be carried out: this was the situation considered in *Chappel* v *Hart* and *Chester* v *Afshar*; the relevance of legal policy is discussed in particular in the latter case by Lord Hope of Craighead at paragraphs 85-88 and by Kirby J in the former case at paragraph 95. In the present case, however, the issues of causation appear to us to be conceptually relatively straightforward: was LW's injury caused by the failure in professional practice of the midwives, and if so, was his injury caused by a single insult, as posited by the pursuer's experts, or by more general causes, as spoken to by the defenders' experts. Neither of these questions appear to us to raise significant issues of legal policy. What is required is an evaluation of the competing expert views, in the light of the primary facts found and the reasoning advanced in support of their views by the experts.

[65] In a case of this nature, we are of opinion that, at least if the primary facts are clear, it is open to an appellate court to interfere with the decision of the trial judge, by analogy with the general approach adopted in *Thomas* v *Thomas* and especially by Lord Reid in *Benmax* v *Austin Motor Co Ltd*. The critical point is that, provided that the primary facts are clear, an appellate court is generally in as good a position to evaluate the evidence as the trial judge and, as Lord Reid observes, it ought not to shrink from that task, although it ought to give weight to the trial judge's opinion (on which see paragraphs [44]-[45] above). In this connection, it must be borne in mind that the trial judge has certain advantages, in particular observing witnesses as they give evidence and immersing himself in the evidence in a way

that is simply not possible for an appellate court. It is important that an appellate court should take these factors into account.

[66] Finally, at a general level, it should be noted that the giving of inadequate reasons, or reasons that are demonstrably faulty, is a ground of challenge in itself. For this purpose, it is not material whether the challenge is to primary facts, inferences or the application of a legal standard; indeed, giving inadequate or unsatisfactory reasons is itself an error of law. For the reasons discussed below, we are of opinion that the Lord Ordinary's reasons in relation to the second causation issue are inadequate, and his decision on that is accordingly open to reconsideration.

General causation

[67] Before turning to the facts of the case, we should say something about the important issue of proof of general causation, in the sense of LW's injury being caused by events traceable to the midwives' negligence rather than other factors such as IUGR (what we have designated the second causation issue). As was freely conceded by the pursuer the foundation upon which her case is built is the neuroradiological evidence and in particular what is shown by magnetic resonance imaging (MRI), as interpreted and extrapolated upon by Dr Kendall and Dr Connolly. We have therefore thought it important to scrutinise that evidence with particular care, both in itself and in the inferences that can be drawn from it

[68] Two further points should be made in relation to the general approach to expert evidence relating to general causation. First, in assessing the competing theories advanced by the parties, it is the total explanation put forward by each that must be considered and assessed against the total explanation put forward by the other. Differences of detail may be important; the evidence relating to the relationship of eclampsia and delivery is perhaps an

example of this. Ultimately, however, it is the total explanation that must be assessed to discover whether the pursuer has proved a case.

[69] Secondly, Professor Murphy and Professor Walker, in particular, made some reference to statistical evidence. Thus Professor Murphy gave evidence, on the basis of the study that she and others had carried out and which have been published in The Lancet, that only 3 to 20% of cases of cerebral palsy in infants born at term are due to intrapartum asphyxia. That means that between 80 and 97% are not caused by intrapartum asphyxia. If that is so, it suggests that the hypothesis put forward on behalf of the pursuer is a relatively rare event. That raises the question of the significance that a court should attach to statistical evidence of this nature. We have reached the conclusion that such evidence is relevant, but will not normally be conclusive. We say "normally", because statistical evidence that suggests that event A almost invariably produces result B might of itself be conclusive. Indeed much common sense observation of causation is of that nature: event A seems invariably to be followed by event B, and from that it is concluded that B is caused by A. Where the correlation is less than that, however, we think that some care must be taken in relying on the statistical evidence. Weight can be given to it, according to how compelling the statistics appear to be, but a court should normally look for other corroborating evidence to justify the statistical inference. Furthermore, the basis for the statistics must in all cases be subjected to critical examination; if it appears to be based on defective epidemiological studies, it should be disregarded.

Factual issues

Credibility and Reliability

[70] The Lord Ordinary concluded that:

(i) no urine test was done;

(ii) no blood pressure was taken; and

(iii) that the pursuer reported to the midwives symptoms "suggestive of pre-eclampsia"

which they failed to record or act upon.

[71] The issues leading to the findings of fact (i), (ii) and (iii) above turn, almost entirely, on the Lord Ordinary's assessment of credibility and reliability. The defender advanced numerous challenges to the Lord Ordinary's findings on credibility. The essence of these were as follows, numbered for convenience.

(i) The Lord Ordinary did not properly consider the defender's case of "confabulation", meaning a process by which a witness over time comes to recall incidents which did not happen.

(ii) He treated the issue of credibility as a contest between the pursuer and her husband on the one hand, and the midwives on the other. He should have considered also (a) the inherent unlikelihood that midwives would have omitted such basic tests; and (b) the effect the evidence of the treating clinicians and midwives had on the pursuer's credibility and reliability. Both of these issues told against a finding that the pursuer was credible and reliable.

(iii) He made an unjustified leap from the conclusion that the pursuer was trying to tell the truth, to the conclusion that she was credible and reliable.

(iv) His observation of Mrs Giles that "… whilst maintaining a general lack of memory of the events, she did at times appear to indicate that she had some knowledge of these events from her memory" was not fair on the evidence .

(v) He gave inadequate reasons for his conclusions on credibility and reliability. In particular his reasoning was deficient in the following respects:-

(i) in his treatment of the entry "C/C" in the PCP, which offered support for a urine test having been carried out;

(ii) so far as events on 8/10 were concerned, in saying that the pursuer's account was "at least in a broad sense supported by the medical records"… but "did not accord in all respects with matters noted in the medical records", he did not explain where the alleged support for the pursuer lay.

(iii) he stated (end of paragraph 94) that the pursuer was in general credible and reliable adding "albeit there are a number of matters of evidence which I am not prepared to accept". It was not clear what these matters were.

Analysis and conclusions on credibility and reliability

[72] We do not consider that there is any merit in the first of these criticisms. The Lord Ordinary's observations that this submission was no more than an assertion, unsupported by any evidence, requires to be read in context. He was clearly meaning that there was no direct or psychological evidence of this. His remarks do not mean that he ignored the general aspects of the evidence, or submissions, about the way the pursuer's account allegedly developed over the years, and the reasons therefore, which he specifically recorded at paragraph 92 of his opinion. Equally, the fourth criticism is not valid. The Lord Ordinary heard and saw Mrs Giles give evidence over a period of two days. On the second day, she commenced her evidence by stating that she was concerned that the previous day she might have given the impression that she agreed with certain of the pursuer's propositions in fact, which she had not intended to do. The Lord Ordinary

records this at paragraph 18, as well as commenting on it in his assessment of credibility. Certain of her answers on the first day may well have given the impression referred to by the Lord Ordinary (eg MS 994 line 5 to 994 line 14).

[73] So far as the remaining criticisms are concerned, the position is less clear cut. The Lord Ordinary correctly considered that the credibility and reliability of the pursuer and her husband on the one hand and that of the two midwives on the other was central to all the factual issues underlying the cases of fault. He did proceed to assess the issue of the credibility of the pursuer (and her husband) largely in the context of the evidence of the midwives. To some extent this is understandable; the factual dispute as to what occurred on 5 October was central to the case. In making his assessment, the Lord Ordinary did not ignore the evidence of the midwives that having regard to the routine nature of the tests, they would both have been carried out. In fact, he records this as paragraph 96, in dealing with credibility, having previously recorded the evidence about this at paragraph 16. As to the PCP, he was alert to the submission that the entry supported the evidence of the midwives that they would have followed usual practice and taken a urine sample. He recorded the midwives' evidence, where they pointed to the entry as evidence that the urine sample was taken (paragraph 100). He noted that it would have been routine practice to do so. The pursuer's evidence was that on 8 October she saw Mrs Giles at the hospital and Mr Giles took the PCP from her. Mrs Giles had no recollection of seeing the pursuer at the hospital on that date. The Lord Ordinary's suggestion that the evidence regarding handing over of the PCP was unchallenged, is only accurate in that Mrs Giles had no recollection of it. There was no concession that this took place. However, it is clear that Mrs Giles saw the pursuer that day at hospital and made an entry in the pursuer's notes that day. (No 7/1 of process, page 341- MS 10308; evidence: -MS 1055). The Lord Ordinary concluded that the

entry "C/C" was likely to have been made by Mrs Giles, since the other midwife used a different notation. The Lord Ordinary however drew back from reaching any conclusion about the circumstances in which the entry had been made. He stated that there was no inference that he felt he could properly draw from the evidence of the PCP. Looking at the issues on paper, an appeal court might attach a greater or lesser importance than did the Lord Ordinary to this evidence, or make more conclusive findings. However, as with other aspects of his reasoning, this matter must be looked at in the context of a proof where he was able to observe the primary witnesses of fact over several days. He was alert to the submission that the entry supported the evidence of the midwives that they would have followed usual practice and taken a urine sample. He recorded the midwives as pointing to the entry as evidence that the urine sample was taken [paragraph 100]. It is clear that he felt unable to draw such a conclusion.

[74] It is not clear what the Lord Ordinary meant by saying that the pursuer's version of events of 8 October was "at least in a broad sense supported by the medical records". Of course, in the very broadest sense, this is true. However, in certain respects — her suggestion that she had a prior preliminary scan by a midwife; her assertion that she continued to suffer symptoms of headaches etc; — the records did not support this. Perhaps the Lord Ordinary had in mind the entry showing the presence of Mrs Giles at the hospital. At least he correctly recorded that the pursuer's evidence did not accord in all respects with the matters noted in the medical records. In our view the gloss which the defender places on the last two sentences of paragraph 94 of the Lord Ordinary's opinion is unfair. Those passages occur in the context of the Lord Ordinary having recorded that there were certain inconsistencies with the medical records. He cannot, in light of the views expressed on credibility as a whole, be taken as meaning that "the pursuer was trying to tell the truth *ergo*

she was credible and reliable": rather he was explaining that the pursuer's memory was wrong about these matters, but that the discrepancies were not such as to cause him to have doubts about a witness whom he otherwise judged to be honest and reliable.

[75] The Lord Ordinary has explained the reasons for his findings on credibility and reliability. No doubt he might have expressed himself more clearly, and it would indeed have been preferable had he done so. However, as we have already noted, this is an area in which to a particular degree the assessment of the Lord Ordinary must be given respect. This court has not had the benefit of seeing and hearing either the pursuer or her husband or either of the midwives. It is not insignificant in this regard to note that the pursuer gave evidence over a period of five days, during which the Lord Ordinary would, as he himself noted, have had the opportunity to observe closely her conduct and demeanour when doing so.

[76] On the other hand, the Lord Ordinary clearly had concerns as to the credibility and reliability of Mrs Giles, who was in the witness box over a period of two days. He considered her demeanour to be "less than wholly satisfactory" for reasons to which we have already adverted, and which, in our view was a conclusion that he was entitled to reach.

[77] It is not the case that the Lord Ordinary has left out of account any significant evidence relating to credibility and reliability and his assessment was made after seeing and hearing witnesses who gave evidence at length and were subjected to detailed and lengthy cross-examination. In that regard we must bear in mind the familiar passage in the speech of Viscount Simon in *Thomas* v *Thomas* (page 47) quoted at paragraph [42] above. As the UKSC explained in *McGraddie* v *McGraddie* 2014 SC (UKSC) 12, the question of credibility and reliability is pre-eminently a matter for the Lord Ordinary:

> "[28] In a case where the court was faced with a stark choice between irreconcilable accounts, the credibility of the parties' testimony was an issue of primary importance. The Lord Ordinary found that the pursuer was a credible witness on the central issue, notwithstanding a number of aspects of the evidence which could be regarded as detracting from his credibility, including the aspects mentioned in para [26]. The question whether the pursuer's evidence was to be regarded as credible and reliable having regard to the other evidence in the case was pre-eminently a matter for the Lord Ordinary."

That is exactly what has happened here. The Lord Ordinary found the pursuer to be a credible witness on the central issue notwithstanding certain aspects of her evidence which might detract from her credibility. It is plain that the attitude and demeanour of the witnesses when giving evidence were matters which impacted upon the Lord Ordinary's assessment of credibility and reliability. The significance of this in relation to reliability was noted by Lord Hope in *Thomson* v *Kvaerner Govan Limited* in the passage quoted at paragraph [42] above. Where, as here, credibility is also in issue, these observations have even greater force.

[78] In the present case we see no basis upon which we might justifiably conclude that the Lord Ordinary erred in his assessment of credibility and reliability. He has made very clear findings of primary fact which should be given respect by this court. That conclusion has certain consequences so far as the issues of negligence and causation are concerned. It means that we must approach those issues on the factual basis that:-

(i) no urine test was carried out;

(ii) no blood pressure was taken; and

(iii) the pursuer reported to the midwives on 5 October symptoms such as might raise concerns as to the possibility of pre-eclampsia but that they did not record or act upon them.

As to these symptoms, at paragraph 93 the Lord Ordinary recorded that they were such as to cause the pursuer to have concerns both "about her health and the unborn baby's wellbeing."

Negligence

[79] There was really no dispute that failure to check urine or blood pressure would have amounted to negligence. The Lord Ordinary accepted the evidence of Mrs Tranter that, of the symptoms reported, headaches, visual disturbance, and blurred vision merited referral to hospital regardless of the presence of other signs. He accepted the evidence of Dr McConville agreeing with this. The Lord Ordinary did not find that the failures to check urine and blood pressure should have resulted in referral to hospital – it would have been impossible for him to do so, particularly given his approach to the evidence of Professor de Swiet. His conclusion related to all three elements together. As he stated, the implication of that (i.e. that all three grounds of negligence were established) was that the pursuer should have been referred to hospital on 5 October. One can understand that: if it were appropriate to refer on the basis of the symptoms alone, a referral would be all the more mandated in the absence of testing blood pressure or urine.

[80] As to the reporting of symptoms suggestive of pre-eclampsia, the Lord Ordinary stated "Having regard to the clear evidence of [the pursuer] and her husband I hold that such symptoms were reported" (paragraph 101). It is obvious from the subsequent context of that paragraph that he accepted that the symptoms of headaches, visual disturbance and blurred vision had been reported. However, it is also reasonable to conclude from his narrative of the evidence of the pursuer and her husband at paragraphs 11 to 20 and his

comments thereanent in paragraph 101, that he was satisfied that the reported symptoms also included:

- feeling hot and puffy in the face;
- being tearful;
- feeling tired;
- having experienced reduced foetal movement;
- having experienced foetal movements of a different nature to previously, very light and like flutters;
- concerns about the size of the child, with the feeling that her stomach had not grown in size since the last visit.

[81] In addressing the question whether these symptoms merited referral to hospital on 5 October, the Lord Ordinary relied on the evidence of Mrs Sandra Tranter and Dr Jean McConville that the symptoms of headaches, visual disturbance and blurred vision in themselves merited referral to hospital regardless of the presence of signs such as hypertension and proteinuria (paragraph 101). In general, he preferred the evidence of these witnesses to that of the pursuer's expert, Dr Julia Sanders. However, the Lord Ordinary's treatment of the evidence of all three witnesses can best be described as "scant", amounting to a mere six paragraphs. In three lines in paragraph 101 he stated that he accepted the evidence of Mrs Tranter and Dr McConville (without specifically stating that he rejected that of Dr Sanders) and concluded that failure to refer had been negligent. In order to address the defender's argument that the Lord Ordinary's approach was inadequate, and left the defender unclear why he had reached the conclusion he did, we require to consider the evidence given by these witnesses and its import.

Mrs Tranter

Maternal Symptoms

[82] Mrs Tranter in her report, to which she spoke in evidence, stated that if the court were to conclude that no urine test had been carried out, the entry "C/C" could not represent clinical observation. She described the failure to record blood pressure as a serious omission, which appeared to indicate that it was not taken. She noted that the pursuer had been complaining of feeling unwell and said that:

> "Had her blood pressure been taken and found to be raised the community midwives should have referred her into the hospital for a medical assessment. "

adding

> "Hypertension in pregnancy, with or without proteinuria, is a deviation from normal requiring a medical review."

[83] In connection with the reported symptoms she noted:

> "women with pre-eclampsia can develop symptoms … which can include headaches, visual disturbances such as blurred vision, epigastric pain and nausea and complaining of feeling unwell. ….Any of these symptoms should therefore be taken seriously by the attending midwife and if found in the presence of hypertension and proteinuria, a woman should be transferred promptly to obstetric medical care."

[84] The reported symptoms of feeling unwell, with headaches and visual disturbance, along with the concerns about foetal movement and growth should have prompted the attending midwives to consider the possibility of pre-eclampsia and to take particular care to check the blood pressure. Had blood pressure been raised, then it should have been re-checked and if it remained raised, they should have referred the pursuer to hospital for a medical review.

[85] Mrs Tranter was cross-examined by counsel for the defender in respect of each individual symptom, and whether individually they would warrant referral. She stated that

even if the blood pressure were not raised, she would still have sent the pursuer to hospital, saying that she needed a "raft of blood tests, she needs her blood pressure checked thoroughly, she needs a CTG, and as they did here, a scan". She also stated (MS 4919, 4920) that the complaint of headaches and blurred vision around the edges, indeed the latter on its own, warranted immediate referral to hospital. On such a referral, it would be expected that a midwife would admit her and carry out baseline observations but the referral would have been for an obstetrician to assess her, not another midwife (MS 4908).

> "Normal healthy young, pregnant women do not get headaches and blurred vision." (MS 4919)

[86] When asked about puffiness, her answer, rather reflecting a point made elsewhere by the Lord Ordinary was that

> "in the context of the other symptoms she was discussing, blurred vision and headaches, it, you're beginning to roll up a picture, aren't you?" (MS 4921)

As counsel proceeded further through each separate symptom, the Lord Ordinary interjected:

> "Q: If she reported all these symptoms to the midwives, then my strong impression is you would have no doubt that she should have been sent to hospital?
>
> A: Absolutely."

[87] In due course she said that even with a normal blood pressure reading, the complaints of headache, blurred vision and reduced foetal movements, alone or in combination, should have led to referral to hospital for a medical review, CTG and possibly ultrasound, that being a matter for the obstetrician to determine (MS 4928). She agreed with Dr Sanders' view that a finding of moderate or severe hypertension would have been an indication for hospital review, but pointed out that what degree of hypertension might have

been present on 5 October was unknown. As to Dr Sanders' comment that whether the referral would have been immediate or delayed until the Monday would depend on the degree of hypertension, she said:

> "Well that's taking it in isolation away from the other symptoms that AW reported, so I don't agree. I think she should, as I keep saying, she should have gone in." (4952)

Foetal Symptoms

[88] In her report Mrs Tranter said that reduced foetal movements are an indication that all is not well and should have prompted the attending midwives to send the pursuer into hospital for a CTG and a medical review. The pursuer's concerns regarding the growth of her foetus and in particular reduced foetal movements should have resulted in her being referred to hospital for a CTG and an obstetric medical assessment. In her evidence she confirmed her opinion that any report of reduced foetal movements should be taken very seriously as an indication that all is not well, and should result in referral to hospital for a CTG (MS 4788). The report of reduced foetal movements should itself have resulted in referral. An ordinarily competent midwife should have taken that step (MS 4922).

Dr McConville

Maternal Symptoms

[89] In her report, Dr McConville stated (4.6):

> "It is my opinion that as AW had already complained of feeling unwell, headaches and reduced foetal movements that the midwives could and should have taken her blood pressure and, whatever the blood pressure recording, should have referred her to the hospital for further investigations to confirm foetal and maternal wellbeing."

[90] During pregnancy women often complain of being unable to read, but visual disturbance with blurring or peripheral disturbance can be a sign of pre-eclampsia, which is

why it is important to ascertain whether there is actual disturbance, which indicates abnormality (MS 5084 to 5085). Headaches and blurred vision are cardinal symptoms of pregnancy-induced hypertension and pre-eclampsia (MS 5081). Dr McConville too was taken through every individual symptom in cross-examination. This led to a question in re-examination:

> "If [AW] had given the midwives in that clinic the list of symptoms and concerns that were put to you by my learned friend as [AW's] symptoms and concerns, would it have been, eh, acceptable practice for them to not ask for assistance from a doctor? – No. They would need to refer". (13726)

Foetal Symptoms

[91] In Dr McConville's opinion a competent midwife exercising ordinary care would have referred the pursuer to hospital on 5/10 because of the reduced foetal movements regardless of blood pressure.

> "I'd be very, very concerned with a mum who is telling me that she has got movements that don't feel right to her.
>
> And the answer is to refer her in for a CTG, is that right? – Yes, you need to refer her to the hospital for a doctor's opinion, and that would include an antenatal check on the labour ward or on a ward, they would run a routine cardiotocograph, yes." (MS 5088-5089).

She repeated this both in cross and re-examination.

Dr Sanders

Maternal Symptoms

[92] If a woman reported a combination of headaches and blurred vision it would make one anxious about the possibility of pre-eclampsia, but these symptoms are not diagnostic and it would be necessary to check blood pressure and urine to exclude that possibility (MS 8028 to 8029). Without the blood pressure the midwife would not have the full picture upon which to make a referral (MS 8045).

> "Any woman that reports classical signs of pre-eclampsia, being a headache, blurred vision, their immediate response is to check her blood..." (MS 8070)

Headaches and blurred vision are such classical signs of pre-eclampsia that no midwife would ignore them, reported in that combination (MS 8394), but having excluded pre-eclampsia through the absence of proteinuria, a midwife could be confident that this was not the cause of the symptoms. Even mild hypertension, in the presence of proteinuria would initiate a hospital referral, but hypertension without proteinuria would not do so.

[93] It was put to her that with a general picture of headaches, blurred vision, hot and puffy face, the baby seemingly not having grown for two weeks plus, and several days of reduction in the strength of the movements, had a midwife for some reason been unable to take blood pressure there would have been a duty to refer. She disagreed, but did so on the basis that she could not conceive of circumstances in which it would not be possible to take blood pressure (MS 8407).

Foetal Symptoms

[94] In examination-in-chief Dr Sanders said that any report of reduced foetal movements from a woman would lead to further enquiry (MS 8031), but it is fair to say that the way her evidence was elicited meant that much greater concentration was given to the question of foetal growth, and her opinion that the concern of a mother or her friend that the bump was not growing would not itself merit referral. She would have been more concerned about any suggestion that the baby had not grown between visits (which was the complaint, as found by the Lord Ordinary). A change in the momentum of growth would be taken seriously. However, if on palpation the size appeared commensurate with gestational dates, a midwife would be reassured. The issue of foetal movement was returned to in cross, where her evidence generally was that in 1996 it would have been more common to ask

about the number of movements and to be reassured by that, rather than examine the character of them, although "a particularly cautious midwife might have referred a woman in, based on the woman's concern about the movements". The first test regarding reduced foetal movement would be a CTG. The only reported symptom that would indicate an ultrasound scan is that of concern over lack of growth. Such concerns might result in a referral appointment within a period of about seven days.

Analysis and conclusions

[95] In our view the conclusions which the Lord Ordinary reached in relation to the evidence of Mrs Tranter and Dr McConville were open to him on the basis of the evidence as a whole. The Lord Ordinary did not, at paragraph 101, explain his reasons for rejecting the evidence of Dr Sanders. It would of course have been better had he done so. It is important to note, however, that the evidence of this witness proceeded on the assumptions that (i) there was a clear urine test carried out; and (ii) either the midwives omitted to check the pursuer's blood pressure, which suggests symptoms of pre-eclampsia were absent , or sufficiently mild not to prompt two experienced midwives to remember to check it; or it was taken and found to be completely normal, again giving no prompts to the midwives to record the finding or plan additional surveillance. In other words, her opinion proceeded on a different factual basis from that which the Lord Ordinary found actually to be the case.

[96] In recording the evidence of Dr Sanders, the Lord Ordinary noted that:-

(a) she had observed that midwives do not necessarily record all pregnancy related complaints which would be recorded only if they were severe; and

(b) that it was not possible to determine from the records which, if any, pregnancy related problems were reported or what discussion took place.

He thus recorded that her evidence proceeded on the basis that had severe symptoms been reported, they would have been recorded. Such an approach accorded with the general tenor of her evidence which was substantially based on usual practice and what was to be expected of an experienced midwife. In relation to the question of blood pressure she considered that there were two possible scenarios, as noted above. Clearly she did not consider the possibility that the midwives had simply erred and failed to check the blood pressure. In the course of an objection outwith the presence of the witness the Lord Ordinary himself had noted that there was in fact that third possibility which required to be considered namely that of mistake, pure and simple, which he would have to decide upon.

[97] Furthermore in taking the evidence of Dr Sanders, counsel for the defender went through each alleged symptom individually, asking about the possible significance and whether a referral to hospital would be merited. At more than one point the Lord Ordinary interjected to indicate that in fact, as the evidence showed, the midwife would not look at individual symptoms but at the whole clinical picture. The Lord Ordinary has reported all these aspects of Dr Sanders' evidence in his summary thereof and in our view it cannot be said that the defender would be unable to understand why the evidence of the pursuer's expert midwives had been accepted over that of Dr Sanders.

Referral for both maternal and foetal concerns?

[98] The pursuer's case proceeded on the basis that on 5 October the pursuer was presenting with symptoms relating to maternal health (potentially indicative of pre-eclampsia) and symptoms relating to foetal health (potentially indicative of a compromised foetus). The pleadings are a model neither of clarity nor brevity. They do not distinguish as

clearly as they might have done between the maternal and the foetal symptoms. However it can be discerned that although it was maintained that the symptoms, taken together, merited referral to hospital, and that failure to do so was negligent, it can also be discerned that the pursuer was averring that either the maternal symptoms alone or the foetal symptoms alone merited referral to hospital.

[99] In article 4 of Condescendence it is averred that the pursuer's concerns regarding lack of growth and reduced foetal movements, indeed the latter alone, merited referral for CTG. It is averred that symptoms of headaches, blurred vision and feeling unwell are classic symptoms of pre-eclampsia and that had the midwives listened to the pursuer's complaints she should have been referred to hospital for investigation (page 12D to 13B of the Reclaiming Print).

[100] The Lord Ordinary made a finding of negligence in respect of the maternal symptoms. He did not consider the foetal symptoms (see paragraph 101). This is accordingly one aspect of the pursuer's case which the Lord Ordinary completely failed to address, and on any view it is thus open to us to do so.

[101] On the question whether foetal symptoms were reported to the midwives, we have noted at paragraph [80] above, that the Lord Ordinary was satisfied that the symptoms reported included foetal movements of a different nature, light and like flutters; and concern over lack of growth since the previous visit. The pursuer's evidence had been referred to a reduction in both the number and the strength of foetal movements.

[102] The evidence indicated that where a mother complained of reduced foetal movements she should be referred to hospital for CTG and that this would be the course adopted by any ordinary competent midwife. This was the very clear evidence of Mrs Tranter and Dr McConville. Dr Sanders' evidence focussed more on the issue of foetal

growth, but it is of note that the pursuer's evidence did not just relate to a concern that her "bump was small" but that on 5 October the baby did not seem have to have grown since the last visit. Her concern about foetal movements related not only to reduced strength, but reduced numbers, which Dr Sanders suggested might have been a primary matter of concern in 1996.

[103] On the basis of the very clear evidence of Mrs Tranter and Dr McConville, there is in our view no difficulty in concluding that the symptoms relating to lack of growth, and more specifically reduced foetal movements, which the Lord Ordinary found to have been reported, were such that the pursuer should have been referred to hospital for CTG and that failure to do so was negligent. The evidence of Dr Sanders was that she would be concerned if a mother complained that her baby had not grown between visits, which was indeed the complaint. She did not dispute that such a complaint might result in a referral, her evidence being that it might not follow immediately.

[104] As we have observed, the Lord Ordinary addressed the question whether the pursuer would have been admitted to hospital on referral on 5 October largely on the basis of the maternal symptoms and whether the pursuer would have had elevated blood pressure at that time. He also considered the question whether there would have been protein in the urine (about which the pursuer's position on the pleadings was not wholly clear). In relation to the question of foetal health, he appears to have considered only the question of foetal growth, and did not take account of the evidence relating to concerns over foetal movement. At paragraph 112 he refers to what he calls the "criteria" for admission – namely checks on blood pressure, urine and fundal height. As to these three matters, he concluded that the pursuer could not prove that on referral on 5 October she would have had high blood pressure or an abnormal urine test. He concluded that fundal height would

have been assessed and found to reveal no abnormality. On the basis that none of these tests would have revealed an abnormality, he concluded that the pursuer would not have been admitted on 5 October and that she would have been sent home.

Maternal Symptoms

[105] The Lord Ordinary was correct to say that the pursuer's case that on 5 October she would have been admitted to hospital on the maternal symptoms hinged on her being able to establish, on a balance of probabilities, that on that date testing of her blood pressure and urine would have revealed results sufficiently abnormal to merit admission. That issue depended on the Lord Ordinary accepting the evidence of Professor de Swiet who, in his report, had addressed the question:

> "What is the likelihood that [the pursuer] would have had proteinuria and hypertension had these signs been looked for when the midwives came on 5/10/96". (See his report 6/14 of process MS 9491).

The Lord Ordinary rejected the evidence of Professor de Swiet as (a) theoretical and (b) unsupported by the clinicians who treated the pursuer on 8 October. The Lord Ordinary set out his reasons for rejecting Professor de Swiet at paragraph 112 which requires to be read with his narration of the defender's submissions about this matter at paragraphs 109 and 110. The Lord Ordinary's view was that the absence of proof in the evidence of Professor de Swiet and his dependence on inferential conjecture might not be problematic but for the powerful clinical and expert evidence that the pre-eclamptic process was highly unpredictable.

[106] The evidence of the clinicians was that they proceeded on the basis of a "working diagnosis" of pre-eclampsia. Dr Crichton's concerns were all for foetal health having regard to the results of the scan. She was not carrying out an assessment of maternal health and did

not examine the pursuer. However, she did give evidence that blood pressure could rise from normal to the levels seen in this case in a matter of minutes. A finding of 1+protein in the urine was not unusual towards the end of pregnancy. On the results recorded it was her opinion that the issue was primarily one of hypertension rather than pre-eclampsia. It was impossible to say what the blood pressure might have been on 5 October: it could have been normal then and abnormal on the Tuesday. The same applied to the issue of protein in the urine.

[107] Dr McLelland, said that the blood pressure recorded was one which would cause significant concern of the possibility of a stroke. She was concerned that this might be a case of pre-eclampsia so she ordered a series of tests which are routine in such cases: (a) a test to see whether foetal cells have been transferred to the maternal circulation; (b) kidney function tests; (c) urates; and (d) liver function tests. Blood count, clotting and kidney function were all normal, as were the urates. These at the least indicated that if this was a case of pre-eclampsia it had not compromised clotting or any major organs. She reviewed the results of the blood tests taken on 5 October and these too were all within normal limits. As to the question of proteinuria, a finding of 1+ protein in the urine may be spurious: alarm bells would only ring on a finding of 2 or 3+. A more detailed quantitative test would be required to confirm the diagnosis of pre-eclampsia, but the clinical picture was driving her to treat this as a case of pre-eclampsia.

[108] Dr Hanretty considered that the pursuer had extreme hypertension. The reading of 1+ proteinuria was not a diagnostic measurement but would rather indicate the need for either a repeat specimen or some attempt at quantification. The pursuer had exaggerated reflexes which he considered consistent with pre-eclampsia and his working diagnosis was one of pre-eclampsia. (Professor Draycott gave evidence that a finding of exaggerated

reflexes "definitely was not" significant, being very common in pregnant women, and as a test neither specific nor sensitive). He was "absolutely not" able to form a view as to what the pursuer's blood pressure would have been on 5 October (MS 2494). Pre-eclampsia was, an extremely unpredictable condition with a variety of manifestations. It was one of the absolutely unpredictable conditions. He went on to say:

> "Had there been a reason to take blood on the 5th … my presumption would be.. that they would be normal"

The general tenor of the evidence of the treating clinicians was thus that it was not possible to say either whether the pursuer's blood pressure would have been elevated on the Saturday, or whether a urine test would have rendered a positive result.

[109] The pursuer relied on the evidence of Professor de Swiet to argue that both tests would have been likely to show abnormalities on 5 October. Professor de Swiet's instructions had been to consider the likelihood of the pursuer having signs of pre-eclampsia (proteinuria and hypertension) when the midwives visited on 5 October (MS9491). Professor de Swiet's hypothesis was that since on 8 October the pursuer had high blood pressure and 1+ proteinuria, she probably had pre-eclampsia, from which it could be deduced that she would have had high blood pressure and proteinuria on the Saturday. He proceeded on the generally held belief amongst clinicians that significant proteinuria develops in women with pre-eclampsia about three weeks before delivery is necessary. He described this as a working rule to help obstetricians and midwives plan the care of the patient. In support of this, he relied upon a study from 1992, appended to his report as Appendix B (Chua and Redman, *Prognosis for pre-eclampsia complicated by 5g or more of proteinuria in 24 hours*, European Journal of Obstetrics & Gynaecology and Reproductive Biology, 43 (1992) pp 9-12). The precise sentence relied upon was that in the general case

"significant proteinuria greater than 0.5g over 24 hours usually appears about three weeks before mandatory delivery or intrauterine death". It should be noted that in the pursuer's case there was no 24-hour collection, there being only one catheter specimen reading of the pursuer's urine, recording 1+, taken on admission on 8 October.

[110] Professor de Swiet developed his thesis under reference to another study ("Redman") appended to his report as Appendix A (Redman, chp 6 *Hypertension* in de Swiet *Medical Disorders in Obstetric Practice* (4th edit, 2002)). This looked at 122 cases of symptomless proteinuric preeclampsia presenting in Oxford before 32 weeks in 1980 – 85. Professor de Swiet said that this study found that conservative management extended the life of the pregnancy beyond the onset of proteinuria by an average time of 15 days. In other words, on average, proteinuria would have been present 15 days before the time that delivery was necessary. On that basis, the pursuer would have had proteinuria for 15 days before she required delivery on 8 October. Proteinuria would have developed on 23 September, well before the midwives' visit of 5 October. Since delivery was necessary on 8 October, it can be said that it was likely that the pursuer had hypertension and proteinuria on 5 October.

[111] It should be noted that the Redman study was of symptomless proteinuric pre-eclampsia, whereas the pursuer's case relies on the presence of symptoms. In light of this, Professor de Swiet also drew on the Chua and Redman study. Apart from that issue of generality, that study examined 42 women with more severe disease. These women all had proteinuria in excess of 5g in a 24 hour urine collection at some time before delivery. Most had severe hypertension also. Fourteen required delivery within 3 days, 12 between 4 and 7 days, 9 between 8 and 11 days, 2 between 12 and 14 days and 5 after 14 days. The median timing of delivery, at which 50% of the women required delivery, was between 4 and 7 days

after presentation with symptoms. Thus proteinuria would have been present for more than 4 days before she needed delivery on 8 October, present by at least 4 October; it could have been present by 1 October based on these median figures.

[112] Professor de Swiet also considered an alternative approach, based on the interval between the development of gestational hypertension and that of pre-eclampsia. Gestational hypertension was hypertension first manifest in pregnancy without proteinuria. Pre-eclampsia was then defined as new onset hypertension and proteinuria. Most, but not all, women who develop pre-eclampsia go through a period of hypertension alone without proteinuria. Gestational hypertension is usually a milder disease than pre-eclampsia.

[113] Professor de Swiet referred to a study (Saudan et al, *Does gestational hypertension become pre-eclampsia?*, British Journal of Obstetrics & Gynaecology, Nov 1998 vol 105 pp1177-1184) of 845 women with gestational hypertension, of whom 42% presenting before 30 weeks developed pre-eclampsia. The median time interval between presentation with gestational hypertension and pre-eclampsia was 33 days. When the pursuer's blood pressure was last checked on 19 September it was normal, so she did not have gestational hypertension then. Therefore the earliest she could have developed gestational hypertension was 20 September (or perhaps later on 19 September). On the assumption that gestational hypertension developed at that time, the pursuer would not have developed pre-eclampsia until 33 days later, namely until 23 October. Professor de Swiet thought this study should be discounted as irrelevant to the pursuer's pregnancy but his reasons for saying so, other than that it did not fit the theory that she had pre-eclampsia on 8 October, are obscure. He did say that he considered it very likely that the patients in the Saudan study had much milder disease than did the pursuer and which progressed much more

slowly. However, the basis for such a conclusion is not easy to discern, and appears to us to be highly conjectural.

[114] Based on the Redman studies, Professor de Swiet concluded that it was highly likely that significant proteinuria would have been present in the pursuer's urine had it been tested on 5 October. Since high blood pressure usually appears before proteinuria in pre-eclampsia, it was also likely that she would have had hypertension on 5 October. Furthermore, symptoms are late features of pre-eclampsia usually appearing after the development of hypertension and proteinuria. Since the pursuer had symptoms, this is a further reason to suggest that she was likely to have had proteinuria and hypertension on 5 October.

[115] Professor Draycott's opinion was that, based on the very high level of the blood pressure on 8 October, it was likely that this would have been elevated on 5 October. He accepted that blood pressure could rise significantly over a relatively short period of time. He was unable to say over what period a rise to these levels might have taken place. It was certainly possible to occur over two days. He considered that it was not clear that there would have been significant proteinuria. He thought it was very difficult retrospectively to define the severity and duration of pre-eclampsia, since there was a spectrum of disorder, with no single way fitting all women. Trying to "retrofit" the kind of duration aspects of pre-eclampsia was very, very difficult. However, he did think the hypothesis of Professor de Swiet was well argued and convincing, although he accepted that it was a matter of conjecture. He thought it probable that the pursuer had been suffering from pre-eclampsia. She certainly required to be managed as if she did have that condition.

[116] Professor Walker's evidence was that high blood pressure in pregnancy did not mean that the patient had pre-eclampsia. Only about a third of woman with high blood

pressure would in fact have pre-eclampsia. Some women would have high blood pressure alone. It was the presence of proteinuria which enabled a diagnosis of pre-eclampsia to be offered. He confirmed the evidence of the clinicians that a finding of 1+ in the urine was of limited significance. A finding at this level was otherwise explicable, and was not above the threshold for classifying someone as having proteinuria. Had the symptoms complained of on 5 October related to pre-eclampsia, the proteinuria would have been significantly greater on admission. Nevertheless he supported the clinical decision to place the pursuer on the pre-eclampsia protocol as being an appropriate way to proceed.

[117] In his view, having regard to the whole picture, including the absence of abnormal blood or liver function tests, and the post-partum evidence of the pursuer's condition she did not in fact have pre-eclampsia at all. Most women with pre-eclampsia would get worse after delivery and this did not happen. In his opinion the pursuer did not have pre-eclampsia and on the Saturday it is likely that her blood pressure had been normal and she would not have had proteinuria. On 8 October she did have an acute elevation of blood pressure, easily controlled by a relatively low dose of labetalol, and did not require any treatment after delivery, although her blood pressure remained mildly elevated for some days. Women with established pre-eclampsia normally had ongoing problems after delivery, often with worsening signs and parameters. The duration of problems after delivery tends to reflect the degree and duration of problems prior to delivery. Since there were no problems after delivery, this suggested there were no longstanding problems prior to delivery. The 1+ proteinuria disappeared after delivery, suggesting it was transient, caused by catheterisation or even the existence of the high blood pressure itself. He said that

> "the absence of signs and symptoms and, particularly, blood parameters which suggests a pre-eclampsia on the Tuesday makes it unlikely that she had any pathology measureable on the Saturday."

[118] Had the pursuer suffered from elevated blood pressure on 5 October, causing her symptoms, she would have had worsening symptoms over the next few days. She would have had evidence of systemic involvement, abnormalities of liver function, elevated urate and low platelets with worsening symptoms after delivery, which did not result. The fact that her blood pressure fell after delivery suggested that the elevation of blood pressure was related to the underlying placental problem plus/minus the small abruption (MS12189). In all probability, the pursuer's blood pressure was normal on 5 October (MS12192). This fitted with the findings in the clinical notes and on admission.

[119] Professor Murphy considered that the diagnosis of pre-eclampsia was correct, although the maternal effects, apart from hypertension, were modest: it was a small element in the context of the bigger picture of ischaemic placental disease (IPD). Although in her report she felt the pre-eclampsia diagnosis was correct, in her evidence she said that she would not have focussed on this as a case of pre-eclampsia. It seemed that whilst she did not dispute a presumptive or working diagnosis of pre-eclampsia, to her, it was only a minor element within a more general pattern of ischaemic placental disease, the striking feature of which was severe intrauterine growth restriction (MS 8707).

[120] Like Professor Walker, Professor Murphy considered the proteinuria to be modest. She noted that the blood tests were entirely normal and the maternal postnatal recovery was almost immediate. She concluded that the pursuer's presentation on admission was not suggestive of a history of pre-eclampsia over the preceding days (MS11399).

[121] In the report to which she spoke in evidence she stated:

> "While Mrs H had marked hypertension on admission and +1 proteinuria, the clinical findings were not at all in keeping with severe maternal pre-eclampsia, and in particular the blood tests were entirely normal, her urine output was good and the

> hypertension resolved rapidly following minimal anti-hypertensive medication. I think it is highly speculative to suggest that the blood pressure and urine would have been abnormal on Saturday, 5th October."

If pressed to express a view as to what the blood pressure would have been, she would have said normal. Blood pressure can change over a very short period of time. The fact that all the blood tests were normal would not fit the usual pattern of early onset pre-eclampsia. However, she agreed that it had been correct to proceed on a working diagnosis of pre-eclampsia.

[122] In relation to the evidence of Professor de Swiet, Professor Walker thought the logic applied was confusing. It was generally understood that once a diagnosis of pre-eclampsia had been made, it was possible to delay delivery for a period of, on average, up to two weeks. However, that did not mean that it would be valid to start with the date of delivery and calculate back a period of two weeks and suggest that the indications of pre-eclampsia must then have been present. The matter should be looked at prospectively, not retrospectively, and much depended on how both the mother and the baby responded to treatment. The data did not suggest that one could work backwards. One may work forwards from diagnosis to delivery but not the reverse. The progress of the condition was highly variable between individuals, and the average was hugely varying. Furthermore, the evidence, in particular that she did not have any elevation of uric acid, that the level of protein in the urine was not significant, and that there was no evidence of a significantly low platelet count, showed that the pursuer did not come within the group of women upon whom the studies relied upon by Professor de Swiet had been based, in respect of whom these markers had been present.

[123] Like Professor Walker, Professor Murphy was highly critical of Professor de Swiet's reliance on an argument based on the potential interval from diagnosis to delivery in the

average patient. It was not possible to work backwards from a finding of 1+ protein in the urine on 8 October and conclude that the same level would have been present on 5 October. She considered such an approach to be "entirely speculative", and not consistent with the evidence which is that one patient may go from well to unwell in the same day, where another may continue with pre-eclampsia on a stable course for two or three weeks. It would be entirely speculative to suggest what the blood pressure and urine tests might have been on 5 October, but if pressed to speculate she would anticipate normal results.
To draw conclusions based on any so-called "average" amongst patients with such a variable condition would be a "potentially dangerous hypothesis". To work backwards from the known results of tests on 8 October was highly speculative.

Analysis and conclusions

[124] As the Lord Ordinary appreciated, there are several problems with Professor de Swiet's approach. First, the evidence does not suggest that the pursuer had significant proteinuria, at any stage, so to the extent that his evidence, or the studies upon which he relied, are based on such a supposition, his evidence is flawed. Secondly, the studies do not provide a firm basis from which to draw conclusions about the pursuer's case. Each was based on a small number of patients. One of them related to asymptomatic pre-eclampsia and the other to severe pre-eclampsia. It is not easy to place the pursuer in either category. In the Chua and Redman study, which addressed delivery after presentation with severe pre-eclampsia, the date thereafter at which delivery was required varied wildly amongst the 42 women studied, from 3 days to more than 14 days: to take a median of these from such a small study, and apply it to the pursuer, who did not have severe pre-eclampsia, is simply not valid. Third, his approach does not allow for the highly unpredictable nature of pre-

eclampsia. Fourth, his reasons for discounting the study which did not support his theory are far from persuasive. Fifth, the evidence of both Professor Walker and Professor Murphy was that such an approach took things backwards: it took prospective data and assumed retrospective results. Both gave very cogent reasons for rejecting Professor de Swiet's hypothesis. It was, as they both said, and the Lord Ordinary concluded, entirely speculative. The fundamental problem was that Professor de Swiet's analysis worked backwards from the time of delivery, but that time depended on a number of different factors, relating to both maternal and foetal condition, and was thus essentially arbitrary. This, it seems to us, is a crucial logical flaw in Professor de Swiet's analysis. We consider that the Lord Ordinary was correct to reject it. Its rejection also placed a query over the evidence of Professor Draycott, so far as his conclusions were drawn from Professor de Swiet's analysis. Without that, the highest that Professor Draycott's evidence came was his opinion that the pursuer would have had hypertension on 5 October. He could not say there would have been significant proteinuria. On the other hand, there was strong evidence from both Professors Walker and Murphy suggesting that the pursuer would not have had either elevated blood pressure or proteinuria on 5 October, based on their extensive clinical experience and the fact that the overall picture, including that existing prior to and post-delivery would not support such a conclusion. There were very good reasons for preferring their evidence on this issue, and whilst the Lord Ordinary's conclusions might again have been more fully expressed, we consider that he was amply justified, on the evidence, in reaching them.

[125] The argument that the pursuer must have had high blood pressure and protein in the urine on 5 October is predicated on several uncertainties. Primary amongst these are assumptions that she did in fact have pre-eclampsia on 5 October; and that it follows from

this that her blood pressure and urine tests would have been abnormal on that date. The evidence was clear that the diagnosis of pre-eclampsia was a "working" diagnosis and that the threshold for a true diagnosis had not been passed. There were several indicators which were not supportive of the diagnosis being the true one: the normal results from the blood, liver and kidney function tests, with the consequent highly satisfactory urine output; and the clinical picture post-partum. The date of delivery is in some senses an arbitrary matter, resulting from a specific medical decision which itself will turn on a range of factors which are not limited to, but may go well beyond, issues of proteinuria and hypertension. Seeking to draw conclusions retrospectively from such a starting point is not a sound basis from which one might establish the date when any symptoms first appeared. The result is that the pursuer failed to establish that she would have been admitted on 5 October on the basis of maternal symptoms. That leaves the issue of referral on the basis of foetal symptoms. The Lord Ordinary makes reference to the evidence of Professor Draycott as to what might have been expected from a scan or CTG carried out between 5 and 7 October. He also refers to the evidence of Dr Hanretty that he would have delivered the child earlier than 8 October because of the intrauterine growth restriction alone, even in the absence of signs of pre-eclampsia. The reason that the Lord Ordinary did not develop any analysis based upon this evidence or the potential consequences thereof, can only be that he accepted the defenders' submission that whether or not a CTG would have been undertaken was dependent on whether or not the patient was admitted.

[126] The Lord Ordinary concluded that the admission criteria would be abnormal blood pressure, abnormal urine test or assessment of fundal height estimated not in accordance with estimated gestation. Whether a CTG would have been carried out would have depended on whether the pursuer had been admitted, which in turn would have been based

only on these three admission "criteria". The extent of investigation of foetal health would have been a measurement of fundal height. Since this would have revealed no abnormality, admission would not have followed from the foetal symptoms. There was evidence to this effect from Dr Crichton, but that was in the context of an understanding that the pursuer had been referred for a variety of symptoms, which would include perceived lack of growth, and a normal number of movements but "fluttery". There was clear evidence that ultrasound would be the first line of investigation of a complaint of lack of growth, but CTG where the complaint was of a lack of movement. Dr Dr Crichton was not asked what would happen had the pursuer been referred by midwives specifically because they felt she needed a CTG. She confirmed that the first port of call for complaint of lack of movement would be CTG. Although there would have been an initial assessment by a midwife, what would happen thereafter would depend on all the findings.

[127] Dr McLelland explained that had the pursuer been referred to the admission suite on 5 October the midwife would do an antenatal check and, as part of an assessment of foetal wellbeing, she "would have had a heart rate tracing at the same time" (MS3311, line 18). This was confirmed by Professor Draycott (MS 2591).

[128] The evidence of the midwives – and for that matter other professional witnesses – was that the foetal symptoms of which the pursuer was complaining on 5 October merited referral for at least a CTG, and possibly medical assessment. It is not the case that medical assessment would inevitably have followed because of the referral having been made by a community midwife. There was clear evidence that the pursuer would have been triaged by a midwife, who would have tested fundal height. It is also likely, given the clear evidence from midwifery experts and Professor Draycott that the symptoms merited it, and that of Dr McLelland that it would have been part of the assessment by a midwife, that a CTG

would have been carried out. Professors Walker and Murphy both considered that a CTG would have been carried out. What that might have shown is not straight forward. The clinicians felt unable to say what it might have shown, since an intervening incident, such as an abruption, might have affected the picture.

[129] The following exchange took place during the evidence of Dr Crichton:

> "are you able .. to say what the CTG trace would have been like on the Saturday? – I don't think I could, no" (MS1725, line 21).
>
> "Would that be just guessing? – I think that would be guessing given the fact that people have found an abruption" (MS1726, line 1).
>
> "So is it possible that the CTG trace would have changed completely whenever the abruption took place? – It certainly has, could have had quite an impact on the CTG" (MS1726, line 4). "

She also considered it impossible to say what might have been the results of an ultrasound carried out on the Monday. The size of the baby and quantity of liquor would not have changed but the Doppler readings might have been different. If a trace had been broadly reassuring, the decision would probably have been to delay to allow the foetus to mature.

[130] Dr Hanretty gave evidence that, absent hypertension, with an admission on the Saturday, the baby would have been likely to be delivered earlier, probably on the Monday, but that was on the supposition that the foetal indications on the Monday were as they were on the Tuesday. Had the position on the Monday been as on the Tuesday, there would have been no merit in delaying for steroids to promote growth or strengthen the lungs, given the parlous state of the baby. However, as we have noted, what the picture actually might have been on the Monday was a matter about which there was considerable uncertainty. Elsewhere Dr Hanretty gave evidence that in the absence of hypertension, LW would have

been delivered within half an hour to one hour, ideally half an hour, of Dr Hanretty first having seen the pursuer at 15.10 on 8 October (MS 2413, line 11).

[131] There was also evidence from expert witnesses as to what might have been the position. To some extent, their views on this depended according to their own theories as to the cause of LW's cerebral palsy and other difficulties. Professor Draycott indicated that

> "The interval between first occurrence of absent end diastolic flow and an abnormal CTG by a physical profile is ranged from 1 to 26 days. Therefore, it's difficult to know whether the CTG would have been normal on 5 October" (MS2578, line 15).
>
> "'It's difficult to know exactly what the CTG would have shown, however, it is unlikely to have been completely normal.' Is that right ? – Correct." (MS 2593 line 7)
>
> "'The CTG may have been normal, but again it's likely there would have been at least intermittent decelerations consistent with cord compression, secondary to reduced liquor volume' – Yeah." (MS 2601, line 12).

Professor Draycott did however accept that a CTG on 5 October could have been normal. An ultrasound scan on the Saturday or Sunday would have been likely to show the same features as they take time to develop. It was unlikely that facilities would have been in place for a scan at the weekend. An ultrasound scan on the Monday would have prompted a course of steroids, with delivery being delayed to allow them to take effect.

[132] Professor Walker said he was "not particularly able to form a view about what a CTG might have shown on 5 October:

> "I would suspect that there would be some degree of abnormality on it because of the chronic nature of what's going on, but I can't tell you exactly what it looked like. The probability is that it wouldn't be exactly the same [as] it was on the 8th. It would probably be better than it was on the 8th, but probably wouldn't be completely normal, so exactly what it, what it was, I can't tell you" (MS7754, line 25)."
>
> "So what the CTG may or may not have been present on the 5th depends on the degree of chronicness of the condition and what happened between the 5th and the 8th, so if it was related to a chronic situation relating to placental dysfunction, then you'd probably have abnormality on the Saturday. If it was largely due to something happening after that, you may have actually relatively normal CTG, or not one with significant abnormality on it" (MS7756, line 1).

His general approach in circumstances such as these would be to delay delivery, a decision to be taken along with other clinicians. His conclusion on this matter was:

> "the probability is it probably was slightly better on the 5th, so you may well be with normal, with Doppler showing end-diastolic flow, you probably had a biophysical profile with some liquor there, so you may get a score of, say, three or four out of eight, CTG which was abnormal, but not very abnormal, you may well have delayed to let steroids take its effect. So decision to deliver would be dependent on what exactly was found at any given time." (MS 7776, line 15).

[133] Professor Murphy gave evidence that:

> "If scanned on the Monday and the findings of IUGR, oligohydramnios and abnormal Dopplers would have been apparent, she would have been admitted for corticosteroids, twice daily CTGs, every 12 hours, and close monitoring until such a time as the benefits of delivery outweighed the risks of prematurity." (MS11400).

[134] Drawing realistic conclusions from this evidence, on the balance of probabilities, is extremely difficult. Despite the reluctance of the clinicians to opine, one might, on the evidence of the experts be able to say on the balance of probabilities that a trace on the Saturday would have been unlikely to be normal. However, what might have followed from that is much more difficult to assess. The clinical decisions which would have followed from an abnormal or unreassuring trace on the Saturday, including whether an ultrasound scan would have been requested, would depend on exactly what the trace showed, and the nature of the whole clinical position at that time. Had a scan been requested, there was ample evidence that it would not have been likely to take place until sometime on the Monday. Although as it happened the pursuer had first a scan and then a CTG, that was because of the particular circumstances of her presentation at the department. There was very clear evidence that had she been referred in the usual way the order of occurrence would have been a CTG first, then, depending on what the CTG showed, an ultrasound scan not before the Monday. Had there been a scan on the Monday, it would

have been likely to show IUGR, and reduced liquor, but what the Doppler readings might have been remains uncertain. . There was a great deal of evidence that the risks of prematurity are such that wherever possible delivery would have been delayed and steroids given to promote growth and strengthen the lungs. As Professor Murphy said, and as was implicit in the evidence of other witnesses, delivery would have followed at the point where the benefits of delivery outweighed the risks of prematurity. That decision would require to be made on the basis of the combination of numerous factors and would have involved careful clinical assessment. On the state of the evidence (which was not perhaps as fully developed as it might have been on the question of what might have happened in the absence of hypertension and concerns for maternal health) it would be a speculative and hypothetical exercise to try to determine when, on balance, delivery might have followed. The pursuer requires to establish that delivery would have taken place earlier than it did. On the state of the evidence this is not a conclusion we are able to draw, on a balance of probabilities.

The second causation issue: review of the Lord Ordinary's decision

[135] The Lord Ordinary's treatment of the second causation issue is summarised at paragraphs [34] to [36] above. The basis of the Lord Ordinary's determination of the issue against the pursuer is set out at paragraph 136 of his opinion (see paragraph [36] above)

[136] The pursuer argues that paragraph 136 discloses a fundamental error in approach on the part of the Lord Ordinary. That there was expert opinion evidence contrary to that relied on by the pursuer that the Lord Ordinary had no reason to reject, did not necessarily mean that the Lord Ordinary could not be satisfied that the pursuer had proved her case. The decision of Lord Hodge in *Honisz* v *Lothian Health Board* 2008 SC 235 and the line of

authority of which it formed part had no application to issues of causation. The pursuer has further criticisms to make of the Lord Ordinary's treatment of the evidence he heard on the second causation issue (over some 26 days of court time and the subject of 166 pages of written submissions by the pursuer alone). She describes his comments on the matter as vague, ambiguous, utterly superficial and completely unsupported by anything resembling a careful analysis of the competing medical evidence or the parties' submissions. They come nowhere near the standard for adequate reasons. Harsh as these strictures may appear to be, we consider them to be justified. The Lord Ordinary has not provided a judicial decision on the second causation issue.

[137] The task of the judge of first instance in a case of disputed medical causation where parties rely on competing expert opinion is not an easy one. The very fact that opinion evidence has been admitted means that the issue has to be determined by reference to a field of knowledge to which the judge, uninstructed by a relevant expert, does not have access. Thus, where such a case is circumstantial, as it often will be and certainly is here, the extent to which the judge can rely on his general knowledge and experience as a reasonable man of the world in order to draw the necessary inferences is limited. He is dependent upon what the experts tell him about the relevant learning in their specialist fields. However, that does not mean that the judge hands over decision-making to the experts and in the event of a dispute among apparently well-qualified experts throw up his hands in despair. As Lord Hope explained in *Dingley*, the judge's task is to identify the real issues in the case and then determine where the balance lies between the competing positions revealed by the evidence on each side. An important part of that task is to assimilate and understand the oral and written evidence and to penetrate the arguments which have been developed by the expert witnesses. Having done that, the judge must make what is his decision on, for

example, a disputed question of medical causation. He will do so applying the appropriate standard, which in a civil case will usually be the balance of probabilities, in determining where the balance is to be struck. The qualification that it is for the judge to strike the balance is important. In *Dingley* Lord Hope warned that the judge must be careful to avoid applying the standard of proof that the expert would apply. Thus, it would be wrong for a judge to apply in the course of his decision-making the standard appropriate to a determination of scientific certainty but, equally, it would be wrong for the judge simply to adopt an expert's view of what in particular circumstance was likely or unlikely, probable or improbable. That would be to delegate to the witness the judge's task of deciding whether or not the case has been proved on the balance of probabilities. It is the judge's view of what is marginally likely to have happened which is important, not that of anyone else.

[138] That opinion evidence is to be evaluated rather than simply adopted, can be seen from the recent judgment in *Kennedy* v *Cordia (Services) LLP* 2016 SC (UKSC) 59 where the Supreme Court reviewed the admissibility and application of expert evidence in civil cases. At paragraph 48 of their opinion, with which the other members of the Court agreed, Lord Reed and Lord Hodge said this:

> "[48] An expert must explain the basis of his or her evidence when it is not personal observation or sensation; mere assertion or "bare *ipse dixit* " carries little weight, as the Lord President (Cooper) famously stated in *Davie* v *Magistrates of Edinburgh*, (p.40). If anything, the suggestion that an unsubstantiated *ipse dixit* carries little weight is understated; in our view such evidence is worthless. Wessels JA stated the matter well in the Supreme Court of South Africa (Appellate Division) in *Coopers (South Africa) (Pty) Ltd* v *Deutsche Gesellschaft für Schädlingsbekämpfung mbH*, (p.371):
>
> > '[A]n expert's opinion represents his reasoned conclusion based on certain facts or data, which are either common cause, or established by his own evidence or that of some other competent witness. Except possibly where it is not controverted, an expert's bald statement of his opinion is not of any real assistance. Proper evaluation of the opinion can only be undertaken if the process of reasoning which led to the conclusion, including the premises from which the reasoning proceeds, are disclosed by the expert.'

> As Lord Prosser pithily stated in *Dingley* v *Chief Constable, Strathclyde Police*, at (p.604): 'As with judicial or other opinions, what carries weight is the reasoning, not the conclusion.'"

[139] It would appear from paragraph 136 of his opinion that the Lord Ordinary has not really understood this. He certainly has not applied it. It is one thing to say, after a full evaluation of everything that has been led, that because of the cogency of the evidence adduced in support of an alternative possible explanation for LW's cerebral palsy, the pursuer's proposed explanation has not been established to the Lord Ordinary's satisfaction on a balance of probabilities. It is another thing to say that simply because there was cogent evidence in support of an alternative possibility which the Lord Ordinary had "no objective reason to reject" (whatever precisely that may mean), he was unable to find that the pursuer had proved her case. The Lord Ordinary's reference to *Honisz* suggests that it is the second of these propositions that he was advancing, in which case he was wrong.

[140] The passage in *Honisz* which the Lord Ordinary evidently had in mind is found at paragraphs 38 and 39 of Lord Hodge's opinion which are as follows:

> "[38] The main area of contention between parties as to the law was what was the proper approach for the court to take to the evidence of the consultant orthopaedic surgeons led by the defenders that they would have adopted the same practices as those which the consultants and senior registrars, against whom negligence is alleged, in fact adopted. Again, however, the matter is one decided by authority which may be summarised briefly in the following propositions.
>
> [39] First, as a general rule, where there are two opposing schools of thought among the relevant group of responsible medical practitioners as to the appropriateness of a particular practice, it is not the function of the court to prefer one school over the other (*Maynard* v *West Midlands Regional Health Authority*, Lord Scarman, p 639F–G). Secondly, however, the court does not defer to the opinion of the relevant professionals to the extent that, if a defender lead evidence that other responsible professionals among the relevant group of medical practitioners would have done what the impugned medical practitioner did, the judge must in all cases conclude that there has been no negligence. This is because, thirdly, in exceptional cases the court may conclude that a practice which responsible medical practitioners have perpetuated does not stand up to rational analysis (*Bolitho*

> v *City and Hackney Health Authority*, Lord Browne-Wilkinson, pp 241G–242F, 243A–E). Where the judge is satisfied that the body of professional opinion, on which a defender relies, is not reasonable or responsible he may find the medical practitioner guilty of negligence, despite that body of opinion sanctioning his conduct. This will rarely occur as the assessment and balancing of risks and benefits are matters of clinical judgment. Thus it will normally require compelling expert evidence to demonstrate that an opinion by another medical expert is one which that other expert could not have held if he had taken care to analyse the basis of the practice. Where experts have applied their minds to the comparative risks and benefits of a course of action and have reached a defensible conclusion, the court will have no basis for rejecting their view and concluding that the pursuer has proved negligence in terms of the *Hunter* v *Hanley* test (see para 36). As Lord Browne-Wilkinson said in *Bolitho* (p 243D–E), 'it is only where the judge can be satisfied that the body of expert opinion cannot logically be supported at all that such opinion will not provide the benchmark by which the defendant's conduct falls to be assessed.'"

What Lord Hodge is addressing here is the question of whether a particular clinical practice, which has been subject to criticism by some witnesses but which nevertheless is supported by a responsible body of medical opinion can be held to be negligent. What he says has no application to the determination of a question of disputed fact, which is what is in issue here. We accordingly accept the pursuer's submission that the Lord Ordinary's approach to the determination of the second causation issue proceeded upon an error of law.

[141] As we have already indicated, there are other criticisms of the Lord Ordinary's treatment of the second causation issue. We take the superficiality of his approach to be due, at least partly, to the fact that, as he explained at paragraph 134 of his opinion, he considered that, strictly, there was no requirement for determination of the issue of the causation of LW's admitted cerebral palsy because he had determined the first causation issue against the pursuer. That of course only applies as long as parties are content to accept the Lord Ordinary's decision. Here, as is not uncommon, parties have not been content to accept the Lord Ordinary's decision and yet they, and this Court, have been provided with only three paragraphs to explain what the Lord Ordinary made of what the pursuer in her note of argument states were 26 days of evidence out of a total 55 days of proof on which

she presented 166 pages of written submissions. This has at least two consequences. First, there has been a failure in a duty owed to the parties. The parties, their representatives and the many expert witnesses they led, clearly had invested very considerable efforts in putting their respective cases to the court. They were entitled to the assurance that these cases had been heard and understood. The Lord Ordinary's opinion does not give that assurance in so far as the second causation issue is concerned. Second, there has been a failure in a duty owed to this Court. Against the eventuality that parties will not be content to accept the decision of the judge of first instance, subject to considerations of proportionality, it is proper practice for that judge to provide a fully analysed and reasoned decision on every material issue which has been argued before him. A commonplace example of a first instance judge doing that is when, in a reparation action, he provides a fully reasoned and quantified decision on damages albeit he has assoilzied the defender. The reasons for this are not hard to see. First, it is the job and therefore the duty of a judge who hears evidence to evaluate that evidence and to make findings in fact where these findings are relevant to an issue which it is necessary to determine at first instance or which it might become necessary to determine on appeal. Second, any rational system of decision-making must seek to make the most efficient and effective use of its resources. The judge who has heard the evidence is the person best able to evaluate that evidence and to provide any appeal court with the benefit of that evaluation in a form which allows the appeal court to do its (different) job. These points are made by Lord Hodge at paragraph 22 in *Royal Bank of Scotland plc* v *Carlyle* 2015 SC (UKSC) 93:

> "It is the first instance judge who is assigned the task of determining the facts, not the appeal court. The reopening of all questions of fact for redetermination on appeal would expose parties to great cost and divert judicial resources for what would often be negligible no benefit in terms of factual accuracy. It is likely that the judge who has heard the evidence over an extended period will have a greater familiarity with

> the evidence and a deeper insight in reaching conclusions of fact than an appeal court whose perception may be narrowed or even distorted by the focused challenge to particular parts of the evidence."

Where a first instance judge does his job properly, making the necessary findings of fact and fully explaining why he decided as he has, it is unlikely that the appellate court will consider it appropriate to reopen questions of fact for what Lord Hodge describes as the "negligible benefit in terms of factual accuracy". However, where the appellate court is prepared to entertain an appeal on the facts, full and reasoned findings by the first instance court will make easier both its task and the task of parties appearing before it. In the present case, in contrast, the Lord Ordinary has made no attempt to provide this Court with the benefit of his familiarity with and his insight into 26 days of evidence. That is a significant loss. It has meant that this Court has had to start from scratch in understanding what this aspect of the case is about. With more than 15,000 pages of material to consider that has not been a negligible task. This is not just a complaint about the imposition of a great deal of unnecessary work, although it is that in part. It is a protest about a waste of resources and consequential inefficient decision-making.

[142] The second causation issue being at large, we therefore turn to consider it.

The second causation issue and how it may be approached

[143] At the beginning of the pursuer's note of argument ("PNoA") there is a summary of the issues arising in the reclaiming motion. There it is explained that: "LW has cerebral palsy. The pursuer's case is that this was caused by brain injuries sustained by LW in an acute hypoxic ischaemic insult ('AHII') which began several minutes before he was born and which ended when he was about six minutes old." Thus, LW's cerebral palsy is the damage in respect of which the pursuer sues. The injury which she avers caused this damage was an

AHII said to occur during a period of minutes on either side of LW's delivery by caesarean section. Timing is important because the pursuer seeks to prove that the damaging event would have been avoided had the pursuer undergone an earlier caesarean section. The defender disputes that LW did in fact suffer damage by reason of such a mechanism.

[144] As we have understood it, the second causation issue involves a number of inter-related questions, prefaced by "Has the pursuer proved …". Two of these questions are overarching: has the pursuer proved the occurrence of an AHII ending at the age of six minutes?; and has the pursuer proved that such an AHII caused LW's cerebral palsy? These questions require to be addressed from the various potentially interlinking perspectives provided by the extensive evidence led at proof from practitioners in a number of medical disciplines. In the pursuer's written submissions in the Outer House (Pursuer's Outer House Submissions –"POHS") (MS 12626) she identifies five such perspectives or criteria for the assessment of whether a child has developed cerebral palsy as a result of an AHII. Her argument is that the evidence in respect of these should lead to the conclusion that LW suffered an AHII which resulted in his cerebral palsy. The perspectives are drawn from the report of the pursuer's neurological expert, Dr Colin Ferrie, and are as follows: (1) the condition of the unborn child in the period leading up to delivery as revealed by the obstetric records including CTG traces; (2) the condition of the baby at birth with particular reference to Apgar scores and the need for resuscitation; (3) the neonatal condition of the baby including the presence or absence of hypoxic ischaemic encephalopathy and/or multisystem hypoxic damage; (4) the type of disability which develops in the child – dystonic/spastic, diplegic/quadriplegic etc – whether this is compatible with an AHII; and (5) the exclusion of other potential causes. To these perspectives the pursuer would add, and indeed would stress the importance of, what is revealed on such brain imaging as is

available. We shall examine each of these perspectives, albeit not in precisely the same order as that set out by the pursuer.

[145] Whether the Lord Ordinary shared our understanding of the extent of the second causation issue is not entirely clear. The Lord Ordinary discusses causation of LW's cerebral palsy at paragraphs 119 to 132 and 134 to 136 of his opinion. The opening paragraph of that passage is in the following terms:

> "[119] There is no dispute that LW suffers from cerebral palsy. The exact nature of LW's cerebral palsy and the extent of his disabilities as a result thereof are the subject of dispute but that issue was not to any material extent the focus of evidence in a proof limited to issues of causation. There was, further, no dispute that LW had sustained an insult to his brain which was causative of his deficits and which had first been clinically determined by MRI scan performed by Dr Zuberi in September 2002. The issue was not whether the insult to the brain was causative of LW's deficits but whether the cause of the brain insult was an event or events for which the defenders were responsible as a result of negligence on the part of their employees, the midwives."

Paragraph 134 includes this:

> "The pursuer's case is critically dependent on the cause of LW's disability being a single event, that being an acute ischaemic hypoxic insult commencing at some time immediately prior to delivery and ending on his resuscitation 6 minutes after delivery. The occurrence of such an event is not in doubt, albeit its timing is. "

[146] The content of other paragraphs of the Lord Ordinary's opinion are not entirely consistent with what appears in paragraphs 119 and 134 and in any event the Lord Ordinary's understanding of the issue becomes less important once matters are at large for this Court. However, by way of clearing the ground, while we would agree that there is no dispute that LW suffers from cerebral palsy, although this was not explored in very great detail in evidence, the exact nature of his cerebral palsy and the extent of LW's disabilities are in dispute (as the Lord Ordinary noted). The Lord Ordinary's expression "insult to his brain" might of course mean a number of things, but if it means an AHII commencing *in utero*, as posited by the pursuer, then there is a dispute as to whether such an insult in fact

occurred and, separately, whether, assuming that it did occur, it was causative of the relevant deficits or causative of what is demonstrated by the MRI scan performed in 2002. If on the other hand the expression means a period of hypoxia subsequent to delivery and lasting some six minutes during which LW was being actively resuscitated, then there is no dispute that such an event occurred and was potentially damaging but neither party contends that it and it alone caused LW's deficits.

Structure and functioning of the brain

[147] The pursuer alleges that LW suffered injury to his brain. Therefore, before going further, it is convenient to set out what is our understanding of the evidence relating to the structure and functioning of the human brain. It is no doubt superficial. It concentrates on the parts of the brain which were discussed in the evidence.

[148] The brain consists of four principal parts: brain stem, cerebellum, diencephalon and cerebrum. The brain stem is continuous with the spinal cord. It consists of the medulla oblongata, the pons and the midbrain. The medulla continues the spinal cord and contains the motor tracts through which connection is made between the brain and the spinal cord and thence to the muscles, allowing the regulation of movement. Broadly speaking, the cerebellum lies behind the brain stem, the diencephalon lies above the brain stem and the cerebrum lies above and overlaps the other three parts. Viewed from above or below the cerebellum can be described as having an appearance resembling a butterfly. The central area is the vermis and the lateral lobes (the "wings of the butterfly") are the cerebellar hemispheres. The diencephalon comprises the thalamus, the hypothalamus, the epithalamus and the pineal gland. The cerebrum lies above and spreads over the diencephalon and cerebellum, occupying most of the skull cavity. The superficial outer

layer of the cerebellum is referred to as the cerebral cortex. The cerebral cortex is between 2 and 4 millimetres thick. It is folded in upon itself. A fold is termed a gyrus. A shallower groove between folds is referred to as a sulcus. The central sulcus separates (transversely) the frontal lobe of the cerebrum from the parietal lobe. A major gyrus, the precentral gyrus, is located immediately forward from the central sulcus. Immediately behind the central sulcus is the postcentral gyrus. This area on either side of the central sulcus is commonly called the parasagittal area. The deeper grooves between folds are known as fissures. The most prominent fissure, the longitudinal fissure, separates the cerebrum into a right half and a left half: the right and left cerebral hemispheres. The hemispheres are, however, connected internally by a large bundle of transverse fibres composed of white matter (see below) called the corpus callosum. By far the greatest part of the cortex is termed the neocortex and is associated with higher cognitive function.

[149] There are two types of cells which make up the nervous tissue of the brain, neurons and neuroglia. Neurons receive and transmit the nerve impulses by means of which the brain carries out its mental, sensory and motor functions. Neurons are connected to each other and to other cells, including muscle cells, by elongated extensions referred to as processes. Dendrites are processes which receive impulses. Axons are processes which transmit impulses. Axons may have a covering of myelin which insulates and therefore makes more effective the ability of the process to transmit a nerve impulse. Collectively, dendrites and axons may be referred to as nerve fibres.

[150] In discussing the tissue comprising the component parts of the brain, it is usual to distinguish between the grey matter and the white matter. The difference in colour is readily apparent on dissection of the brain and is due, on the one hand, to the pale myelin coating of the axonal processes in the white matter, and on the other, to the relative absence

of myelin in the (darker) grey matter. The cerebral cortex is formed from layers of grey matter. White matter lies below each of these layers. The white matter of the cerebrum, underlying the cerebral cortex, consists of myelinated and un-myelinated axons organised into tracts consisting of three principal types of fibres. One of these groups is that of the commissural fibres which transmit impulses from the gyri in one cerebral hemisphere to the corresponding gyri in the other cerebral hemisphere. The corpus callosum is one of these groups of commissural fibres.

[151] When describing the component parts of the brain the expression "nucleus" is used to refer to an identifiable cluster of cell bodies. The basal ganglia may be so described; they are several pairs of nuclei, the respective members of each pair being situated in opposite cerebral hemispheres. They are composed of bodies of grey matter embedded within the white matter of the cerebrum (they may be described as deep grey matter nuclei – MS 1525). Component parts of the basal ganglia are the caudate nucleus and the lentiform (otherwise lenticular) nucleus. Each lentiform nucleus is divided into a lateral part called the putamen (plural putamina) and a medial part called the globus pallidus.

[152] Within the brain there are four cavities or ventricles filled with cerebrospinal fluid. A lateral ventricle is located in each hemisphere of the cerebrum. The third ventricle is a narrow cavity along the midline above the hypothalamus and between the right and left halves of the thalamus. The fourth ventricle lies between the brain stem and the cerebellum. In that the ventricles are cavities within the brain, they will become enlarged in the event of loss of brain tissue. Areas of white matter which surround the ventricles may be described as periventricular. If deprived of oxygen this periventricular white matter may suffer damage in the form of softening, otherwise referred to as leukomalacia or periventricular leukomalacia ("PVL").

[153] To an extent, particular areas of the brain are associated with discrete functions. For example, certain areas of the cerebrum (and also the thalamus) are associated with intellectual processes and cognition. However, the function of the brain in controlling posture and movement (otherwise the motor function) is complex. It involves several regions of the brain and utilises both pyramidal (or direct) and the extrapyramidal (or indirect) pathways. The primary motor area of the cerebral cortex, otherwise the motor cortex, is located in the precentral gyrus of the frontal lobe of the cerebrum. It is this which controls voluntary contractions of specific muscles or groups of muscles. The nerve impulses which exercise this control are conveyed by upper motor neurons, the axons of which descend through the cerebrum and from there to muscles by way of the medulla oblongata where bundles of these axons form bulges known as pyramids (hence pyramidal pathways). However, this voluntary control is supplemented, modulated and inhibited by nerve impulses conducted along extrapyramidal pathways involving, *inter alia*, the basal ganglia, the thalamus and the cerebellum. The degradation of these structures in an individual will impair, to a greater or lesser extent, his ability to move smoothly and to hold himself erect while at rest (cf description of dystonia by Dr Kendall - MS 1465 to 1466).

LW's condition – what is averred to be the damage

[154] LW was delivered pre-term, at 32 weeks' gestation. His birth weight was 920 grams. That is a very low weight even for a baby of 32 weeks gestation. His head circumference was on the second centile for babies of 32 weeks gestation (MS 6403). The pursuer accepts that LW suffered from IUGR due to placental insufficiency (MS 12649), but maintains that any neurological impairment resulting from low birth weight and/or prematurity is slight, and would not have prevented LW proceeding to higher education, obtaining employment

and living a full life. That is what would have been the case, so the pursuer avers, had LW "been born before the likely circulatory collapse after 1527 hours [on 8 October 1996]" (MS 46).

[155] The damage in respect of which the pursuer sues is summarised as being cerebral palsy with accompanying evidence of delayed motor development and delayed overall development (MS 34). It is averred that LW has a mixed (dyskinetic/spastic) quadriplegic palsy of dyskinetic type; severe learning difficulties and behavioural problems; obsessions, compulsions and anxiety; difficulties with movement and speech; marked spasticity in his lower legs and marked tightness in his hamstring muscles. It is said that his hand movements are clumsy and not fully coordinated; that he requires assistance with toileting and bathing; that he has some retention of intellectual abilities and is able to read and spell and has a simple grasp of numbers; that he requires full assistance in all aspects of daily living; and that he constantly demands attention and reassurance and demonstrates obsessional and repetitive behaviour.

[156] The pursuer avers in Condescendence VI that the injury which caused this damage was an AHII consequential on circulatory collapse lasting longer than ten minutes and occurring in the period of about 30 minutes leading up to the birth or to the point where resuscitation became effective shortly thereafter. She avers that the physical effects on LW's brain have been demonstrated on magnetic resonance imaging ("MRI") and that the MRI scan is consistent with what appeared on ultrasound imaging which was carried out on 9, 14, and 23 October 1996, there being neither significant change in the periventricular white matter nor cysts.

[157] In response, the defender makes no formal admissions beyond that LW was born in poor condition, but it is apparent from the pleadings that there is a certain amount of

common ground between the parties. The defender accepts that LW suffers from cerebral palsy (described by the defender as a diplegia, the significance of which is discussed below). It also accepts that damage to LW's brain, including damage to the basal ganglia, is shown on MRI; that LW suffered from IUGR; and that there was evidence at delivery of a retroplacental clot of 25 per cent of the placental area. The defender does not on the other hand accept that LW suffered any injury as a result of an AHII. However, if that is wrong, it is the defender's position that any acute hypoxic injury suffered by LW subsequent to 5 October 1996 was minor in nature and has not affected his subsequent symptoms.

Neurological evidence as to damage: the nature of LW's disabilities

[158] The pursuer relies principally upon the evidence of Dr Colin Ferrie, paediatric neurologist, for the identification of the nature and extent of LW'S disabilities; their proper description and classification and the recognised associations between these disabilities and certain sorts of brain damage. The defender relies principally on the evidence of Dr Martin Kirkpatrick, also a paediatric neurologist. Dr Ferrie examined LW on 27 March 2012 in the offices of the solicitors instructed by the pursuer. He prepared a report, dated 3 March 2013 (6/18 of process MS 9518). Dr Kirkpatrick's report is dated 16 October 2011 (7/13 of process MS 12146). It followed on a meeting with LW and his family on 30 July 2011. The pursuer was cross-examined under particular reference to paragraphs 6 and 7 of Dr Kirkpatrick's report with a view to her confirming the accuracy of Dr Kirkpatrick's understanding of LW'S condition (MS 866)

[159] Dr Ferrie and Dr Kirkpatrick agreed that LW has cerebral palsy (explained by Dr Ferrie as a term used to denote a group of conditions characterised by abnormalities of movement and posture caused by non-progressive disorders of the developing brain),

learning difficulties and behavioural problems. LW therefore has disabilities falling into three distinct groups: one relating to movement and posture; one relating to intellectual functioning; and one relating to behaviour. In paragraphs 3.7 and 3.10 of his report Dr Ferrie explains that because both conditions arise as a consequence of damage to or dysfunction of the brain, cerebral palsy and learning difficulties commonly co-exist. He further explains that behavioural problems, of various types, are very common in children with both cerebral palsy and learning difficulties. Dr Ferrie's report thus acknowledges that LW's various disabilities may arise from different causes. That is not the pursuer's primary position. Her case is that all of LW's cognitive deficits and behavioural difficulties, as well as his motor disabilities, are the result of a single AHII. However, that requires her to concede that taking LW's disabilities as a whole they are not the most typical presentation for a child with cerebral palsy from such a cause (MS 12724). That was accepted by Dr Ferrie in cross-examination (MS 2283) and also reflects the view of Dr Kirkpatrick (MS 6936). He considered that LW's learning difficulties and his behavioural issues were "a very unusual pattern to see" and that there was a mismatch between the severity of his learning difficulties and the severity of his diplegia.

[160] Dr Kirkpatrick accepted that LW had sustained basal ganglia damage but he did not accept that this had caused LW's learning disability or behavioural problems (MS 6934, 7164).

[161] As we have already observed, while it is not controversial that LW suffers from cerebral palsy, a question arises as to how LW's condition should be classified. Depending on the exact nature of the abnormality of movement and posture, cerebral palsy falls into different types (Dr Kirkpatrick - MS 6856 to 6859). Spastic cerebral palsy is characterised by an increase in muscle tone. On examination one also expects to find features such as

exaggerated tendon reflexes, ankle clonus (spasm) and extensor plantar responses. Dyskinetic (also called dystonic, extrapyramidal and athetoid) cerebral palsy is characterised by marked fluctuation in muscle tone and consequent difficulty in controlling movement, according to factors such as position and emotion (dystonia) also often by the occurrence of abnormal writhing type movements (athetosis). Dystonia indicates difficulty in making smooth movements (MS 1464, MS 1568). It can be defined as a movement disorder characterised by sustained or repetitive muscle contractions resulting in twisting or writhing or abnormal fixed positions. In ataxic cerebral palsy the principal problem is with balance. Cerebral palsy can also be classified according to the distribution of affected limbs. In quadriplegic cerebral palsy all four limbs are involved to a significant extent but with the arms more severely affected. In diplegic cerebral palsy the lower limbs are mainly or exclusively involved. In hemiplegic cerebral palsy one side of the body is affected, usually with the upper limb being more severely affected than the lower limb. Finally, the degree of functional impact of the cerebral palsy can be used to classify it as mild, moderate or severe.

[162] As appears from his report, it was Dr Ferrie's opinion that LW's cerebral palsy is quadriplegic in distribution but with his lower limbs more severely affected than his upper limbs. The predominant feature is dystonia, indicating that his cerebral palsy is predominantly dyskinetic in type. However, his tendon reflexes are excessively brisk, indicating a degree of spasticity (MS 9544 to 9545). Given that LW is independently ambulant, albeit with some difficulty, Dr Ferrie would classify his cerebral palsy as mild. There is significant involvement of the bulbar muscles (the muscles required for chewing, swallowing and the articulation of speech).

[163] Dr Ferrie associated dyskinetic cerebral palsy with an acute hypoxic event (which from paragraph 3.56 of his report is to be understood as a complete deprivation of oxygen

for a period not exceeding 20 minutes) and consequent damage to the basal ganglia and thalami, whereas he associated cerebral palsy with both dyskinetic and spastic features with more chronic hypoxic events (chronic partial hypoxic insults) and consequent wider damage (MS 9558). Dr Ferrie concluded that LW's condition (predominantly dyskinetic quadriplegic cerebral palsy with some spasticity and significant learning difficulties and behavioural problems) was consistent with that to be expected following an AHII immediately prior to delivery (report paragraph 3.33 - MS 9552). LW's neurodevelopmental problems were not attributable to placental insufficiency or a chronic hypoxic event. If either of those had been the cause, his MRI scan would have shown a different form of damage, namely, periventricular leukomalacia (PVL) and his cerebral palsy would have been spastic diplegic in type (report paragraphs 3.42 and 3.57 and MS 1824 and 1829, 9554 and 9558).

[164] At this point, we observe parenthetically that although Dr Ferrie seemed quite clear when giving evidence that the absence of PVL pointed to the damage having been the result of acute as opposed to chronic hypoxia, in his report he concedes that "the situation is more complicated in the premature baby, since it is known that such insults [ie those which are relatively long] sometimes give rise to PVL but in other instances give rise to damage to the basal ganglia and thalami" (report paragraph 3.56, MS 9558). This would indicate that PVL is not always found in cases of chronic hypoxia. The significance or otherwise of PVL not having been identified on MRI scan is complicated when consideration is given to the evidence of Dr Connolly, as discussed below. If we have understood that evidence it was to the effect that LW's case was an unusual case of hypoxic brain damage in not demonstrating PVL given that he was at only 32 weeks gestation when delivered. In the passages to which we refer below we do not see Dr Connolly to distinguish between cases of chronic hypoxia and acute hypoxia.

[165] In evidence, Dr Ferrie explained that the most significant reason for his view as to there being a degree of spasticity was LW's foot deformities which were due to joint contractures, and joint contractures were more a feature of spastic forms of cerebral palsy (MS 1799). To an extent, he agreed with Dr Kirkpatrick's description of LW's cerebral palsy as "best described as mixed spastic dyskinetic diplegia". The principal difference between Dr Ferrie and Dr Kirkpatrick was that Dr Ferrie considered LW to have quadriplegia, not diplegia. He thought the upper limbs were significantly involved, although he acknowledged that this question of upper limb involvement to the extent justifying a diagnosis of quadriplegia was one on which neurologists might differ (cf Dr Kirkpatrick's opinion – MS 1804 to 1805, 2207, 2210). In cross examination, Dr Ferrie maintained his opinion. He considered that there was very little between himself and Dr Kirkpatrick in the detail of what they each found (MS 2216). The principal difference between them was that Dr Kirkpatrick thought that LW's cerebral palsy was best described as a mixed spastic dyskinetic diplegia (MS 2217), whereas Dr Ferrie's classification was a mixed spastic dyskinetic quadriplegia because of what he saw as significant upper limb movement (MS 2217). Dr Ferrie also considered that Dr Kirkpatrick underplayed the significance of dyskinesia. Dr Ferrie thought the dyskinesia was significant given the bulbar muscle involvement (MS 2217, 1808). In addition, he thought that Dr Kirkpatrick attributed the motor problems in LW's legs to spasticity. Dr Ferrie disagreed with that. The history which he had obtained from LW's parents, his own findings on examination and his assessment of the clinical records pointed to there being considerable variability in the degree of stiffness and that was more consistent with dyskinesia (MS 2255).

[166] Dr Kirkpatrick's assessment was that LW has signs of variable spasticity in his lower limbs with brisk tendon reflexes and up-going plantar responses in keeping with diplegic

cerebral palsy (critically LW's lower limbs are more severely affected than his upper limbs – MS 7100). There are no signs of fixed contracture at his hips or knees. He has very marked varus deformity of both his ankles and a degree of fixed equinus deformity such that both his feet are twisted on their outer edges. In Dr Kirkpatrick's opinion LW's motor disability would be best described as a mixed spastic/dyskinetic diplegia. He did not believe that LW's was a case of quadriplegic cerebral palsy (MS 6904 to 6914). Dr Kirkpatrick ascribed the element of dyskinesia (awkward movement) to basal ganglia damage but not LW's cognitive impairment nor his behavioural problems (MS 6933, 7138). On the assumption that it were to be found that there had been a perinatal AHII which had caused the damage to the putamina identified on MRI, Dr Kirkpatrick was asked to consider what would be the difference to LW's condition had the AHII not occurred. According to Dr Kirkpatrick LW would not then suffer from dyskinesia, his upper limb movement would be improved, it is conceivable that his swallowing difficulties would be less apparent and his lower limb function might be improved to a minimal extent. He would however still suffer from spastic diplegia, he would still have behavioural issues, he would still have issues relating to anxiety, he would still have obsessive-compulsive issues and he would still have learning difficulties (MS 7096).

[167] Although he was not led as an expert, one of the treating clinicians, Dr Zuberi, was asked how he would classify LW's cerebral palsy. His answer was "spastic dystonic cerebral palsy mainly affecting his lower limbs". The follow-up question whether that would be typical of a basal ganglia damaged child to which he answered: "Not typically, no. Typically, a child with a basal ganglia injury would have a dyskinetic cerebral palsy with choreoathetosis [writhing and twisting movements of the limbs], if it was just simply the basal ganglia that were involved. Basal ganglia plus other parts of the brain, then you have

dystonia and spasticity" (MS 6115). When asked what would be the effect of basal ganglia injury in isolation on cognitive function, his response was that in such cases cognitive function is relatively well preserved; individuals can have normal or near normal cognition (MS 6116). These answers were alluded to in cross-examination but not challenged (MS 6133). Dr Zuberi confirmed to the cross-examiner that the appearance of LW's MRI scan was consistent with him having experienced a period, even a relatively short period, when his brain had a relatively reduced blood flow and oxygen supply (MS 6135).

[168] Something that we shall have occasion to repeat is that we do not consider it necessary that a particular strand or element within the evidence must be established to our satisfaction on a balance of probabilities before it can be taken into account in determining whether the pursuer has proved her case. Regard may be had to possibilities. Moreover, when looking at a particular area of evidence it will often be appropriate to take no more than a provisional view of what it amounts to, deferring reaching a final view (if that ever proves possible) until it can be seen in the context of the whole evidence of the case. However, while keeping that firmly in mind, it will be convenient, in the course of our summaries of particular chapters of the evidence, to indicate from time to time what our understanding of what that evidence is.

[169] What we take from this chapter of evidence is that LW suffers from a movement disorder properly described as cerebral palsy. However, cerebral palsy is not a discrete condition. Rather, it is the description applied to a group of conditions with different characteristics. Just precisely where LW's condition fits within that group is a matter of judgement as to which competent neurologists might differ. One possible cause of cerebral palsy is damage to structures within the brain consequent on deprivation of oxygen (hypoxia). There are other possible causes (see below). A distinction can be made between

acute and complete deprivation of oxygen on the one hand (an AHII) and a chronic partial deprivation of oxygen on the other. These different sorts of event are associated with different outcomes. For example, quadriplegic involvement is associated with there having been an AHII, whereas diplegic involvement is associated with there having been a chronic event.

[170] It is to anticipate what was disclosed by the neuro-radiological evidence, which is discussed below, but we see it to be uncontroversial that LW's basal ganglia, and particularly the putamina, have suffered damage and that a possible mechanism for that damage is hypoxic insult. Basal ganglia damage would explain some but not all aspects of LW's movement disorder. It would not explain his cognitive deficits or his behavioural problems. Thus, a comprehensive explanation of LW's condition must lie in there having been damage to (hypoxic or otherwise) or developmental failure of other structures within the brain. If the explanation or cause was an AHII, as Dr Ferrie argued that it was, that would seem to point to a longer rather than a shorter acute event, as described by Dr Ferrie at paragraphs 3.56 to 3.59 of his report (and see MS 1861 to 1864). What Dr Ferrie envisaged as a longer event ("relatively long" as he puts it) when giving evidence was something of the order of 20 minutes of complete deprivation of oxygen. That period assumes the ability of a healthy foetus to resist brain damage for about 10 minutes despite being deprived of oxygen. This may be described as resilience. We shall have more to say about that below.

The possible causes of cerebral palsy

[171] While cerebral palsy may be caused by intrapartum hypoxia (ie hypoxia during labour) there are other possible causes. That this is the modern understanding appears from Professor MacLennan's article in the BMJ reporting on the work of the International Cerebral

Palsy Task Force (MS 12220 to 12225). The pursuer acknowledges as much. She explains that there are "myriad causes" of the condition and that intrapartum hypoxia is the cause in only about 10 to 15% of cases (MS 12626 and 12627, under reference to the report and evidence of Dr Ferrie at MS 9546 and 1749).

[172] Among the numerous possible causes of cerebral palsy (otherwise risk factors) listed by the pursuer are: genetic or constitutional problems, antenatal or perinatal insults (including chronic partial and acute hypoxic insults), infections, respiratory problems, jaundice and hypoglaecemia (MS 12618). To these the defender would add, as specific to LW's case, pronounced IUGR and a significantly preterm delivery.

[173] Further statistics indicating that intrapartum hypoxia is the cause in only a minority of cases of cerebral palsy and perhaps only a small minority of cases, at least in the case of infants born at term by way of vaginal delivery were provided in evidence by Professor Murphy at MS 8869 et *seq*, and in particular at MS 8872 to 8873. At this point she is dealing with a study of babies delivered at less than 32 weeks gestation in which she had taken the lead (Murphy et al, *Case-control study of antenatal and intrapartum risk factors for cerebral palsy in very preterm singleton babies*, The Lancet (1995) vol 346 p1449 (6/157 of process - MS 9829)). She explained that the causative factors in cerebral palsy are heterogeneous. On the basis of the study 3 to 20% of cases of cerebral palsy in infants born at term are due to intrapartum asphyxia. As Professor Murphy put it when giving evidence:

> "So there's the problem. Between 80 and 97 per cent are not due to intrapartum asphyxia, which means they've happened before you put the CTG on, or they happen after you take the CTG off" (MS 8872-8873).

[174] Of course LW was not born at term and he was not born by way of vaginal delivery (and therefore, strictly speaking there can be no question of him having experienced *intrapartum* hypoxia or asphyxia). Moreover, as he suffered from pronounced IUGR he was

very much smaller (and possibly less developed in other ways) than even a preterm infant of his gestational age. It is not necessary to go further than Professor Murphy's study in the Lancet to discover that this may make a difference. The study found that maternal pre-eclampsia and delivery without labour were associated with a reduced risk of cerebral palsy. There was no increased risk of cerebral palsy with IUGR (MS 8858). However, an underlying risk associated with pre-eclampsia and IUGR is placental abruption and consequent brain damage (MS 8863). This is but one of a number of points in the evidence at which the question arises as to what extent it is appropriate to apply general experience of infants born at term or delivered preterm but at a stage of development commensurate with their gestational age, to LW's case.

Neuro-radiological evidence: suggested signs of injury

Imaging of LW's brain: techniques

[175] Two techniques have been used in order to obtain images of LW's brain. The first to be employed (on 9, 14, 23 October and 8 November 1996) was cranial ultrasound. The second was magnetic resonance imaging ("MRI"). To the layman the science underpinning MRI is not straightforward (MS 1373 to 1379). We have not attempted to master it. As it appears to us it is sufficient to note that MRI is a widely used method of obtaining contrast images of internal organs by means of radio signals generated within a magnetic field. It is regarded as a reliable means of elucidating the aetiology or at least pathogenesis of cerebral palsy (Krägeloh-Mann *The role of magnetic resonance imaging in elucidating the pathogenesis of cerebral palsy: a systematic review*, Developmental Medicine and Child Neurology 2007, 49: 144-151, production 7/22, MS 12246), but not every case can be explained by reference to an imaging diagnosis: there are instances of damage to and developmental disorder of the brain

which do not show up on MRI scanning, thus there may be significant disability but normal imaging; conversely the image may indicate serious damage but the patient may escape lightly (MS 5274, MS 6798, MS 6852). Moreover, as with similar images, such as conventional x-rays, there is room for differences in interpretation of an MRI scan as among suitably qualified radiologists, as was demonstrated in the present case. However, the proposition (derived from the (first) report by Professor Chiswick, the defender's neonatology expert) that any discussion on causation must rely in part on the appearance of brain imaging was put to each of Dr Kirkpatrick, Professor Walker and Professor Murphy and each agreed with it (MS 6469, 7170, 8261 and 8824).

Imaging of LW's brain: history

[176] In 2002 LW was referred to a paediatric neurologist, Dr Sameer Zuberi. The context was that the pursuer and her husband were concerned about the possibility of a genetic cause for LW's disabilities. Dr Zuberi referred LW for MRI scanning of the brain. The scan was carried out on 10 September 2002. It was reported on by Dr Alexander Maclennan, consultant paediatric radiologist. His report is dated 18 September 2002. It is in these terms:

> "Major structures are present and there is no evidence of developmental brain malformation. The scan is abnormal however in that the lentiform nuclei are of reduced volume with bilateral T2 hyper intensities along the lateral margins. The appearance suggests that there has been bilateral infarction of the putamen. The ventricles are a little dilated but there is no clear evidence of PVL. There is dilatation of the anterior aspect of temporal horns and it may be that there is local symmetric loss of [hippocampus] tissue although there is signal abnormality in this region. Overall the appearances are in keeping with the residuum of an acute ischaemic event affecting the basal ganglia and possibly hippocampi".

[177] Dr Maclennan gave evidence (MS 6742 to 6817). He explained that on reviewing the MRI when responding to questions submitted by the pursuer's solicitors he also identified small unreported areas of increased T2 and a fluid-attenuated inversion-recovery ("FLAIR") signal in the periventrical white matter and by the right occipital horn (MS 6765) but that

this was not enough for him to conclude that there was established PVL. The expression that he used to describe what he saw as being suggestive of PVL was "*forme fruste*", in other words an atypical or attenuated manifestation of the phenomenon.

[178] He confirmed that he could see no positive evidence of damage to the hippocampus (MS6807).

[179] Dr Maclennan explained that, at least at the time when he was undergoing training in paediatric neuroradiology, the teaching was that PVL was an injury of the second trimester, the product of chronic hypoxic ischaemia, whereas basal ganglia abnormalities were much more likely to be due to acute hypoxia (MS 6783, 6797). However, he was careful to stress that he saw his role as that of a witness of fact; he disclaimed the role of expert (MS 6810).

[180] Dr Zuberi wrote to LW's parents on 27 October 2002 following the MRI. In his letter he stated that he had found some abnormalities in the appearance of the basal ganglia and possibly some abnormalities in the hippocampus. He further stated that the scan findings were consistent with LW having had a period during which his brain had had a relatively reduced blood and oxygen supply. This event, or more than one event, could, in Dr Zuberi's opinion, have happened at any time over many weeks before LW was born.

Imaging of LW's brain: evidence at proof

[181] The pursuer led expert evidence on the significance of what appeared in the available images. Her experts were Dr Brian Kendall, a retired consultant neuroradiologist (aged 85 when he gave evidence and described by the pursuer's counsel as an "elder statesman" of neuroradiology) and Dr Daniel Connolly, a practising consultant neuroradiologist. The defender did not lead expert neuroradiological evidence, something said by the pursuer to constitute a major flaw in the defender's case.

[182] Dr Kendall's report of February 2013 (6/16 of process, MS 9510) is concise. He notes the ultrasound scans which report as suggesting slight increase in echogenicity (and therefore, as he explained in evidence, damage – MS 1372) in the region of the lentiform nuclei. He describes the cranial MRI as a good quality study consisting of the following sections and sequences: (1) coronal T1, T2 weighted and T2 weighted FLAIR; and (2) axial T2 and dual echo T2 weighted. They show the following abnormalities: (1) a high signal in the putamina of the lentiform nuclei, which are atrophic; and (2) an increased signal from the white matter in the regions of the pre and post central gyri. Dr Kendall's interpretation of these signals is of damage to the indicated regions of the brain. He goes on to explain that these are regions of high metabolism in the maturing foetus and therefore vulnerable to profound circulatory insufficiency. Mammalian experiments (a reference to work reviewed by Ronald Myers in a chapter in *Advances in Neurology* entitled *Four Patterns of Perinatal Brain Damage and Their Conditions of Occurrence in Primates* published in 1976, 6/135 of process, MS 9706 ("Myers")) and clinical experience suggest that damage begins in these regions in previously normal foetuses after about ten minutes of profound circulatory insufficiency (this ten minutes or so can be described as a period of "resilience" during which a previously normal foetus can resist damage). Continuation of the circulatory insufficiency for longer than 25 minutes often results in death or more extensive and more severe brain damage.

[183] In addition Dr Kendall found there to be an increased signal on the FLAIR sequence in the deep cerebral white matter. The lateral ventricles are slightly dilated. While these features are not specific, Dr Kendall explained them as also consistent with a period of profound circulatory insufficiency of relatively long duration, probably close to twenty five minutes. There is no evidence of additional damage in the border zone regions between the

cortical distributions of the main cerebral arteries, which if present may be indicative of more prolonged peripheral perfusion failure. No other abnormality is shown. In particular there is no evidence of additional brain damage. There is no evidence of any malformation of the brain. All the damage described could have been caused in less than 30 minutes leading up to delivery and thereafter until an adequate circulation was restored by resuscitation.

[184] Dr Kendall illustrated his evidence on the location of what he interpreted as damage by reference to productions 6/37 (MS 9584) and 6/133 (not reproduced in the material made available to this Court.)

[185] Thus, on Dr Kendall's assessment of the MRI scans there was damage to the putamina of the lentiform nuclei and damage to the regions of the pre and post central gyri but no damage to the thalami or the hippocampi (MS 1434). There was a degree of enlargement of the ventricles indicative of deep white matter damage but there was no clear-cut PVL (MS 1441 to 1445). Dr Kendall was referred to the report by Dr Maclennan but was not otherwise cross-examined on his interpretation of the relevant MRI scans.

[186] Dr Connolly interpreted the ultrasound scans as demonstrating that as at 9 and 14 October LW's brain was swollen, indicating that it was "acutely poorly" (MS 3076). Dr Connolly's interpretation of the MRI scans corresponded with those of Dr Maclennan and Dr Kendall in identifying damage to the posterior putamen bilaterally. He also identified damage to the right anterolateral thalamus, albeit that it was quite subtle, which Dr Kendall had expressly not done. Agreeing with Dr Kendall, Dr Connolly noted minor high T2 signal within the deep white matter of both cerebral hemispheres, an indicator of white matter damage, but he could not see the indications of damage to the white matter in

the regions of the pre and post central gyri reported by Dr Kendall (MS 3101, 3103). There was brain volume loss.

[187] Dr Connolly referred to "the classic triad" of areas of the brain likely to be affected by hypoxia: the posterior putamen, the anterolateral thalamus and the paracentral white matter (MS 3109). We understood Dr Connolly's reference to paracentral white matter to be to the same area described by Dr Kendall as the pre and post central gyri. Thus, although between them he and Dr Kendall found damage in all these areas, neither of the experts individually identified every component of the triad. Neither Dr Kendall nor Dr Connolly identified damage to the head of the caudate nucleus, the medial temporal lobes, the superior vermis, the sub-thalamic nuclei, hippocampus, the optic radiations or the paracentral white matter (otherwise the paracentral lobule); all of which are areas of potential damage in the event of hypoxia. Dr Connolly accepted that the usual pattern of damage consequent on hypoxic ischaemia in a child of less than 35 weeks gestation would include PVL, which he had not identified in LW's scans (MS 5740). To that extent LW's was not a typical presentation (MS 5742). Moreover, while hypoxic ischaemia is the commonest cause of basal ganglia damage in the perinatal period (Dr Kirkpatrick – MS 6853) the pursuer accepts that basal ganglia damage in a pre-term child is not common (stated by Krägeloh-Mann as 3.5% of cases of pre-term cerebral palsy). That would seem to remove damage to the putamen from Dr Connolly's classic triad where what is under consideration is damage to the brain of a baby delivered at 32 weeks' gestation, if by "classic" is meant commonly found.

[188] The defender did not lead evidence from a neuroradiologist but as part of its preparation for proof it had instructed a report on the MRI scans by Dr Maeve McPhillips, Consultant Paediatric Radiologist at the Royal Hospital for Sick Children, Edinburgh. That

report had been seen by Professor Chiswick, the defender's neonatology expert, and he had incorporated part of Dr McPhillips's report into his report (MS 12144). According to Professor Chiswick, Dr McPhillips had reported the scans as showing high signal in the putamen bilaterally with loss of volume. Similar, but more subtle, high signal was seen in the ventrolateral thalamus bilaterally. The ventricles showed mild dilatation and there was a reduction in white matter volume and thinning of the corpus callosum, particularly posteriorly. There was periventricular high signal on the FLAIR sequence in the parietal and occipital regions extending into the white matter of the pre and postcentral gyri. Some of the affected gyri were abnormal in shape with a narrower base than normal in keeping with deep white matter injury. There was no inherent structural abnormality or malformation of the brain.

[189] Dr McPhillips's reported findings were put to Dr Kendall. He rejected what she had to say about the thalamus and the narrowing of the bases of the gyri (MS 1459, 1461). He maintained his opinion that there had been peripheral perfusion failure but accepted that there was no evidence of it having affected the cortex.

[190] Dr Kirkpatrick saw "some white matter abnormalities" but did not identify PVL on viewing the MRI scans (MS 7015 to 7017).

Evaluation of the evidence on imaging of LW's brain

[191] In her submissions the pursuer pointed to a number of points from the neuroradiological evidence which she suggested were essentially uncontradicted (MS 13820, also MS 12637 to 12642). It is convenient, by way of providing a summary of what we take from the evidence of brain imaging, now to indicate the extent to which we would accept what the pursuer contends for in this aspect of her case.

[192] The MRI scans, carried out when LW was aged 6, show areas of damage which the pursuer divides into grey matter damage and white matter damage. On any view LW has suffered grey matter damage in the form of damage to the putamina, which form part of the lentiform nuclei of the basal ganglia; that much is generally agreed and we take that to be established. According to Dr Connolly, but not according to Dr Kendall, there is also damage to the thalamus. Dr Connolly explained that what he saw was "quite subtle" and he accepted that what he considered that he saw may or may not have been present. While Dr Connolly's identification would appear to be supported by hearsay of Dr McPhillips's assessment, there is a divergence of opinion which we cannot resolve by reference to the material available to us. We therefore see damage to the thalamus as no more than a possibility.

[193] Damage to the lentiform nuclei was shown by the series of ultra-sound scans carried out in October and November 1996. It would therefore appear that whatever caused the damage to the putamina did not postdate 9 October 1996.

[194] Both Dr Kendall and Dr Connolly identified damage to the deep cerebral white matter. There was slight dilation of the lateral ventricles but as Dr Maclennan put it, no clear evidence of PVL. Dr Kendall, but not Dr Connolly, noted evidence of damage to the white matter in the region of the pre and postcentral gyri (that being, as we understood it, in the white matter immediately underlying the cerebral cortex). Contrary to the pursuer's submission, we do not understand Dr Kendall's interpretation to be supported by what Dr Maclennan spoke to in evidence (MS 6765, 6809) and accordingly, while we appreciate that the region of the pre and postcentral gyri is associated with voluntary motor function, again we can only view this as no more than a possibility.

[195] Dr Kirkpatrick did not identify PVL on viewing the MRI scans. Neither did anyone else, at least not with any degree of confidence. The highest the evidence came was Dr Maclennan's *forme fruste*. We therefore take it not to have been demonstrated on MRI.

Neuroradiological evidence: correlation of disabilities with imaging

[196] According to Dr Kendall the damage shown in the putamina was a possible cause of the dystonic element in LW's cerebral palsy, indeed involvement of the putamina was specific to dystonic movement difficulties. If there were damage to the pre and postcentral gyri, this could account for muscle spasticity, ataxia (not being conscious of the position of the limbs), dysarthria (indistinct speech), and oromotor dysfunction (difficulty in chewing and swallowing) (MS 1467 to 1469).

[197] Dr Connolly expressed the opinion that the features he reported as apparent on the MRI scans were in keeping with an acute profound asphyxia. They were, in his opinion, also consistent with dystonic quadriparesis, which Dr Connolly defined as motor dysfunction significantly involving four limbs (production 6/138 of process - MS 9726, MS 3108, 3127). However, when giving evidence-in-chief Dr Connolly accepted that whereas damage to the basal ganglia, including the putamina, can account for movement disorders they cannot account for cognitive impairment. From the radiological perspective cognitive impairment would require diffuse damage to white matter and loss of brain volume (MS 3143 to 3144, 5772, 5863 to 5865). In not having a well-preserved intellect, LW did not present a typical picture (MS 5753). Dr Connolly was cross-examined under reference to two papers of which he was an author: Connolly et al *Involvement of the Anterior Lobe of the Cerebellar Vermis in Perinatal Profound Hypoxia*, production 6/142 – MS 9745; and Crossman et al, *Anatomical Localization of Dyskinesia in Children with 'Profound' Perinatal Hypoxic Ischemic Injury*, production 6/143 – MS 9749). He accepted that in not demonstrating PVL LW did not

present the typical pattern associated with children born at 32 weeks with hypoxic damage (MS 5740 to 5742). As we have already noted when discussing Dr Ferrie's evidence on the significance of the absence of a finding of PVL, neither in that part of his evidence recorded at MS 5740 to 5742, nor anywhere else that we have noted, does Dr Connolly make a distinction as between chronic and acute hypoxia when discussing PVL in the context of a significantly preterm infant. Dr Connolly further accepted that the damage that he had identified (including damage to the putamina) would not be expected to cause dyskinesia (lack of coordination), in the absence of damage to the subthalamic nucleus (not identified in LW's MRI scans) (MS 5743). He argued that damage to the putamina is a marker for an injury which is likely to have caused damage elsewhere (MS 5757). However, in cross-examination in response to a question drawing attention to the complexities of LW's case, he said this:

> "I can accept that some of [LW's] long-term problems are due to genetic and so forth; however, there is clear evidence of bilateral putaminal damage… and …on the balance of probabilities, I would attribute at least a proportion of his current …neurological disability to an event, the precise by the minute timing of which I'm not in a position to… decide… given that we've only got about a three hour period of CTG trace" (MS 5864 to 5865).

Thus, what was spoken to in evidence as revealed by the imaging might explain some but not all of LW's physical disabilities.

[198] LW also suffers from cognitive deficits. Neither the basal ganglia nor the thalamus is principally concerned with cognitive functioning. However, given the consensus on the presence of white matter damage, the evidence was that what was shown in the MRI scans might explain LW's intellectual impairment (Dr Kendall – MS 1546; Dr Ferrie – MS 1875; Dr Connolly – MS 3144, 5832). LW's behavioural problems could not be meaningfully linked to the radiological evidence; there were many children who had dyskinetic cerebral

palsy who had behavioural problems but there were many such children who had no behavioural difficulties.

Suggested mechanism of injury

Hypoxia and hypoxaemia

[199] As already noted, it is the pursuer's case that LW's disabilities are the result of an injurious event in the form of an AHII. This she says was caused by a circulatory collapse secondary to a placental abruption which occurred possibly at or about 1527 on 8 October but in any event within a few hours before delivery (POHS MS 12736 to 12740). We shall have more to say about placental abruption but first we shall look at the pursuer's case supporting the occurrence of an AHII.

[200] The pursuer's case is that there was an AHII straddling the point of delivery by caesarean section, that is beginning prior to birth and continuing until LW was resuscitated at six minutes after delivery. That LW required resuscitation indicates that he did suffer a period of hypoxia and therefore is evidence which the pursuer can point to which is at least consistent with an AHII, leaving the question as to when that AHII commenced. It is on that basis that Professor Murphy accepts that there was indeed an AHII, although, as is pointed out by the pursuer, Professor Murphy appears to have thought that resuscitation took nine minutes (MS 8908 to 8909).

[201] The pursuer explains the mechanism of an AHII such as she avers was suffered by LW in the POHS at MS 12623 and 12677. The brain requires an almost constant supply of oxygen and glucose to fuel the metabolic processes necessary to maintain the vitality of its cellular structure (Dr Kendall – MS 1415). If deprived of oxygen and glucose for a period measured in minutes, neurons will die. As we would understand Dr Kendall's report, he

records there two physical signs of neuron death which can be detected on MRI scanning: atrophy of the putamina, and gliotic response. As we understand it, gliotic response is a reference to changes to the surrounding neuroglia consequent on damage to or destruction of neurons whatever the reason for that damage or destruction may be (Dr Kirkpatrick – MS 7090). It is an immune response. The consequent changes may be likened to the process of scarring.

[202] Areas of high metabolism are particularly demanding of oxygen and glucose and therefore are particularly susceptible to damage in the event of ischaemia or hypoxia. Oxygen is transported to the brain (as oxy-haemoglobin within red blood cells) by the arterial circulation. In the case of the foetus, respiration in the sense of gaseous exchange does not involve the lungs; oxygen is obtained by diffusion from the maternal blood to the placenta and thence to the foetal blood stream (the foetus is connected to the placenta by the umbilical cord, a structure usually consisting of two arteries and a vein). The circulation of the foetal blood stream, pumped by the foetal heart, then delivers that oxygen (bound with foetal haemoglobin) to the foetal brain. Accordingly, the adequate oxygenation of the foetal brain depends, first, on effective diffusion through the placenta to provide the foetal blood with oxygen and, second, on the effective action of the foetal heart to drive that blood to the brain. Here, the damage complained of by the pursuer is said to have been caused by ischaemia; that is an insufficient blood supply to the brain due to ineffective action of the foetal heart, otherwise circulatory insufficiency or circulatory collapse (MS 12677). Given the role of the circulation of the blood in transporting oxygen to cells, including brain cells, circulatory insufficiency will of necessity lead to a state of hypoxia or asphyxia (terms both defined by the pursuer as meaning a low level or lack of oxygen - MS 12625). While ischaemia means lack of blood, where the intention is to specify or to emphasise a low level

of oxygen in the blood the term used is hypoxaemia (MS 12625). Hypoxia or asphyxia may therefore be regarded as the effect whereas circulatory insufficiency is the cause, albeit that Dr Kendall explained that terminology is not always employed strictly, the expressions "profound asphyxia" and "profound circulatory insufficiency" being used interchangeably in the literature (MS 1438).

[203] Dr Kendall distinguished between two situations of circulatory insufficiency: peripheral perfusion failure ("PPF") and profound circulatory insufficiency ("PCI"). As he explained (MS 1412), the blood supply to the brain flows through the carotid and vertebral arteries in the neck to an area at the base of the brain called the Circle of Willis (otherwise the cerebral arterial circle). From there blood is supplied to the nuclei of the brain close (proximal) to the Circle of Willis through relatively short arterial branches. These proximal nuclei include the putamina. The more distant (distal) cerebral cortex on the other hand receives its blood supply through longer branches which pass through the middle of the brain, then go round its outside and then inwards. A sufficient supply of blood to particular tissue (otherwise sufficient perfusion) depends upon the heart exerting sufficient pressure to drive the blood to the particular area. However, should blood pressure fall there will be a difference in the effect on areas which are distal as opposed to the effect on areas which are proximal to the major arteries. Dr Kendall posited a situation where, because of the anatomy of the cerebral blood vessels which he described, there might be a peripheral perfusion failure (PPF) affecting the relatively distal pre and postcentral gyri of the cerebral cortex and yet no such perfusion failure in respect of the relatively proximal putamina. This could be distinguished from the situation arising on a further drop in blood pressure or complete cessation of heartbeat producing profound circulatory insufficiency (PCI) affecting all areas. This was the model that Dr Kendall proposed as applicable to LW's case: a period

of "resilience" during which the foetus resists the damaging effects of hypoxia, followed by a period of damaging PPF followed by a period of more damaging PCI.

[204] While hypoxia (equally hypoxaemia) is a possible effect and circulatory insufficiency is a possible cause, it would appear from the evidence of Dr Kendall that the interrelationship may be more complex. At MS 1417 he describes how the foetus attempts to compensate as a reaction to developing hypoxaemia. With a view to increasing blood flow and favouring the brain over other organs, the heart rate is increased, the cerebral arteries are dilated and blood supply to peripheral parts is restricted. Dr Kendall drew an analogy with the runner whose requirement for oxygen is increased by reason of muscular exertion and whose body compensates for that. However, in the event of continuing hypoxaemia, body tissue becomes acidotic (a shift in the acid/alkaline balance of the blood towards acidity, usually determined by a ph value of less than 7.35, due to there being an insufficient amount of oxygen to maintain aerobic metabolism and a consequent shift to anaerobic metabolism and the production of lactic acid). The runner experiences this build-up of lactic acid (otherwise "blood lactate" or "lactate") through aching muscles. In the case of the foetus, acidosis adversely affects the ability of the heart to pump enough blood to maintain foetal blood pressure which then begins to fall. This causes PPF and then eventually PCI. This is what the pursuer contends happened to LW. In the minutes preceding his birth he suffered a circulatory collapse which endured until he was about six minutes old.

Dr Kendall posited a sequence of events which included a period of PPF followed by a period of PCI, both damaging, albeit differentially as among different areas of the brain. He explained that in the face of PCI the proximal foetal brain will begin to be damaged, the areas more immediately affected being those with the highest rates of metabolism. The experimental basis for this part of his evidence was the work reported on by Myers.

Drawing on a number of studies, Myers considers four different patterns of injury which may affect the brain of foetal or newborn monkeys owing to their being deliberately asphyxiated under four different sets of conditions: (1) total asphyxia leading to a brainstem pattern of injury; (2) partial asphyxia (acidotic hypoxia) leading to brain swelling and cortex injury; (3) partial asphyxia leading to white matter injury; and (4) partial plus total asphyxia leading to basal ganglia injury. Myers notes the location of lesions consequent on hypoxic and/or anoxic episodes and concludes that lesions of the basal ganglia are uniquely associated with an episode of severe partial asphyxia followed by an episode of complete asphyxia. Dr Kendall regarded pattern (4), "partial plus total asphyxia leading to basal ganglia injury", as informative in relation to his assessment of the damage to LW's brain. He suggested that the damage to the putamina seen on the MRI occurred during the period of PCI whereas damage to the pre and postcentral gyri may have begun during the period of PPF (MS 1429 to 1440, 1462).

[205] Dr Kendall accepted that his reliance on Myers was open to the criticism that it depended on the assumption that the results of experiments conducted on infant monkeys at or about term could be applied to a significantly preterm human infant. We also notice, for what it is worth, that in at least some of the studies summarised by Myers as exemplifying the pattern (4) type injury, the monkeys had been partially asphyxiated for 50 minutes, a much longer period than that which Dr Kendall supposed had been the case with LW. The defender further notes (MS 13084) that Myers's pattern (4) injury to the basal ganglia included "major lesions ...which affected the caudate nuclei, the putamen and often the globus pallidus". The MRI scan of LW's brain does not demonstrate lesions affecting the caudate nuclei or the globus pallidus (Dr Connolly – MS 5748). Neither, on Dr Kendall's interpretation, does it demonstrate damage to the thalami or hippocampi, both areas of high

metabolism, like the putamina (MS 1411). As they appear to us, these are quite significant limitations on the application of Myers findings and particularly those in relation to pattern (4) type injury, to LW's case. Dr Kendall was the only witness who placed any reliance on Myers pattern (4) when considering the mechanism of LW's posited injury. He frankly acknowledged that the approach was open to criticism.

[206] Again as we have understood matters, the capacity of a foetus to resist the brain damaging effects of hypoxia which Dr Kendall and Dr Connolly refer to as resilience, is what is to be expected where there has been no previous insult and gestation has otherwise proceeded normally. The foetus has reserves (as we understand it, of glycogen) which it has not required to call upon in order to metabolise anaerobically (see Placental Pathology, Intrauterine Growth Restriction and Subsequent Child Development in Baker and Sibley, *Clinics in Development Medicine, The Placenta and Neurodisability* (2006) - MS 12234). Given the history of placental insufficiency which is discussed below, there is a real question as to whether LW did have the capacity to be resilient in the event of the AHII which the pursuer contends must be taken to have commenced not long before his birth. In cross-examination at MS 6689 to 6698 Professor Chiswick confirmed his view expressed in evidence-in-chief that LW would not have been resilient in the face of an AHII (also report paragraph 9.5). That is at least consistent with the description of LW's condition by Dr Pearse as "teetering" on the brink of collapse (adopted by the pursuer - MS 12666 to 12668). It is another instance of the problem of applying general propositions to LW's case, given its individual features: he was preterm, he was most probably suffering from placental insufficiency and, as a result, he was severely growth restricted. Having regard to Dr Pearse's characterisation of LW "teetering" over what might be quite a long period prior to delivery there may be little likelihood of any period of resilience. It is true that Dr Ferrie and Dr Connolly proposed

some unascertainable amount of resilience (MS 2162, 5791) but we cannot regard that as other than optimistic speculation.

Period of AHII required to produce identified damage

[207] A feature of the pursuer's case is that the AHII which is proposed as the cause of the damage which is sued for must have occurred within a period of no more than 19 minutes, beginning at 1639 when the last foetal heart rate is recorded and ending 6 minutes after delivery when LW's heart rate, 60 bpm when first measured, had risen to 100 bpm. The defender expresses scepticism about a hypothesis which presupposes a very close coincidence in time as between the ending of the CTG trace and the beginning of the AHII but, even on the general understanding based on Myers that a previously healthy foetus can resist the damaging effects of hypoxia for about ten minutes before beginning incrementally to succumb to these effects over a further period of 10 to 15 minutes before death, a period of 19 minutes is sufficient for what the pursuer avers occurred in LW's case (see eg Dr Pearse – MS 3936, 4130 to 4134). Obviously, in the absence of resilience a shorter period will suffice to cause a given amount of damage. That of course means that once there is a question mark over whether and to what extent LW was capable of resilience, estimates as to the time period associated with damage become doubtful. That there is a question mark is clear. We take Dr Pearse, for example, as having accepted that (MS 3926 to 3927). It is true that the witnesses proffered possible periods of resilience (for example Professor Chiswick, whose evidence at MS 6689, 6697 and 6698 is criticised by the pursuer as vacillating between different estimates) but no explanations were provided in support and this seems to us necessarily to be a matter of speculation and nothing more.

[208] There was evidence as to the period of circulatory insufficiency required to cause the damage identified on the MRI. Dr Kendall supposed that a period of half an hour of PPF and then PCI would be enough to account for all the damage (including the white matter damage) seen by him on the MRI (MS 1446, 1454, 1476). Within that he postulated a period of PCI of some 10 minutes but not much longer, to account for the damage to the putamina and the fact that, on his assessment, the thalamus and hippocampi had not been damaged (MS 1459). LW would not have survived an event lasting much more than 25 minutes (MS 1562).

[209] Dr Kendall argued that once it was accepted that the damage he identified required a period of hypoxia within which to occur, then one must suppose a beginning and an end to that period. Spontaneous termination of a period of circulatory insufficiency was unlikely (MS 1478, 12141). In an unborn baby the only indicator of blood pressure and therefore whether there is effective delivery of oxygen to tissues is the pulse or heart rate. LW's bradycardia at birth indicated that he was then hypoxic. His resuscitation at six minutes after delivery when his heart rate was restored to 100 beats per minute therefore marks the end of the period of damaging hypoxia. Given that LW survived but that he suffered the damage identified on MRI and attributed by Dr Kendall to hypoxia, then, assuming maximum resilience, the AHII must have commenced no more than about 20 minutes before LW was delivered.

[210] Dr Connolly also gave evidence about what may have given rise to what he had identified on the MRI scans. Speaking generally, he correlated a pattern of increasing damage and latterly loss in brain volume with a period of complete or near anoxia: ten minutes of resilience; the first five minutes of damage demonstrated by a little bit of signal change; the second five minutes of damage with signal change, a bit of volume loss and

effects in all of the classical areas; beyond 10 minutes of damage, all areas involved, the start of significant volume loss; with death resulting after 15 minutes of damage (MS 3095, 3115). Applying that to LW's case and having regard to the terms of the report by the pursuer's obstetrical expert, Professor Draycott, Dr Connolly opined that LW'S condition had been caused by an episode of asphyxia at or about the time of his delivery and terminating with his being resuscitated. To that extent he agreed with Dr Kendall but, disagreeing with Dr Kendall, he posited one period of what he described as acute profound asphyxia rather than such a period preceded by one of PPF (MS 3134).

[211] Professor Chiswick contended that if there was a brain damaging AHII it occurred after and was triggered by delivery, its damaging effect commencing immediately because LW had no resilience (MS 6697 to 6699). This, as the pursuer points out, is to suppose that the damage was done within the six minutes required to restore LW's heart rate to 100bpm (MS 6393 to 6394) but, as we have already observed an absence of resilience would seem to be consistent with Dr Pearse's characterisation of LW "teetering" during the afternoon of 8 October 1996 and, absent resilience, damage will begin to occur sooner with a shorter period required for a given amount of damage. The possibility that the bradycardia had been brought about by the stress of being born had also been canvassed with Dr Coutts, one of the treating neonatologists (MS 5516 to 5517). He indicated that this can occur, even in cases of caesarean section (which is less demanding on the foetus than vaginal delivery).

[212] The question of the extent to which LW had any available reserves at the relevant time to enable him to resist the damaging effects of hypoxia adds a further uncertainty. The pursuer's witnesses seemed prepared to contemplate damage beginning to be caused after quite a short period of time if one assumes reduced resilience. As Dr Ferrie put it: "if you said to me could he have started to suffer actual damage after a couple of minutes of the

insult, or after nine minutes, I would say I don't know any way to distinguish between the two" (MS 2163 to 2164). Drawing on Myers, Dr Connolly proposed five or ten minutes as the maximum that LW could survive if he had no resilience (MS 5791). In cross-examination Dr Connolly accepted that damage to the putamina could have occurred in three or four minutes of hypoxia (MS 5771 to 5772). He revised that "informed estimation" in re-examination to seven to eight minutes based on his assessment of white matter damage (MS 5877 to 5896). As already touched on, although he referred to the Myers studies Dr Connolly did not seek to rely on the details of pattern (4) type injury when giving his estimates of the period of hypoxia required to cause particular damage. Dr Kendall, with his frank acknowledgement of the limitations of the approach, stood alone in this respect.

Correlation of an AHII and pattern of damage identified on MRI

[213] Whether one prefers Dr Kendall's model of asphyxiation in two stages or one prefers Dr Connolly's one-stage model what is essential, if the pursuer is to succeed in her case as pled, is that she prove that all the relevant damage was caused by an AHII, that is a continuous event of about 30 minutes duration concluding when LW was six minutes old (article VI of condescendence (MS 34). For this purpose the relevant injury must include the damage to the putamina which has been reported by all the neuroradiologists who have viewed the MRI scans. In that the neuroradiologists are agreed that the MRI scans show damage to specific areas of the brain (although they are not entirely agreed as to what these areas are) then the cause of that damage, which the pursuer avers to have been an AHII, may be said to have been discriminating as to the areas which it has affected. We have mentioned that the pursuer accepts that basal ganglia damage is an uncommon finding in preterm children with cerebral palsy. However, if discrimination in favour of the putamina

can be explained by reference to the mechanism of an AHII then that would strengthen the pursuer's case that it was an AHII that caused the relevant damage. As already mentioned, Dr Kendall sought to do just that by his explanation that the putamen is an area of high metabolism (MS 1409 to 1415), a factor also dealt with by Dr Connolly (MS 3095, 3105). As we have already mentioned, from our understanding of the evidence an area of high metabolism will be particularly demanding of oxygen and therefore particularly sensitive to a lack of oxygen, hence the potentially discriminating effect of an AHII. However, Dr Kendall also pointed to the fact that there were areas of high metabolism in which he had not detected damage, which weakens the force of his argument – MS 1411 (also Dr Connolly – MS 5748, 5874 and 9746 to 9747).

[214] A further complication is flagged by Dr Kendall's reference to "the maturing foetus". As with the rest of the body the brain continues to develop through what are normally the 40 weeks or so of gestation. The metabolic demands of particular areas of the brain are not constant throughout that period. The pursuer recognises this point and indeed founds on it with a view to locating the date of the relevant damage to about that of LW's delivery. She relies on the evidence of Dr Kendall who, in chief, explained that (associated with the process of myelination of the white matter) the regions of the pre and postcentral gyri and also the putamina became areas of high metabolism at about 32 weeks gestation (MS 1410 to 1411, 1552). The matter was touched on in cross-examination but Dr Kendall was not directly challenged on his evidence that the putamina become areas of high metabolism at about 32 weeks (MS 1552 to 1553). It is therefore somewhat surprising to see in the defender's note of argument (DNoA) at paragraph B5.8 (MS13970) the assertion that Dr Kirkpatrick had given very clear evidence that basal ganglia damage would not be the

type of damage expected in a preterm infant resulting from an AHII as these would not be the areas of high metabolism at 32 weeks.

[215] We have not been impressed by the passage of Dr Kirkpatrick's evidence to which the defender refers as negating the high metabolism of the putamina at 32 weeks (MS 7257). It arose in re-examination in the context of questioning about the "normally understood pathways of damage to the putamen". Dr Kirkpatrick said: "I have not seen a situation occur of a baby who has ...been preterm and has had a hypoxic ischaemic injury who has sustained basal ganglia damage, and there are good physiological reasons behind that that are well understood in terms of the vulnerability of different parts of the brain, according to different gestations." That is as far as it went. It was not followed up and therefore whatever the "good physiological reasons" may have been they are not available to us and, as observed above under reference to *Dingley*, it is reasoning that carries weight not conclusions. Moreover, if what Dr Kirkpatrick had in mind in the passage quoted above was what is asserted by the defender at MS 13970 then the point would not appear to have occurred to him at the time he drafted his report of October 2011 (7/13 of process – MS 12146). The report notes that LW was born preterm and indeed points to that as putting in doubt the utility of the "pathway documents" (discussed below) which have been framed by reference to babies born at term. Nevertheless Dr Kirkpatrick accepts that "there is evidence of basal ganglia damage that might point to an acute ischaemic event". At paragraph 10.10.2 of his report Dr Kirkpatrick notes the basal ganglia damage and perhaps hippocampal damage and goes on to say "I would agree that those areas are indeed metabolically active and more vulnerable to deprivation of blood and oxygen to the brain." (MS 12166). In evidence Dr Kirkpatrick did say that he had not seen a case of basal ganglia damage in a preterm baby (MS 7257) and it would appear that he was concerned about the

validity of extrapolating data relevant to term babies to preterm babies (MS 7054) but nowhere, and certainly not anywhere where we have been directed, does he make the assertion attributed to him at DNoA paragraph B5.8. Towards the end of his evidence-in-chief Dr Kirkpatrick confirmed that the commonest cause of a basal ganglia lesion in a term baby is a hypoxic ischaemic event. He explained that hypoxia is not the only possible cause of basal ganglia damage and at several previous points in his evidence he had emphasised that he had never seen an instance of basal ganglia damage caused by hypoxia in a preterm baby. However he did not go the distance of excluding it in LW's case (MS 7138 to 7139) as he might have done had it been his view that damage to the putamina would be unlikely at 32 weeks' gestation. In cross-examination it was put to Dr Kirkpatrick that Dr Kendall had said that the putamen "only becomes vulnerable to damage at about 30 weeks gestation" to which his response went no further than "I was not aware of that".

[216] Now, LW was growth restricted. That might raise the question, at least to a layman, as to whether the state of development of his brain at any particular gestational age was at the same stage as would be reached by an infant who did not suffer from IUGR. This might impact on the proposition that by 30 weeks the putamina had reached the stage where they would be particularly susceptible to hypoxic injury. We take this question to have been touched on by the defender in submissions to the Lord Ordinary (MS 13080). We do not however see it to have been pursued in the evidence, at least not in any passages to which reference has specifically been made.

Alternative mechanisms of hypoxic injury

[217] One alternative cause of the damage identified on MRI is that it was caused by a chronic hypoxic antenatal event, or series of events. Another is that it was caused by a

postnatal acute hypoxic event or series of events. As a matter of pleading these possible alternatives are put forward in answer 6 (MS 36) where the defender avers:

> "There was a retroplacental blood clot of 25 per cent of the placental area. Placental blood flow would have been diminished for some time …In respect of the subsequent MRI scan appearances, the changes in the basal ganglia cannot be attributed to an episode of profound acute hypoxia occurring just prior to delivery (as the pursuer postulates on record). At least some of the appearances are compatible with events over a period of weeks in a severely growth retarded infant. … [LW] would have had severe neurological impairment if admission had been on 5 October 1996. …Any acute hypoxic injury suffered by [LW] was minor in nature and has not affected his subsequent symptoms. …any acute hypoxic injury which [LW] suffered is likely to have occurred in the period immediately after his delivery."

[218] In submission, the pursuer, consistent with her case, contended that LW did not suffer brain damage as a result of chronic partial brain damage *in utero*. In support of her submission she referred to the reports from Dr Ferrie and Dr Pearse and the evidence of Dr Kendall, Dr Connolly and Dr Maclennan (POHS - MS 12643-12645, PNA - MS 13820-13822). Looking at that evidence it occurs to us that there might be value in clarifying the terminology to be used (the defender comments on terminology at MS 13078). At MS 9663 Dr Pearse distinguishes between "two types of perinatal asphyxia". These are what he refers to as "chronic partial asphyxia" and "acute near total asphyxia". Both types involve a restriction in the oxygen supply to the brain with consequent damage. The first involves an incomplete restriction over a longer period (at least an hour). The second involves a complete or almost complete restriction over a shorter period (about 25 minutes, but no longer because in the case of a resilient foetus such a period of oxygen deprivation will result in death). At paragraphs 3.56 and 3.57 of his report (MS 9558) Dr Ferrie draws the same distinction although he refers to the first type as a "chronic partial insult" and the second type as "sudden severe". His time periods are the same as those referred to by Dr Pearse. The distinction looks to be the same as or at least similar to that made by

Dr Kendall between PPF and PCI (MS 1412-1413), although in LW's case Dr Kendall was supposing that the total period of asphyxia did not exceed 26 minutes with damage having been caused by an incomplete restriction of oxygen to the brain by 15 minutes into that 26 minute period. This is to differentiate between "chronic" and "acute" in a very much more precise way than in the line taken by the defender in answer 6. What the defender says there is that any damage (whether in the sense of an adverse condition caused by injury or an adverse condition caused by failure of normal development) was done before, and perhaps well before, 5 October 1996 but if that is not so and there was an acute hypoxic injury then it post-dated LW's birth.

[219] What the defender has to say about chronic partial hypoxic injury in the long term pre 5 October 1996 sense is best included in the discussion of IUGR and placental insufficiency which follows below. We therefore turn to the second alternative mechanism which is postnatal acute hypoxic injury associated with the period required for LW's resuscitation, in other words during the first six minutes of life. This mechanism was supported by Professor Chiswick whose report explained LW's cerebral palsy in terms of hypoxaemia, accepting that damage to the basal ganglia and thalami (he assumes damage to the thalami because he is relying on Dr McPhillips's report on the MRI scan) "probably occurred immediately after birth during resuscitation when his circulation was poor" (report paragraphs 9.6, 12.1 b - MS 12140, 12143). In evidence he confirmed that "based on the MRI scan it is …likely that [LW] did suffer an acute profound hypoxic insult, the issue is the timing of this insult" (MS 6533). At MS 6533 Professor Chiswick goes on to mention his experience of preterm infants who have not gone through labour and who have suffered basal ganglia damage "as a result of an insult in the neonatal period". While these other cases may have occurred later than immediately after birth we see Professor Chiswick

contemplating the possibility (described by him as a probability) of LW's basal ganglia damage having resulted from hypoxia during the first six minutes of his life. Dr Ferrie indicated much the same when he volunteered (MS 9186):

> "[LW] actually was in, I think everybody accepts, a precarious situation… he… was growth restricted etc and the, the sort of 10-minute rule, if you like, which applies in most cases, is to healthy foetuses, which he certainly was not … it is quite possible that 6 minutes was enough to, to do the damage and you could argue that it could have been less as well. And, of course, his degree of… physical impairment as a consequence of the… basal ganglia damage, and I'm talking about his motor impairment, is not at the very severe end of the scale."

This of course is consistent with Dr Connolly's evidence that damage to the putamina could have occurred in three or four minutes of hypoxia (MS 5771 to 5772).

[220] In summary, as far as the grey matter is concerned, we accept that the putamina suffered damage. It is possible that there is also damage to the thalami and to the white matter of the pre and postcentral gyri. There is an indication of damage to the deep white matter but PVL is not demonstrated. The putamina, thalami and pre and postcentral gyri were identified as susceptible to damage as a result of hypoxia, not simply in the sense of being capable of being so damaged but also in the sense of being particularly vulnerable to such damage by reason of their high energy requirements. If it were accepted that the damage shown on the MRI scans was caused by an acute hypoxic event, given the ultrasound scan on 9 October showed signs of what we would understand to be the same damage, that hypoxic event must have been proximate in date to LW's delivery. Since, as Dr Kendall explained, an acute hypoxic event is unlikely to be self-limiting (in other words, it is unlikely to come to an end spontaneously other than perhaps in the event of cord compression being relieved by movement of the foetus as mentioned by Professor Walker at MS 7452) and that a foetus is unlikely to survive 25 minutes without oxygen, any such acute event must also have been proximate in time to LW's delivery. However, the evidence does

not exclude the possibility that the damage to the basal ganglia, could have been done during a six minute period of hypoxia immediately following delivery.

[221] The pursuer submits that it is highly significant that Professor Chiswick accepted that the damage to the basal ganglia must have been caused by an acute episode of profound hypoxia. That indeed was his evidence but he distinguished between the grey matter damage which points to an acute event and the white matter damage which does not necessarily do so. On his evidence, the grey matter damage to the basal ganglia, was the result of an acute hypoxic event. His proposed candidate for that was the six minutes period post-delivery. Professor Chiswick accepted that the entirety of the damage seen on the MRI scans could not be explained by the six minute event (MS 13314). The white matter damage must be explained otherwise. That Professor Chiswick associated that with a more chronic process (MS 6624 to 6626, 6644, 12141, 12143). We therefore turn to the evidence which included references to the possibility of a more chronic hypoxic process.

IUGR and placental insufficiency

[222] A further point of difference between the monkeys which had been experimented on in the studies reviewed by Myers and LW, is that LW suffered from IUGR. His birth weight was 920 gms (0.4th centile), head circumference 26.5cm (0.4th to 2nd centile), and length 39cm (9th centile). These measurements are what a foetus might be expected to attain by about 26 weeks.

[223] We have already touched on the role of the placenta in allowing the diffusion of oxygen, glucose and other necessary nutrients from the maternal blood stream into the foetal bloodstream. Although the respective circulations are separate, the placenta brings maternal and foetal blood vessels into close proximity thus permitting diffusion of gases and

nutrients across cell membranes (placental function and dysfunction are described in Dr Howatson's written answers at MS 9786 to 9791; they were also addressed by Professor Draycott at MS 2566 to 2573). Part of the mechanism connecting the mother with the developing foetus is constituted by the chorionic villi which are projections containing foetal blood vessels. Oxygen and nutrients in the maternal blood diffuse across the cell membranes into the capillaries of the villi while waste products diffuse in the opposite direction. Effective gas exchange and a sufficient supply of nutrients are necessary for the development of the foetus. It follows that should, for whatever reason, the placenta fail to carry out these functions, a situation which may be described as placental insufficiency may arise with the result that the development of the foetus is adversely affected.

[224] The pursuer accepts that LW was adversely affected by placental insufficiency in the antenatal period with consequent malnutrition and hence IUGR (MS 12649). She specifies the immediate cause of placental insufficiency in LW's case as a narrowing of the umbilical artery secondary to the pursuer's alleged pre-eclamptic toxaemia ("PET"). The pursuer says that this is the mechanism demonstrated by the ultrasound examination (the "Doppler reading") carried out by Dr Crichton at about 1311 hours on 8 October 1996, and interpreted by Professor Draycott as intermittent absent end diastolic flow (MS 1686, 2566 to 2573; see also Dr Crichton at MS 1647). While the umbilical arteries take deoxygenated blood from the foetus to the placenta and the umbilical vein takes oxygenated blood and nutrients from the placenta to the foetus, resistance to flow in the arteries is associated with "poor blood flow to the baby [and] reduces the nutrients and oxygen to the baby", as Professor Draycott put it (MS 2571). Thus, on the pursuer's interpretation of the evidence, on 8 October 1996, but necessarily for a period of weeks beforehand if the narrowing of the umbilical artery is the only reason for LW's IUGR (MS 1693, 8629), LW was receiving a sub-optimal supply of

oxygen and necessary nutrients (and see Dr Pearse - MS 3895). The pursuer describes this as a period of asphyxia during which LW's brain was inadequately perfused by oxygen and therefore increasingly vulnerable to an acute hypoxic insult, albeit not yet damaged; she adopts Dr Pearse's expression "teetering" (MS 12666 to 12668). That may be one possibility, although in the absence of supporting evidence it is no more than an optimistic assertion. It is not the only possibility. As was explained by Professor Chiswick, the extent of LW's IUGR points to placental dysfunction over a period of some weeks (MS 6632). Professor Walker was prepared to contemplate months (MS 12196). Professor Chiswick distinguished between failure in the supply of nutrients and failure in the supply of oxygen but

> "it is often the case that when the nutrient supply fails the oxygen fails. So …there is every reason to… suppose that in the antepartum period there was… hypoxia ischaemia,… operating over a period of time" (Professor Chiswick - MS 6632 to 6333; see also Professor Draycott – MS 2571).

Professor Chiswick then went on to postulate a scenario where growth failure started two or three weeks before 32 weeks gestation, where the foetus was growing poorly but was not at the stage of being chronically hypoxic. However, by the time of the biophysical profile on 8 October there was evidence of a sufficient degree of placental dysfunction to cause hypoxia, a situation which continued up to the time of delivery, as evidenced by the CTG trace. Professor Chiswick suggested that this may have been going on for just over a week prior to delivery (MS 6634). It was possible that the white matter damage which had been observed had been caused through this mechanism (MS 6631). Thus, there would appear to be agreement as between the parties' respective experts that LW was subject to chronic antenatal hypoxia (in the sense of suboptimal oxygenation of the foetal bloodstream). The difference between them was that the pursuer's expert asserted (albeit without any explanation) that there was no material brain damage prior to a posited circulatory collapse

just prior to delivery, while the defender's expert thought that antenatal damage was likely and that the biophysical profile supported that.

[225] Evidence of the mechanism which led to LW's IUGR was provided by the answers given by Dr Allan Howatson to a series of written questions submitted to him by those acting for the defender. Dr Howatson was the consultant paediatric and perinatal pathologist who had carried out macroscopic examination of the pursuer's placenta on 9 or 10 October and a microscopic examination on 18 October 1996. His report on these examinations and that of the umbilical cord is contained within the hospital records at MS 10234. His written answers and the relevant questions are at MS 9778 and 9785. Dr Howatson did not testify but it was agreed by parties that the questions and answers should be taken as evidence in the case.

[226] Dr Howatson identified a small area of infarcted villous tissue in the placenta which he attributed to deficient intervillous circulation as a result of pathological changes in the decidual arterioles. Many of the villi showed stromal fibrosis. Dr Howatson explained that this is the result of the cessation of foetal blood flow to the affected villi; they are avascular and accordingly non-functional. He also identified a widespread excess of syncytial knots which, he explained, is an indication of placental ischaemia. That the placenta was "not normal" was spoken to in evidence by Dr McLean who had carried out the caesarean section (MS 5411).

[227] The pursuer's position in relation to this aspect of the case is to accept that inadequate nutrition and oxygenation with consequent IUGR are part of the background to the events of 8 October, but to maintain that it was only background to a circulatory collapse initiating an AHII minutes before LW's delivery attributable to a steadily worsening

condition of foetal compromise which was brought to a crisis by a placental abruption (MS 12668, 13821 to 13824).

Placental abruption

[228] Placental abruption might be seen as an extreme example of placental insufficiency. The part of the placenta which is attached to the wall of the uterus is referred to as the decidua. After the birth of a baby the placenta will spontaneously detach from the wall of the uterus, allowing it to be delivered as the afterbirth. However, the placenta may detach prematurely, in whole or in part, before gestation is complete and the foetus is still within the uterus. This event is referred to as an abruption (MS 11915). Its immediate cause is haemorrhage at the decidual/placental interface and, because it disrupts the connection between the maternal and foetal blood vessels to a greater or lesser extent, it will adversely impact on the wellbeing of the foetus. According to a paper produced together with Professor Murphy's report (Oyelese and Ananth, *Placental Abruption*, Obstetrics and Gynaecology vol 108, no 4, October 2006, p1005 – MS 11914), placental abruption is associated with IUGR, in the vast majority of cases it being the end result of a chronic process dating to the first trimester, and both it and IUGR share a common cause. Again according to Oyelese and Ananth, abruption occurs frequently in the setting of pre-eclampsia. An association among pre-eclampsia, small for gestational age and abruption is noted in another of the papers produced with Professor Murphy's report (Ananth and Vintzileos, *Ischaemic placental disease: epidemiology and risk factors*, European Journal of Obstetrics & Gynaecology and Reproductive Biology 159 (2011) 77-82 – MS 11885).

[229] It is the pursuer's position that the evidence establishes that the pursuer suffered two placental abruptions, the significant one of which occurred in a period of a few hours

leading up to LW's birth (MS 12732 to 12740, 13822 to 13827). The point is made succinctly in the PNoA at MS 13824: "Abruption occurring shortly before birth caused the circulatory collapse and the AHII event." The defender points to the evidence which might support a significant abruption at about midnight on 7 October, including the pursuer's account of not feeling any foetal movement during the morning of 8 October (MS 13302 to 13304, with reference to MS 767). This, suggests the defender, might have "caused an acute but self-limiting insult to the foetus's brain" (MS 13303). We do not pretend to understand how the defender justifies "self-limiting" but given the appearance of the placenta after delivery, the issue between the parties is not whether an abruption or abruptions occurred but, rather, when they occurred and what is their significance.

[230] One significance for the pursuer's case of a placental abruption occurring not long before delivery is that it is available to take on the role of a "sentinel event" or the immediate precursor of such an event. In the present context a sentinel event is an identified unexpected pathological event occurring in the course of the pursuer's antenatal care which gave rise to the adverse outcome under consideration or, more simply, provides an explanation for what followed (identification of a sentinel event is one of the criteria to be found in the "pathway documents" discussed below). Here the adverse outcome was hypoxic brain damage due to circulatory collapse and consequent bradycardia. The pursuer's case is that the placental abruption precipitated circulatory collapse and a consequent AHII (albeit not necessarily immediately); the placental abruption therefore explains the adverse outcome and allows the pursuer to argue that had it been avoided, as it would have been by the delivery of LW on 7 October or earlier, he would not have suffered the injury and consequent damage in respect of which the pursuer makes this claim. She submits that that case is endorsed by the evidence of her experts: Dr Kendall at MS 1558 to

1560, 1580; Dr Ferrie at MS 1840, 1871 to 1872, 1881 to 1885, 1947, 2185 to 2188, 2283; Professor Draycott at MS 2636 to 2640, 2832 to 2834, 2841 2844, 2892; and Dr Pearse at MS 3750.

[231] The defender submits that the pursuer has failed to prove a sentinel event in the "dark period" or, as the precursor of such an event, the occurrence of an abruption close in time to the "dark period". It points to the history of the pursuer suffering abdominal pain at midnight on 7 October, what Dr Crichton saw on ultrasound examination and the BPS that she recorded as consistent with the pursuer having suffered an abruption prior to her admission to hospital on 8 October.

[232] In support of the contention that she suffered an abruption not long before LW's birth the pursuer points first to the operation note made by Dr McLean: "IUGR probable abruption …placenta + retroplacental clot delivered – about ¼ of area. Placenta very thin and friable…. [Estimated blood loss] less than average". Dr McLean spoke in evidence to having delivered the placenta together with a retroplacental clot taking up about a quarter of the placenta (MS 5407 to 5411, 101265, 12732). The pursuer then turns to the evidence of Dr Howatson: his findings on his examination of the placenta support the occurrence of at least two placental abruptions, one more or less coterminous with delivery, superimposed on the history of chronic placental insufficiency to which we have already referred.

[233] Dr Howatson identified what he described as an "old blood clot" and "focal fresh haemorrhage". These findings indicated a minimum of two episodes of placental related bleeding. The first episode was a marginal placental detachment producing the old blood clot. That episode may have been one incident or a series of consecutive smaller incidents over a short period of time. It occurred several days earlier than delivery (Howatson answer 32) (MS 9797). The absence of haemosiderin-laden macrophages observed in the

sections subject to histological examination might suggest that the old clot was less than 4-5 days old. The focal fresh haemorrhage indicated a second episode which was retroplacental and which occurred within a few hours of delivery. It had not caused such a compression or indentation of the placenta as would be seen by a large retained placental haemorrhage present for many hours or days. Whereas in the case of a large acute abruption the clinical presentation may be associated with abdominal or back pain, sometimes accompanied by vaginal bleeding, smaller abruptions may be painless and may not present with vaginal bleeding (Howatson answer 44) (MS 9787).

[234] On the pursuer's approach, the retroplacental clot and the fresh focal haemorrhage reported by Dr Howatson are indicative of an abruption or series of abruptions which occurred in the period of "a few hours" prior to delivery or more or less at the point of delivery. That abruption close to the point of delivery is the sentinel event and the retroplacental clot is the evidence of the sentinel event. The old clot may have been no more than a few days old but it was not associated with a brain damaging episode. Neither was the episode of pain reported by the pursuer as having occurred at midnight on 7 October 1996. Now, for such a placental abruption as is identified by the pursuer to be the sentinel event, timing is important in that the fewer the hours between the abruption and the AHII posited by the pursuer as ongoing at the point of delivery, the easier it may be to infer a causal connection. For that reason, the pursuer invited a critical examination of the evidence of Dr Crichton on the matter. It will be recalled that, on the basis of what she saw during the ultrasound scan she thought that there might be a retroplacental clot and that therefore the pursuer might have suffered or to be suffering a placental abruption (MS 1671). The ultrasound examination was carried out at about 1300 hours on 8 October (perhaps beginning at 1304 – MS 1668). If Dr Crichton had indeed identified a retroplacental clot that

would be to put the occurrence of the abruption at least 4 hours before delivery at 1652 hours.

[235] Dr Crichton was led by the pursuer. She was not asked about abruption during her evidence-in-chief beyond confirming that she had recorded the possibility of a retroplacental clot (MS 1628) although in the course of a long answer to a question on another point she mentioned that she was concerned that there had been an abruption (MS 1639). Her more specific evidence on the topic arose out of cross (which was not challenged in re-examination). She confirmed that on carrying out the ultrasound scan something about the image suggested that there was "a clot there", hence her note (MS 1671). She went on to say that what she saw was consistent with Dr McLean's operation note describing the condition of the placenta at the time of delivery (MS 1674). As we have indicated, she was not challenged about any of that.

[236] Dr McLean spoke to not having found the uterus tense, and not having noted fresh bleeding or blood staining of the liquor at delivery, whereas the estimate of blood loss ("EBL") from the operation (at 500 mls) was less than average for a caesarean section (MS 5412 and 5418). According to Dr McLean, these are factors which would point away from the conclusion that the pursuer "was experiencing an abruption" at the time of delivery (MS 5414). In that she was supported by Dr Hanretty who assisted in carrying out the section (MS 2468). In this context he referred to the below average EBL (MS 2467). As these witnesses were aware that there was clear evidence that the pursuer had experienced an abruption, we would interpret their testimony, like that of Dr Crichton, as pointing to its occurrence being earlier rather than later, and certainly not contemporary with the delivery.

[237] We accept, as the pursuer submitted, that it was supposition on the part of Dr Crichton to say that what she saw during the ultrasound scan correlated with what

Dr McLean found at caesarean section and that, similarly, it was supposition on the part of Dr McLean to say that what was found at section was what had been seen earlier by Dr Crichton (MS 5412). The fact remains that at about 1300 Dr Crichton thought that she had identified the presence of a retroplacental clot indicative of the occurrence of placental abruption, and that later it was found that an abruption had indeed occurred and that there was indeed a retroplacental clot. That does not exclude the possibility of Dr Crichton having been in error as to what was apparent on the ultrasound, and Dr Crichton did explain that "abruption is not usually a scan diagnosis, and that's what we would teach our junior staff" (MS 1671) but it is going too far to say, as the pursuer says in submission, that it cannot be taken from Dr Crichton's evidence that a retroplacental clot of any size and associated with any particular abruption had occurred at any particular time relative to the ultrasound scan. In considering the weight to be given to Dr Crichton's evidence as to what she considered was demonstrated by the scan on 8 October 1996, it is relevant to note her qualifications and experience, as elicited at the beginning of her cross-examination (MS 1643 to 1646). In October 1996 Dr Crichton was a senior registrar. She had graduated Doctor of Medicine (MD) in 1995. She obtained her first consultant post in December 1996. This was at the Queen Mother's Hospital. Dr Crichton had a particular interest in obstetric ultrasound. She had obtained a diploma in the subject in 1994. She has subsequently published on the topic. On her appointment as a consultant Dr Crichton took over responsibility for the foetal ultrasound department at the Queen Mother's. According to Professor Walker, at the relevant time the Queen Mother's Hospital was a world leader in ultrasound scanning.

[238] The pursuer relies on the evidence of Professor Draycott to counter what might otherwise be taken from the evidence of the treating clinicians. He doubted that the pursuer's report of a two minute episode of pain at midnight on 7 October accounted for the

fresh haemorrhage affecting 25 per cent of the surface of the placenta, although it might account for an abruption which gave rise to the old clot (MS 2865). On the significance of the EBL, he explained that if there had been a recent abruption (in the sense of some minutes or up to an hour before delivery) one would not anticipate "more blood than normal" (MS 2624); a small abruption occurring shortly before the time of birth would result in a smaller accumulation of blood, whereas an earlier abruption would cause a more extensive accumulation (MS 2840). At MS 2581 to 2583 and 9477 Professor Draycott gave evidence to the effect that the occurrence of an abruption at about 1527 was "biologically plausible" although he declined to give any estimate of probability. We consider that the defender was justified in describing this evidence as tentative. It also seems to suppose that the pursuer experienced an episode of pain at this point, something not established in evidence.

[239] In her report Professor Murphy stated that acute placental abruption is usually associated with a fall in haemoglobin and platelet count and coagulation dysfunction with abnormally low fibrinogen. In the pursuer's case, however, all of the blood tests were entirely normal and not in keeping with either severe early onset pre-eclampsia or acute onset placental abruption (MS 8481 to 8482). Thus the blood tests were not consistent with an abruption evolving at the times when these tests were carried out. Professor Murphy was asked to comment on the assumption in Dr Kendall's report that an abruption had occurred at 1527 hours on 8 October in her evidence at MS 8742 to 8743. She rejected the proposition that there had been an abruption at that time. In her view there was clear and compelling evidence that the placental abruption occurred prior to the pursuer's admission to hospital. There was no evidence of a placental abruption at 1527 on 8 October. There was no vaginal bleeding, no abdominal pain, and no uterine activity on the CTG. While there were a number of decelerations and a resetting of the foetal baseline heart rate, there was a

clear physiological explanation for that, which was the lowering of the mother's blood pressure. If there had been a placental abruption at 1527 causing immediate hypoxia, there would have been some 85 minutes of hypoxia before delivery, which LW would not have survived.

[240] Professor Walker's evidence was that the old clot was anything from 12 hours to 3 to 4 days old but there was no evidence from which the occurrence of the abruption could be timed (MS 7702 to 7711). There were no symptoms of abruption (apart from the sharp pain at midnight on 7 October), there was no massive blood loss at delivery and there was no need for pre- or post-operative management. The abruption could not have been very big and was probably about 100mls (which was the difference between the placenta at delivery and trimmed weight at pathology). The coagulation parameters were normal before and after delivery also suggesting that this was not a significant abruption (MS 7558 to 7561).

[241] A complete placental abruption will sever contact between the maternal and foetal bloodstreams and lead to the death of the foetus through the processes and within the timescales discussed by Dr Kendall. However, the retroplacental abruption described by Dr Howatson in the present case was not complete and we would understand all the relevant experts as prepared to contemplate LW surviving for at least a matter of hours and perhaps considerably longer after such an event had occurred (because the greater part of the placenta remained attached to the lining of the wall of the uterus allowing the continued, albeit perhaps reduced, diffusion of dissolved oxygen and nutrients from the maternal blood). That would not of course be so if the abruption triggered an immediate circulatory collapse. The pursuer's case is that LW did suffer a circulatory collapse and that "the abruption is important because it was this which caused [LW's] circulatory collapse in the minutes leading up to his birth" (PNoA MS 13825). Evidence of an abruption "in the

minutes leading up to [LW's] birth" would provide support for the pursuer's case in that it would more readily allow the inference that LW's postnatal bradycardia was a continuation of an ante-natal collapse in his circulation. However, we have not found support in the evidence for an abruption occurring at that time. Rather, that evidence seems to us to point to the retroplacental abruption having occurred before, and possibly some time before, Dr Crichton carried out the ultrasound examination at 1300 on 8 October. That is not, however, necessarily fatal to the pursuer's case, at least as it is pled, since she argues that a causative abruption, "may well have occurred some time before the bradycardia and circulatory collapse" (MS13825).

Obstetric evidence: foetal circulation and CTG

[242] The pursuer's case is that the immediate cause of LW's damage was what was referred to in the evidence as circulatory collapse, a state marked by bradycardia where the foetal heart is no longer delivering a sufficient quantity of oxygenated blood to the foetal brain. An alternative description of the condition is profound cardiorespiratory depression. The pursuer has this event as beginning just before and continuing for six minutes after delivery. She points to LW'S condition at birth: cyanosed, having a low heart rate and no respiration, and requiring the administration of adrenalin and cardiac massage to achieve this. This, as the defender submits, is a circumstantial case; it depends upon the inference being drawn from LW's condition at birth that he was in a state of bradycardia for at least ten minutes before birth. Professor Draycott accepted that no direct clinical sign of foetal bradycardia or circulatory collapse is recorded (MS 2758).

[243] The defender points to the biophysical profile carried out by Dr Crichton as indicating that LW was already brain damaged by the time of his delivery but, through the

evidence of Dr Coutts at MS 5497, 5504 to 5521, it also challenges the extent to which LW can be said to have been in a state of circulatory collapse. As we have previously noted, Dr Coutts pointed to LW's cyanosed condition as an indication that he still had blood flowing to his extremities.

[244] Evidence as to LW's cardiac activity *in utero* is provided by the CTG trace. The record is however incomplete to the extent that CTG monitoring was discontinued during the pursuer's transfer to the labour suite and for perhaps 13 minutes prior to delivery (the "dark period"). It appears to be uncontroversial that the appearance of the CTG trace was a matter of concern to the responsible clinicians at the time and that when it was reviewed for the purposes of the action it could properly be described as abnormal or pathological, showing reduced variability throughout, albeit that it did not demonstrate the bradycardia which was evident when LW was delivered. Sandra Tranter comments on the two sections of CTG trace with which she was provided. The first lasted approximately 9 minutes. It showed an unreactive foetal heart baseline of just over and then just under 150 bpm. The second section commences at 1446 immediately after the pursuer's transfer to the labour suite. The foetal heart rate was 150 bpm, which is within the normal range of 120-160 bpm, with reduced baseline variability. There was an unprovoked deceleration at 1510 with some loss of contact. The CTG continues to show a lack of baseline variability until just after 1530 when the baseline drops to 125 bpm, decelerations occur and there are periods of loss of contact. On Ms Tranter's reading of the CTG it ends at approximately 1644. The midwifery notes (MS 10263) record the foetal heart rate as being 122 bpm at 1639; while the Apgar score of 0 for heart rate at birth suggested that a heartbeat was absent. The report of the pursuer's other midwifery expert, Dr Jean McConville, is to similar effect (MS 9427); from approximately 1429 there is an increase in uterine tone. In her opinion this was a suspicious

pattern, which by 1540 had developed into a pathological pattern (Professor Chiswick, in his report at paragraph 9.3, although deferring to obstetrical evidence, describes the CTG as pathological from 1445 – MS 12139). Dr McConville explained the unreactive trace by analogy with a baby being "tired". She identified deterioration after 1540 (MS 4973). Professor Draycott's assessment was of the foetus becoming more and more unwell (MS 2549). He agreed with the proposition that the foetus was not coping well and offered the opinion that "something has changed acutely to make that happen" (MS 2969). In her report Professor Murphy notes that CTG classification and interpretation is based on experience with term foetuses but describes the trace for the period 1405 to 1425 as pathological. She uses the same description for the trace for the period 1445 to 1620, recommenced at 1630 and concludes: "Each of the two CTGs are (sic) pathological from the outset suggesting foetal compromise that predates the start of the first CTG" (MS11390).

[245] Whereas he described the trace as "pre-terminal" (MS 2495) Dr Hanretty was not prepared to accept that the CTG trace demonstrated a deterioration after 1527; that was to over-interpret what was not a sufficiently sensitive diagnostic technique (MS 2505).

[246] The last record of a heartbeat was made at 1639 (or 1637 according to Dr Thorburn's watch). With delivery timed at 1652 there was therefore a total of about 19 or 20 minutes between the last antenatal foetal heart rate reading and the return to 100 bpm at 6 minutes of life.

[247] The pursuer's case requires LW to have been in a state of bradycardia prior to section. Dr McLean, who carried out the operation, was not aware of anything happening from the time of discontinuation of the CTG trace to the moment of delivery (MS 5442). Dr Thorburn, the attending anaesthetist, did not note a drop in maternal blood pressure (MS 5379).

[248] The defender points to an absence of any clinical finding indicative of foetal bradycardia and goes a step further. On the basis of the evidence of Drs Hanretty, Pearse and Walker, in 1996 it was the practice to check the foetal heart rate just before section (MS 13970). That, argues the defender, despite Dr McLean's evidence on the point not being clear, permits the inference that the heart rate was checked (presumably using a Pinard horn or other form of stethoscope). The absence of a clinical note as to the result of that then permits the inference that there was a heartbeat on the view that if no heartbeat had been detected then that would have been noted, so reducing the "dark period" (MS 13117). That is the argument. We have not been persuaded. The defender is entitled to say that there is no evidence of bradycardia prior to section but not that bradycardia can be excluded until any later point of time than the end of the CTG trace.

Neonatal evidence

The pathway documents

[249] In this context a "pathway" is a particular course of events ("a chain of linked events" is the way Dr Kirkpatrick put it in his report – MS 12164) leading to a particular clinical outcome, the importance of such being that where a recognised pathway can be identified by reference to certain criteria then it is possible to conclude that there is a relationship of cause and effect between a particular event (one step on the pathway) and a particular outcome (a later step on the pathway), but not otherwise. Here what is proposed as the cause is an AHII and what is proposed as the effect is LW's cerebral palsy.

[250] Two "pathway documents" were lodged in process by the defender with a view to establishing what were the relevant criteria and then submitting that some or other of them were not present in LW's case. The documents were: Alastair MacLennan for the

International Cerebral Palsy Task Force, *A template for defining a causal relation between acute intrapartum events and cerebral palsy: international consensus statement*, British Medical Journal 319, 16 October 1999, pp 1054-9 (production 7/20, MS 12220) ("the ICP template"); and the American College of Obstetricians and Gynaecologists, *Executive Summary of the report of ACOG's Task Force* on *Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogenesis and Pathophysiology* (production 7/19, MS 12218) ("the ACOG template"). In the BMJ article Professor MacLennan describes work initiated by the Perinatal Society of Australia and New Zealand in order to bring together the modern literature on the causation of cerebral palsy and to try to define an objective template of evidence the better to identify cases of cerebral palsy where the neuropathology began or became established around labour and birth with a view to achieving an international consensus statement to help, among others, the courts more easily to understand the pathology causing cerebral palsy. The Executive Summary explains that ACOG had convened a Task Force on Neonatal Encephalopathy to collate and review the best scientific data on the subject. The ACOG Task Force adopted the International Cerebral Palsy Task Force template with modifications.

[251] The pathway documents concern babies born at term which immediately raises a question as to their application to a case, such as the present, of a baby born significantly before term (a point accepted by Dr Kirkpatrick at MS 7033). Moreover, the pursuer would criticise them as too exacting or unduly rigorous (MS 1585, MS 12628 to 12629). A further matter which must be kept very firmly in mind is that notwithstanding the fact that the Task Forces may have had medico-legal objectives ("to help, among others, the courts") satisfying or not satisfying pathway criteria cannot be simply taken as the equivalent of satisfying this court, whose duty it is to determine a specific issue, whether a particular pursuer has established her probandum, on a balance of probability. However, while we shall have to

return to these points, we understood it to be uncontroversial that each of the criteria in the pathway documents has a bearing on the question as to whether a particular case of cerebral palsy may be regarded as caused by an acute hypoxic event. Parties led and cross-examined on evidence with a view either to demonstrating that a criterion was present or its absence was immaterial, on the one hand, or that it was absent, on the other.

[252] The ICP template proposes three essential criteria: (1) evidence of a metabolic acidosis in intrapartum foetal, umbilical arterial cord, or very early neonatal blood samples (pH < 7.00 and base deficit >12 mmol/l); (2) early onset of severe or moderate neonatal encephalopathy in infants of >34weeks' gestation; and (3) cerebral palsy of the spastic quadriplegic or dyskinetic type. It then proposes a further five criteria that together suggest an intrapartum timing but that by themselves are non-specific: (4) a sentinel (signal) hypoxic event occurring immediately before or during labour; (5) a sudden, rapid, and sustained deterioration of the foetal heart rate pattern usually after the hypoxic sentinel event where the pattern was previously normal; (6) Apgar scores of 0-6 for longer than 5 minutes; (7) early evidence of multisystem involvement; and (8) early imaging evidence of acute cerebral abnormality.

[253] The ACOG Task Force adopted the ICP template's distinction as between essential criteria, all of which had to be met if a particular case of cerebral palsy was to have ascribed an intrapartum cause, and additional criteria which suggest intrapartum timing but are non-specific to asphyxial insults. However, it added the exclusion of other identifiable aetiologies to the list of essential criteria and somewhat amended the five criteria that, collectively, suggest intrapartum timing (defined as close proximity to labour and delivery, eg 48 hours). Thus, the ACOG essential criteria were: (1) evidence of a metabolic acidosis in foetal umbilical cord arterial blood obtained at delivery (pH <7 and base deficit =12mmol/L);

(2) early onset of severe or moderate neonatal encephalopathy in infants born at 34 or more weeks of gestation; (3) cerebral palsy of the spastic quadriplegic or dyskinetic type; (4) exclusion of other identifiable aetiologies such as trauma, coagulation disorders, infectious conditions, or genetic disorders. The criteria collectively suggestive of intrapartum timing were: (1) a sentinel (signal) hypoxic event occurring immediately before or during labour; (2) a sudden and sustained foetal bradycardia or the absence of foetal heart rate variability in the presence of persistent, late, or variable decelerations, usually after a hypoxic sentinel event when the pattern was previously normal; (3) Apgar scores of 0-3 beyond 5 minutes; (4) onset of multisystem involvement within 72 hours of birth; and (5) early imaging study showing evidence of acute non-focal cerebral abnormality.

[254] There is therefore a very substantial overlap of relevant criteria as between the two Task Forces. We turn to look at what was the evidence as to the presence or absence of all but one of these criteria in LW's case. We shall consider other possible aetiologies later.

Acidosis

[255] Both Task Forces propose a finding of acidosis (an alteration of the acid/alkali balance of the blood towards acidity, defined as pH measured at <7 and base deficit >12mmol/L at birth or very shortly thereafter) as an essential criterion if a case of cerebral palsy is to be explained as having been caused by intrapartum asphyxia. As we understand it, acidosis is associated with hypoxia because of the production of lactic acid during episodes of anaerobic metabolism (Shah et al, *Recovery of metabolic acidosis in term infants with postasphyxial hypoxic-ischemic encephalopathy*, Acta Paediatrica 92, 941-947, 2003 – MS 9809). Acidosis can be counteracted by the administration of sodium bicarbonate and this is a routine treatment but it does not appear to have been adopted in the hours following LW's birth (MS3920). Equally, hyperventilation, which is a method by which the body can reduce

acidosis through exhalation of carbon dioxide and consequent reduction of carbonic acid in the blood, was not available to LW as he was being ventilated artificially. Accordingly, as her position is that LW suffered an event equivalent to intrapartum asphyxia (an AHII straddling the point of delivery by caesarean section), the testimony of Dr Turner speaking to there having been no evidence of acidosis about the time of birth (MS 6299) would appear significantly to undermine the pursuer's case.

[256] Dr Turner explained his testimony that there was no evidence of acidosis at the time of birth by pointing to the fact that it did not appear to the clinicians treating LW in the immediately antenatal period that there was a problem and to the result of a measurement of LW's blood gases based on a capillary blood specimen taken at about 1845 hours on 8 October, at which time he was being ventilated 90 per cent oxygen. On testing that sample the ph level was found to be 7.38 (otherwise described as a base excess, of 0.3mmol/L) a slightly alkaline and therefore normal result for the acid-base balance of blood gases at the time the sample was taken. Oxygen saturation was normal at 89 per cent (production 7/2/272, MS 6408). That much is not controversial. What came to be controversial was the significance of this result, given that no earlier measurement was available and that therefore there is no finding of acidosis in the umbilical arterial cord or in any other very early neonatal blood sample, as required by the Task Force's criteria. Dr Turner's evidence was to the effect that acidosis at birth could be excluded given the measurement at 1845 and the rate at which he considered an infant was able to correct its acid-base balance. This testimony was given in chief on 26 November 2013 (MS 6299 to 6302).

[257] The way in which further evidence was led on the significance of the passage of nearly two hours between the end of the *ex hypothesi* AHII and the taking of the blood sample, took a particular course is narrated in the pursuer's Outer House submission

("POHS") at MS 12683 to 12692. The topic of acidosis is discussed in the defender's Outer House submission ("DOHS") at MS 13184 to 13216.

[258] It was the pursuer's position that she had been surprised by Dr Turner's evidence. There was some justification for that. In his initial report, dated 26 August 2013 at paragraph 9.7 (MS 12130), Professor Chiswick opined that recovery to a normal acid-base balance within two hours is not inconsistent with an AHII at birth (MS 6425, 12140). On the basis that this seemed about to be departed from, the pursuer had objected to leading opinion evidence from Dr Turner on the significance of the normal measurement at 1845. However, after some discussion, the objection was withdrawn on the pursuer accepting that whether the 1845 measurement excluded acidosis at birth had been put in issue during the evidence of previous witnesses. It is convenient to note how that evidence stood at the point of objection. Prior to 26 November the pursuer had led Drs Kendall, Ferrie and Pearse. All had said something about the significance of the blood gas measurement. Although he acknowledged that the fact was not documented, Dr Kendall had had no doubt that LW would have been acidotic at birth MS (1586). That appears to have been no more than a reiteration of his confidence in the hypothesis that LW had suffered an AHII. Dr Kendall was not challenged on the basis of the blood taken at 1845. Dr Ferrie, consistent with what appeared in his report, stated that while a sample taken within an hour could be helpful, one at two hours after resuscitation was too late (MS 1777). He maintained that position in cross (MS 1960 to 1961). Dr Pearse was cross-examined by reference to the proposition that if LW had had a damaging acute hypoxia it was unlikely that the base excess would have been as it was measured at 1845. Dr Pearse responded by distinguishing between cases of acute profound asphyxia ("APA"), on the one hand, and chronic partial asphyxia ("CPA"), on the other. Babies who suffer an APA recover more quickly than those who suffer from CPA.

This is to be explained by the fact that during CPA there is a substantial build-up of lactic acid in the tissues whereas in an APA there is a relatively small amount of lactic acid formed in the highly metabolic brain cells (Dr Pearse – MS 3930). Accordingly, once circulation and therefore oxygenation are established, a baby can clear acidosis more rapidly in a case of APA than it can in a case of CPA (MS 3919 to 3920). For Dr Pearse, the absence of acidosis at 1845 was an indication that LW's was a case of an APA rather than CPA (consistent with the pursuer's case). He added that there would be low levels of lactate if an infant were hypoglaecaemic because sugar is the substrate for the production of lactic acid (MS 4079). The court had also heard from another of the treating clinicians, Dr Coutts, who, without objection, had been asked about the rate of correction of metabolic acidosis. Dr Coutts considered that if there has been a "very significant event" shortly before birth, one would expect to find some degree of acidosis two hours later (MS 5546).

[259] Given the circumstances, the Lord Ordinary acceded to the defender's motion to be allowed to defer cross-examination of Dr Turner on this point and for leave to recall Dr Ferrie. Both these witnesses accordingly returned to court, on 22 and 23 April 2014 respectively. In the interval before the witnesses' return there was something of an epistolary debate. Dr Ferrie wrote a letter dated 19 December 2013 (MS 9816), referring to the above-mentioned paper Shah et al (2003) (MS 12254). Professor Chiswick responded by way of a (second) report dated 2 January 2014 (MS 12468) which was in turn responded to by Dr Ferrie in a further letter dated 2 February 2014 (MS 9869). Shah et al (2003) reports a study which provides a range of rates of recovery from metabolic acidosis in term infants with post-asphyxial hypoxic-ischaemic encephalopathy. Dr Ferrie's purpose in citing the paper was to demonstrate that at the mean rate of recovery reported in the paper LW could have recovered from a base deficit of 12.13 mmol/L in the 113 minutes available between

resuscitation and taking the relevant blood sample. At the upper level of the range of rates of recovery, LW could have recovered from a base deficit of 15.52 mmol/L. However, as he explained when giving evidence in replication (MS 9158), Dr Ferrie was not using Shah et al (2003) to extrapolate back to any particular base deficit at birth but merely trying to show that it was possible for an infant to make a correction to normal following an acute hypoxic event and to do so within a period of a little less than two hours.

[260] That did not seem different from what appeared in Professor Chiswick's first report at paragraph 9.7, and on returning to court on 23 April 2014 Dr Ferrie confirmed that he agreed with the Professor that a normal acid-base balance at two hours is not inconsistent with an AHII at birth (MS 9104). Professor Chiswick's evidence, given on 28 November 2013 (MS 6595 to 6598), was not however quite so clear-cut. He explained that rate of recovery in base deficit due to a hypoxic intrapartum event depended on the nature and length of the event. It was not impossible to have a baby recovering within an hour. It depended on individual circumstances but if, for example there had been a period of 20 minutes of acute profound asphyxia before birth it would be unlikely that "at one hour things would be ok".

[261] By the time Dr Ferrie returned to court Professor Chiswick's position would appear to have shifted further. His report of 2 January 2014 is in the form of a critique of the application of the results the study in Shah et al (2003) to LW's case: the characteristics of the infants in the study were different from LW (who would not have been admitted into the study) and Dr Ferrie's choice of figures for his back-calculation were inapposite. Redoing the calculation assuming a base deficit of 16 mmol/L at birth, the figure used by Shah et al for entry into the study, Professor Chiswick arrived at a base deficit of 5.3mmol/L at 113 minutes. He then referred to the results of a further study, Murki et al (2004) (MS 12473),

before expressing the opinion that "on the balance of probability" LW did not suffer a brain-damaging asphyxia insult shortly before birth.

[262] When Dr Turner and Dr Ferrie returned in April 2014 their evidence in relation to acidosis was focused on the two papers, Shah et al (2003) and Murki et al (2004). Ill-health prevented Professor Chiswick being recalled (his second report was however received into evidence) with the result that in a sense Dr Turner acted as his substitute whereas Dr Ferrie, who is a paediatric neurologist and not a neonatologist (MS 9148) was the substitute for the pursuer's neonatology expert, Dr Pearse. This led to the criticism of Dr Turner by the pursuer that he was not properly an expert witness (in the sense of a medico-legal expert) and the criticism of Dr Ferrie by the defender that he was giving opinion evidence outwith his field of expertise.

[263] During their evidence in replication neither Dr Turner nor Dr Ferrie moved materially from the positions that they had previously adopted. There was agreement that the ability of an infant to reduce its base deficit depended on the effectiveness of its lungs and kidneys; that the lungs and kidneys of a preterm will be less effective than a term infant with the result that the rate of correction with a preterm will be slower (MS 9124); and that given that his ante-natal oxygen supply was to some degree compromised LW would be at a disadvantage when compared with a previously healthy term infant.

[264] Dr Turner reiterated his view that the normal blood gases at 1845, and also at 2000 (MS 9009) were not consistent with the acidosis to be expected immediately following an AHII.

[265] Dr Ferrie did not accept that he should defer to neonatologists on acidosis (MS 9169 to 9170). In any event, his view was supported by a neonatologist colleague with whom he had discussed the resolution of acidosis. Dr Ferrie emphasised that he was going no further

than expressing the view that the blood gas results were consistent with either party's pleaded case. Shah et al (2003) was from a respected journal published by one of the leading children's hospitals in the world, the Hospital for Sick Children in Toronto (MS 9106); it is used quite frequently in medico-legal cases. The fact that the study group was not preterm means that its results have to be applied with a degree of caution but that does not render them of no assistance (MS 9108). The fact that the study concerns intrapartum deliveries is of no real relevance as it concerned what happens after delivery (MS 9109 to 9110). The fact that the study group all demonstrated encephalopathy is not of great significance because, like LW, these children were artificially ventilated. In Dr Ferrie's view LW could have recovered from a significant state of metabolic acidosis within 2 hours.

[266] The defender submits that it is not anatomically possible for LW to have been acidotic at the time of birth (DOHS – MS 13186). That is not the evidence but equally, there is no clinical finding of a base deficit indicative of an AHII. The pursuer invites the conclusion that the effect of this chapter of the evidence is neutral in that, as Dr Ferrie put it, it is consistent with either party's case. That is not quite how we would view it. Clearly, an infant can clear the base deficit associated with a significant hypoxic event in a period measured in hours. Professor Chiswick was originally prepared to envisage this happening within a period of two hours and we do not have an explanation for his change of mind on that (if he did change his mind) other than what can be calculated from the results of the papers referred to in his second report. We have noted what Dr Pearse had to say about an APA and hypoglaecaemia. There is therefore no question of excluding the possibility of a base deficit at birth consequent on an ongoing AHII, as proposed by the pursuer, simply by reason of normal results at 1845 but the absence of a finding means that the pursuer's case gets no support from this quarter and notwithstanding the pursuer's criticisms we have

been unable entirely to discount the evidence of Dr Turner and Dr Coutts to the effect that they did not suspect acidosis at the time and would not have expected the normal results at 1845 had there been a significant event at birth. It is of course true that it is to be expected that LW would be acidotic immediately after resuscitation, irrespective as to whether there was a longer AHII commencing prior to delivery and causing all the brain damage demonstrated on MRI, as contended for by the pursuer's witnesses, or a shorter AHII commencing after delivery and damaging only the putamina, as suggested by Professor Chiswick. However, Professor Chiswick was not contradicted when he said that time required for consequential acidosis to be cleared depends on the length and severity of the relevant hypoxic event. If that is correct, and it would seem to make sense, then the defender is entitled to argue that the absence of acidosis at 1845 is more consistent with the relatively short AHII proposed by the defender and less consistent with the longer AHII proposed by the pursuer.

Encephalopathy

[267] In the present context encephalopathy means clinical signs of malfunction of the brain (Dr Ferrie at MS 1780 to 1785). Dr Turner stated that in LW's case there had been no such signs (MS 6298). That was the understanding of Professor Chiswick (MS 12469). On his review of the records Dr Ferrie concluded that there were some features which suggested encephalopathy, including unexplained oxygen desaturation and bradycardia which *may* be manifestations of epileptic seizures in babies, but that there was insufficient information to state this with certainty and accurately to grade it (MS 1783). Dr Pearse was more inclined to surmise that incidents of agitation might be explained as convulsions (MS 3607 to 3611, 3764, and 3796), albeit that in his report at page 57 (MS 9668) he states: "[LW] did not really

show incontrovertible signs of hypoxic ischaemic encephalopathy but one does not necessarily expect these in babies at 32 weeks gestation".

[268] As far as the Task Force criteria are concerned it will be recollected that both ICP and ACOG pathway documents would suggest this criterion is only of relevance to babies of more than 34 weeks gestation and both in his report and in evidence the defender's neonatal expert, Professor Chiswick, expressed the view that unlike term infants preterm infants rarely show typical clinical features of hypoxic-ischaemic encephalopathy (MS 12140, 6604 to 6606). Dr Kirkpatrick, the paediatric neurologist, on the other hand expressed the opinion that assuming there to have been an AHII it cannot have occurred at the time of birth because of the absence of significant neonatal encephalopathy (MS 7033). This was notwithstanding his being cross-examined by reference to the pathway documents (MS 7179). He considered it difficult to conceive of a situation where a pathway of damaging hypoxic ischaemia severe enough to cause neurological damage would not include encephalopathy as part of the pathway (MS 7175). This was based on his clinical experience as a paediatric neurologist.

[269] We therefore consider that whether LW demonstrated neonatal encephalopathy is conjectural at best but we are not inclined to exclude the possibility of an AHII on that basis alone. While that is the direction pointed to by Dr Kirkpatrick's evidence his appears to be a minority position.

Type of cerebral palsy

[270] The evidence as to the type of LW's cerebral palsy is discussed above. Both Dr Ferrie and Dr Kirkpatrick identified spastic and dyskinetic features. Dr Ferrie described LW as suffering from quadriplegia. If that assessment is accepted then this pathways criterion is met in each of its elements. Dr Kirkpatrick however would describe LW's condition as

diplegic. We see him as getting support from Dr Zuberi's assessment of LW's condition. However, this is a difference that we cannot resolve.

Sentinel event

[271] As previously observed, the pursuer's candidate as the sentinel event is a retroplacental abruption which occurred, to use Dr Howatson's terminology, "within a few hours of delivery". The qualification as to timing is important. Whilst there would seem to be no doubt but that the pursuer suffered an abruption, the longer the time gap between the abruption and the supposed collapse the more difficult it would appear to be to infer a direct causal connection. If the abruption cannot be pointed to as the direct cause of the process leading to an AHII it does not qualify as a sentinel hypoxic (or hypoxia inducing) event as that expression is used in the pathway documents.

[272] On balance, the more recent of the abruptions described by Dr Howatson appears to us to be a rather weak candidate for a sentinel event in the sense of a cause clearly associated with a result. It was only partial. While there is no question but that it occurred, it is not known when it occurred. Nor was there evidence as to how precisely it was said to have given rise to the posited circulatory collapse.

Cardiac function

[273] One of the pathway criteria is a sudden, rapid, and sustained deterioration of the foetal heart rate.

[274] It is not controversial that LW's heart rate immediately after delivery was less than 60 bpm (MS 10464). However, as we have previously noted, Dr Coutts gave evidence that the cyanosed appearance of LW at delivery was an indication that at that point he had a peripheral circulation (MS 5497). Dr Coutts was not present at delivery (MS 5489); he became directly involved later (Dr Gallagher was the paediatrician who took immediate

charge of LW, she was a house officer, Dr Coutts was the consultant on call). However, Dr Coutts disputed that the condition of LW on delivery, as reported to him, was consistent with being in a state of circulatory collapse. When asked "In your view, did this baby, under the care of your unit, have evidence of a circulatory collapse at birth?" he replied:

> "No, in that we would expect a baby at that extreme to be, as we mentioned, very pale, very floppy. You might get a good heart rate response within this time period because you've managed to get the baby to have some respiratory support, but we expect the baby to remain, if it had circulatory collapse, to remain pale for some time and slowly pink up in a very strange blotchy sort of way, when you're kind of seeing a baby get better, but to remain …floppy and inactive." (MS 5520).

In other words, in the opinion of Dr Coutts, LW did not present as would be expected of an infant suffering and then recovering from a circulatory collapse.

[275] While Dr Coutts's evidence may challenge the pursuer's characterisation of LW being in a state of circulatory collapse, if one is simply concerned with the question whether LW's case met the pathway criteria then the answer would have to be in the affirmative; there was bradycardia. The six-minute postnatal bradycardia was a sudden, rapid, and sustained deterioration of the foetal heart rate but of course the defender is prepared to accept that this was probably a brain damaging event, albeit a postnatal brain damaging event.

Apgar score

[276] LW's Apgar scores were recorded as 3 at one minute (corrected to 4 – MS 6394), 4 at five minutes and 7 at 10 minutes. That meets the 0-6 for longer than 5 minutes ICP criterion. It does not meet the 0-3 beyond 5 minutes ACOG criterion. Dr Kendall accepted that the Apgar scores pointed to any AHII not having been very long (MS 1565).

Multisystem involvement

[277] Dr Kendall confirmed that asphyxia can lead to adverse effects on organs other than the brain, although there could be brain damage without the involvement of other symptoms (MS 1576). Professor Chiswick's understanding was that LW did not develop multisystem involvement (MS 12469). The evidence of Dr Coutts was to the effect that the results of blood tests were within normal parameters, as was urinary output, negating liver or kidney damage and therefore dysfunction (MS 5547). Dr Pearse, on the other hand, pointed to indications in the records of kidney dysfunction (MS 3626, 3949 to 3950, 9671), albeit that Dr Ferrie, perhaps straying outwith his particular area, described the evidence of multisystem hypoxic damage as equivocal (MS 2108). We take Dr Ferrie to acknowledge that such damage would be expected (see paragraph 3.60 of his report - MS 9559). However, he offers the information that ischaemic insults are known to cause brain damage in a minority of subjects without there being clear evidence of multisystem hypoxic damage, something with which Dr Pearse, who challenges the validity of this as a pathway criterion would agree (MS 9671).

[278] We cannot find multisystem involvement to have been established but we have no reason to reject the evidence of Drs Kendall, Ferrie and Pearse to the effect that this is not an inevitable consequence of brain-damaging hypoxia. Accordingly, while the pursuer's case gets no support from this quarter, neither is it necessarily undermined by the absence of damage to other organs.

Imaging

[279] The templates include early imaging study showing evidence of acute non-focal cerebral abnormality as an additional criterion. In LW's case the MRI scan was not carried out at an early stage but ultrasound scanning was. Dr Connolly spoke to the ultrasound

scans showing (non-focal) swelling which we would understand to be consistent with a recent hypoxic insult. Dr Kendall considered that the cranial ultrasound scans carried out on 9, 14, 23 October and 8 November 1996 were consistent with what appeared on the later MRI scans (MS 1372). To that extent the criterion is met. However, the question then arises as to what abnormalities are shown on the MRI scans, and what is their significance. This is discussed above.

Alternative causation

The issue

[280] It will be recalled that one of the ACOG essential criteria is the exclusion of other identifiable aetiologies such as trauma, coagulation disorders, infectious conditions, or genetic disorders. We shall have more to say about this but this is not how we have gone about our decision making. It would be to impose an inappropriately high standard on the pursuer to require her to prove that there is no other possible explanation for LW's condition than an AHII. However, quite independent of the pathway documents, it obviously would assist the pursuer's case were we to find that all the suggested alternative causes were, to a greater or lesser extent, unlikely. Equally, were we to find that one or other of the suggested alternatives was a very possible cause of LW's disabilities that would assist the defender.

[281] Among the possibilities is that more than one cause was operative.

Genetic

[282] LW has a number of dysmorphic features but Dr Kendall described his brain as normally formed (MS 1463). In her report Professor Murphy states at 7.1, that:

> "From the information available to me, there does not appear to be any strong evidence of an underlying genetic, metabolic, or structural aetiology for

> developmental delay in [LW's] case. There was no labour. The possible origins of his cerebral damage lie in the antenatal and neonatal period" (MS 11392).

[283] Drawing support from Dr Kirkpatrick's evidence, the defender argues that a genetic explanation (perhaps in the form of a "private" syndrome, described by Professor Chiswick as "a cluster of deviant features that do not fit into a recognised syndrome") for LW's disabilities cannot be excluded. While that may be so, the evidence for a genetic explanation would seem slight. It is not an explanation which commended itself to Professor Chiswick (MS 6468, 12138).

IUGR and prematurity

[284] As we have already noted, LW had a birth weight consistent with that of a normally grown foetus of 26 weeks gestation. In other words he had failed to develop normally. We take it to be accepted that this was due to placental insufficiency and consequent IUGR, hence Professor Murphy's description of LW as a severely compromised foetus. To an extent IUGR secondary to placental insufficiency is a chronic hypoxic process, although the foetus is dependent on the placenta for other nutrients which are essential to its development. Dr Pearse confirmed that the evidence was that LW had suffered a very severe shortage of nutrients for a period well before 8 October 1996 (MS 3892). Professor Chiswick was to similar effect (MS 6632). As we have also already noted, Professor Murphy confirmed that the result of the study published in the Lancet (*Case-control study of antenatal and intrapartum risk factors for cerebral palsy in very preterm singleton babies*, The Lancet vol 346 p1449, 6/157 of process - MS 9829), was that while there is an underlying risk of placental abruption and consequent brain damage associated with pre-eclampsia (MS 8863) and IUGR there was no increased risk of cerebral palsy associated with IUGR (MS 8858). However, notwithstanding this particular finding, it seemed to be generally accepted among the

witnesses who were asked about it, that a relationship of cause and effect exists whereby placental insufficiency may inhibit the normal development of the foetus and in particular the foetal brain in ways which have a permanent impact on motor and cognitive function; manifesting themselves as, among other things, cerebral palsy and learning difficulties (eg Dr Ferrie – MS 3137). Professor Draycott confirmed the statistical association between growth restriction and cerebral palsy (MS 2824). Dr Connolly accepted that independent of brain damage there "can be a relationship" between IUGR and cerebral palsy (MS 5768). Dr Kirkpatrick was more forthright. On his evidence cerebral palsy is sometimes the result of an infant being IUGR and preterm (MS 6965, 7012). He referred to the paper by Shah and Kingdom *Long-term neurocognitive outcomes of SGA/IUGR infants*, Obstetrics, Gynaecology, Reproductive Medicine 21:5 p142 (2011) (MS 12254) and to the chapter by Dr Paul Eunson, Placental Pathology, Intrauterine Growth Restriction and Subsequent Child Development in Baker and Sibley, *Clinics in Development Medicine, The Placenta and Neurodisability* (2006) where there is a discussion of various disabilities associated with IUGR, including cerebral palsy (MS 12234). That is not to say that IUGR will necessarily have an adverse long-term outcome but it may have. In LW's case Professor Walker offered the opinion based on his "experience and knowledge" that while some factors may relate to an acute hypoxic event, IUGR was enough to explain virtually all of the findings (MS 7795). Prematurity was an independent risk factor (MS 7769). Dr Kirkpatrick's position was somewhat different. He did not ascribe all of LW's disabilities to prematurity and IUGR (MS 7125), albeit that he suggested that they might be attributed to growth restriction in the early weeks of pregnancy. As we shall go on to discuss, he attributed the grey matter damage to acute postnatal hypoxia.

Chronic hypoxia secondary to placental insufficiency

[285] In the present case chronic hypoxia, in the sense of a reduced or suboptimal supply of oxygen to the foetal brain over a period well in excess of the hour or so of "chronic partial asphyxia" discussed by Dr Pearse, overlaps with IUGR, as a potential explanation for LW's disabilities. As Dr Ferrie said in his report: "The possibility that [LW's] neurodevelopmental problems could have arisen as a consequence of placental insufficiency requires serious consideration" (MS 9554). To point to IUGR is to put the stress on failure to develop. To point to chronic hypoxia is to put the stress on damage. In any event chronic hypoxia merits separate mention given the uncontroversial Doppler readings recorded by Dr Crichton and the pathology of the placenta as reported on by Dr Howatson. The pursuer acknowledges this evidence but relegates it to the background while bringing to the foreground a posited abruption a few hours prior to delivery followed by a circulatory collapse during the "dark period". Whether that is the correct priority is a matter we shall have to consider but there can be no doubt but that LW was being inadequately oxygenated over a significant period prior to his birth. The Doppler readings provide a snapshot at about 1300 on 8 October. Professor Draycott interpreted them as demonstrating intermittent absent end diastolic flow, in other words a cycle which included periods where there was no blood flow whatsoever to the foetus. Professor Draycott was concentrating on the (single) umbilical artery at a point in time some four hours prior to delivery but the pathology of the placenta reported on by Dr Howatson, and in particular his finding of a widespread excess of syncytial knots, indicated a longer standing structural problem adversely affecting the mechanisms for the transfer of oxygen (but also, for example, glucose) from the maternal circulation to the foetal circulation and thence to the foetal brain. This is quite separate from the incidence of abruptions. Dr Howatson found evidence for two abruptions. He opined that they both

had occurred within four or five days of delivery. They will have further compromised the ability of the placenta to transfer oxygen and nutrients to the foetus but, if Dr Howatson was correct on timing, this will have only come towards the end of an extended period of damaging placental dysfunction, as demonstrated by the excessive number of syncytial knots in the placenta and the consequent retarded growth of the foetus. That LW's BPS was scored at zero and his CTG was unreactive are consistent with neurological impairment or at least LW being "not physiologically normal" as at the time of his admission to hospital (see Professor Walker - MS 7478, 7500; Professor Draycott - MS 2759). Chronic hypoxia due to placental insufficiency would explain why that was so.

Acute hypoxia secondary to the stress of delivery

[286] That LW was in a state of bradycardia at birth with an absence of respiratory effort is not controversial but the defender does not accept that he suffered from an AHII, or at least does not do so without the qualification that it did not begin before delivery and accordingly did not last more than six minutes. The defender points to the view of the treating paediatricians that while LW had had "a poor start to life" there had been no acute event (MS 6155, 6220). The post-delivery bradycardia could be explained by the stress of being born (Dr Coutts - MS 5516). If that were so LW would be exposed to hypoxia but only for a period of some six minutes. That raises the question, discussed above, particularly at paragraphs [217] to [221], whether the uncontroversial damage to the putamina and the deep white matter might have been the result of six minutes without oxygen? This does not admit of an unequivocal answer. In his report Dr Connolly identifies a number of variables which may affect the outcome of a given period of hypoxia including, "severity, duration, amount of reserve and gestational maturity" (MS 5751). What Dr Connolly there describes as "reserve" was described elsewhere in the evidence as resilience. If LW had had the

resilience of a normal healthy foetus at term and assuming that his reaction to hypoxia conformed to what has been taken from the Myers studies we would understand that it is unlikely that he would have sustained the damage demonstrated on MRI following a six-minute period of hypoxia. However, on any view LW was not a healthy foetus. Developmentally he was not normal. He was very significantly preterm. He may have had no resilience whatsoever. Accordingly, it would appear to be at least possible that six minutes of hypoxia might have been sufficient to cause the demonstrated damage (Dr Connolly – MS 5791, 5862; Dr Ferrie – MS 9186; Professor Chiswick – MS 6393 to 6394, 6698 to 6699, 6697 to 6698).

[287] At this point we should say something about the evidence of Professor Murphy and Professor Walker, who were led by the defenders as experts in obstetrics; Professor Murphy is a perinatal obstetrician and Professor Walker is a specialist in high-risk obstetric cases. Professor Murphy explained that her views were based firmly on clinical experience and explanations that had been derived from her clinical experience, rather than epidemiological studies, although as already indicated she made some reference to these. She repeatedly emphasised that this was a severely compromised foetus. That view is supported by the fact that LW was distinctly small for dates at the time when he was born, and must have been so for some time before that. Professor Murphy stated that acute placental abruption is usually associated with a fall in haemoglobin and platelet count and coagulation dysfunction with abnormally low fibrinogen. In the pursuer's case, however, all of the blood tests were entirely normal and not in keeping with either severe early onset pre-eclampsia or acute onset placental abruption (MS 8481 to 8482). Consequently the blood tests were not consistent with an abruption that was evolving at the times when they were taken.

[288] Professor Murphy's general position was that LW's disability was the result of complex causative factors (MS 8680 to 8685). She thought it inherently implausible that the primary causative factor would be a ten-minute insult of a hypoxic ischaemic or asphyxiated nature immediately prior to birth. Overall it was difficult to reach a clear diagnosis, and that appeared in keeping with more than one insult. Moreover, the clinical findings were multiple: a chronic prolonged insult in the antenatal period, which is often associated with white matter damage, and multiple episodes in the neonatal period, which are also associated with white matter damage. In explaining cerebral palsy, she would not include a hypothesis that related to the last 10 to 30 minutes prior to birth, especially as the CTG recording in its last 10 to 15 minutes prior to delivery was unchanged virtually from what was shown at admission. Professor Murphy could see no evidence of an acute event in the 30 minutes preceding birth. Moreover, LW was very compromised at the time of admission to hospital, from chronic events and a previous placental abruption. Professor Murphy thought that his brain was already damaged prior to presentation at hospital, but her second preferred hypothesis was that six minutes of circulatory collapse might be the critical event that led to the basal ganglia damage against a background of existing white matter damage from antenatal events (assuming no resilience).

[289] Professor Murphy further described the striking symptoms that are standard in cases of pre-eclampsia (MS 8702 to 8703). In such cases the woman generally presents herself to hospital, and once symptoms exist she generally deteriorates rapidly and delivery is required; the symptoms are a clear marker of deterioration. Professor Murphy thought that it was entirely speculative whether blood pressure and urine would have been abnormal had the pursuer been referred to the hospital on Saturday 5 October (MS 8710 to 8713). If the CTG had been abnormal at that stage, the pursuer would probably have been

delivered by emergency Caesarean section, and it is likely that the neonatal outcome would have been the same as actually occurred. Placental ischaemic disease had resulted in an extremely small, symmetrically small baby with a small brain and low birth weight; that was an important component in the risk. In addition, placental abruption with a retroplacental clot had already occurred by the time of the admission. The CTG might well have been abnormal at the time of admission.

[290] Professor Murphy further commented on Dr Kendall's report (MS 8742 to 8743 and MS 8874 *et seq*). She considered that too much emphasis was being placed on intrapartum asphyxia rather than events at an antenatal stage. She considered this to be a flaw in Dr Kendall's approach which had misled other witnesses; there had been no labour in this case, and consequently no help would be gained by looking at studies based on intrapartum asphyxia. Finally, Professor Murphy referred to the statistical evidence discussed in Murphy et al (1995), the paper mentioned at paragraph [173] above.

[291] Professor Walker, like Professor Murphy, placed considerable stress on LW's condition prior to 5 October. He stated (report at MS 12185 et seq) that LW was significantly growth restricted with the weight equivalent of a baby of around 27 weeks' gestation, five weeks less than his actual gestation. His head was small and continued to be so after birth, which suggested symmetrical growth restriction. That was likely to have been a long-standing problem, lasting for weeks or months prior to delivery, and not the result of a recent insult or fall off of growth. Furthermore, LW was born with a low heart rate and no respiratory effort, and required intensive care at the time when he was born, with a low initial Apgar score. Overall, Professor Walker thought that LW's mixed pattern of abnormalities suggested an underlying abnormality and long-term intrauterine growth deficiency rather than an immediate pre-delivery acute hypoxic insult. Furthermore, the

pursuer's signs and symptoms at the time of the birth were generally not indicative of pre-eclampsia, apart from complaints of blurred vision at 22.30. On this basis, Professor Walker thought that an earlier delivery, even days previously, would in all probability not have made any substantial difference and might, due to increased prematurity, increase the risk of death or disability (report, paragraph 9.7).

[292] Professor Walker considered that the pursuer did not have pre-eclampsia by any definition, but at most an acute elevation of blood pressure which was easily controlled by doses of labetalol. Women with established pre-eclampsia normally have ongoing problems after delivery, often with worsening signs. Professor Walker provided an elaborate analysis of the events immediately preceding and surrounding LW's delivery. His overall conclusion was that LW suffered long-standing intrauterine insults from either congenital abnormality, infection or gross placental insufficiency or a combination of these. He thought that the problems were of long-standing: six weeks and probably more prior to 5 October. Initially he expressed the view that no acute event *in utero* contributed or caused the problems (report, section 13). In examination-in-chief, however, he was referred to the information relating to the MRI scan, and he accepted that it contained some evidence of an acute hypoxic event which had occurred prior to 8 October and which had caused some of the factors affecting LW (MS 7794 to 7795). Nevertheless, the primary pathology was severe growth restriction, and IUGR and severe placental dysfunction provided the major insult that started the whole process resulting in LW's injuries (MS 8143, 8175 to 8176).

[293] The opinion evidence of Professor Murphy, in particular, and also to some extent Professor Walker is of importance in confirming our understanding of the import of the essentially uncontroversial primary evidence in two respects. First, it reinforces what is suggested by the primary evidence which is that all or nearly all of the features of LW's

presentation can be explained by placental insufficiency and IUGR, having effect well before the events of 5 to 8 October. Professor Murphy's evidence does not appear to us to have been undermined in any significant respect, and it is based on her extensive experience in the obstetric field, as supplemented by limited reference to epidemiological studies. Professor Walker conceded that his evidence was affected by the MRI scan, but like Professor Murphy he stressed the importance of IUGR and placental insufficiency in explaining LW's injuries. LW's condition can, on obstetric evidence, be explained by long-standing problems which pre-dated his delivery. Secondly, the fact that experienced obstetricians attribute the injuries to long-standing causes rather than an acute event immediately before the time of birth is a significant factor in assessing whether the pursuer has proved a case on a balance of probabilities. Theirs is an interpretation of the primary evidence, founded on the relevant science and not undermined by cross-examination, which can be relied on by the defenders in support of the application of the approach to proof of causation exemplified by *Rhesa Shipping Co SA* v *Edmunds ("The Popi M")*, [1985] 1 WLR 948, discussed below at paragraph 297.

LW's stormy neonatal course

[294] The defender draws attention to various insults that LW suffered in the neonatal period including incidents of apnoea, incidents of bradycardia, necrotising enterocolitis, persistent ductus ateriosis, jaundice and respiratory distress syndrome (DOHS MS 13319 to 13330). It submits that the cumulative effect of what it describes as LW's "stormy neonatal course" is clearly associated with a considerable risk of brain damage.

[295] We have noted the defender's submission. Our impression of the evidence is that it did not go beyond identifying that the various conditions mentioned by the defender which LW would appear to have suffered from were recognised risk factors for brain damage and

cerebral palsy. In contrast to the attention given to IUGR, placental insufficiency and the significance of the six-minute period of bradycardia, the possible consequences of the various conditions which LW experienced during the 99 days he spent in hospital following his birth were not explored in very great detail. As a result there is simply not the material available to elevate the various risk factors to which LW may have been exposed in the first weeks and months of his life into possible causes for his cerebral palsy.

[296] As the defender recognises (MS 13320) there is a further difficulty for an argument that regard should be had to LW's neonatal experience. It arises from the appearance of the cranial ultrasounds carried out on 9, 14, 23 October and 8 November 1996. These ultrasounds are at least suggestive of the presence of brain damage at their respective dates, the nature of which is consistent with what is shown in the later MRI scans (Dr Kendall – MS 1372).

Decision on the second causation issue

General method

[297] As is familiar, the onus is on the pursuer to prove her case. The standard of proof is on a balance of probabilities. That is not a very high standard; all that it amounts to is just more likely than not (what Lord Prosser in *Dingley* (p 603F) describes as "marginal probability"). Nevertheless, while scientific proof is not required, in so far as matters are at large for this court, as the second causation issue is, the pursuer must satisfy us that the essential facts upon which her case relies probably occurred. If she does not do that she fails. That is so irrespective of what view the court might take about the alternative possible explanations of LW's cerebral palsy that have been put forward by the defender. Just because the defender has advanced alternative explanations does not mean that if it fails to

prove any of them, the pursuer must succeed. As was recently discussed in *McGlinchey* v *General Motors UK Ltd* [2012] CSIH 91, in *Rhesa Shipping Co SA (The Popi M)* at p 951, Lord Brandon said this in the course of a speech with which the other members of the Judicial Committee agreed:

> "…it is always open to a court, even after the kind of prolonged inquiry with a mass of expert evidence… to conclude… that the proximate cause of the ship's loss, even on a balance of probabilities, remains in doubt, with the consequence that the shipowners have failed to discharge the burden of proof…".

[298] If the pursuer is to succeed she must therefore satisfy the court that the balance of probability tips in her favour but in order to prove her case she need not prove every one of her averments, or at least not to that standard. What she requires to prove are the essential facts. The essential facts in this case can be stated as a proposition: that LW's condition was caused by brain injuries sustained as the result of an AHII which began within the period of 20 or so minutes before he was born and which ended when he was about six minutes old. It is to that proposition which we must apply a probabilistic test. We take the expression "probabilistic test" from the judgment of Sedley LJ in *Karanakaran* v *Secretary of State for the Home Department* [2000] INLR 122 at 152, where he observes:

> "…a civil judge will not make a discrete assessment of the probable veracity of each item of the evidence: he or she will reach a conclusion on the probable factuality of an alleged event by evaluating *all* the evidence about it *for what it is worth*. Some will be so unreliable as to be worthless; some will amount to no more than straws in the wind; some will be indicative but not, by itself, probative; some may be compelling but contraindicated by other evidence. It is only at the end-point that, for want of a better yardstick, a probabilistic test is applied. Similarly a jury trying a criminal case may be told by the trial judge that in deciding whether they are sure of the defendant's guilt they do not have to discard every piece of evidence which they are not individually sure is true: they should of course discard anything they think suspect and anything which in law must be disregarded, but for the rest each element of the evidence should be given the weight and prominence they think right and the final question answered in the light of all of it. So it is fallacious to think of probability (or certainty) as a uniform criterion of fact-finding in our courts: it is no more than the final touchstone, appropriate to the nature of the issue, for testing a body of evidence of often diverse cogency."

[299] The context of *Karanakaran* was an immigration appeal where the issue was whether the appellant had a well-founded fear of persecution. It might be said that that is more a matter of the evaluation of facts than the finding of facts. However, in the passage quoted Sedley LJ is speaking generally about the process of civil and criminal fact-finding. The process that he describes is not one where a piece of evidence requires to be established on the balance of probabilities before it can contribute to the final decision. Rather, every piece of evidence, including reasonable inferences from facts or suggested facts, whether tending to support what is averred or tending to controvert what is averred, must be considered and, unless it is entirely rejected as incredible or unreliable, it must be given its appropriate weight and put on the relevant side of the balance. All the possibilities suggested by a review of the evidence, and the support that one possibility may give to another possibility, remain in play until the point when the decision is made arrives. Only then is the probabilistic test applied to the essential facts.

[300] Lord Prosser was describing a similar process in a passage from his opinion, as Lord Ordinary, in *Sodden* v *The Prudential Assurance Co Ltd* which was quoted by the Lord Justice Clerk (Ross) when delivering the opinion of the court in the reclaiming motion (unreported, 15 January 1999):

> "It is not easy to hold that the defenders have discharged the onus of proof which lies upon them ...One must however take all possibilities together, rather than in isolation from one another. And in doing so ... I find myself compelled to the opinion that the defenders have, on a balance of probabilities, established that the fire was indeed started by the pursuer."

[301] That is the process we have attempted to apply: having regard to all the possibilities before determining what we consider probable.

Expert evidence

[302] With the exception of the pursuer and her husband all of the witnesses had a particular expertise in the fields of medicine and midwifery. However, some of these "experts" were clinicians who, in one way or another, had been involved with the care and treatment of the pursuer and LW and who were called to speak to matters of fact. The others were experts as that expression is more commonly used in the context of litigation, in other words medico-legal experts or forensic experts who were called to give opinion evidence. Two general matters were raised by parties in their respective submissions which we shall address now.

[303] The first matter is whether the treating clinicians who had been called to speak to matters of fact (specifically Drs Turner, Coutts, Hanretty, MacLean, McLelland, Maclennan and Zuberi) should have been asked questions designed to elicit opinion evidence on causation. The pursuer submitted that, on a number of occasions, the defender had sought to do this, notwithstanding objection (eg MS 5527, 5685). This, it was said, was inappropriate and unhelpful for a number of reasons. First, the treating clinicians could not be said to be objective, they are too close to the action and to colleagues who might be being criticised for the standard of their care. There was a real risk of unconscious bias. Second, allowing the treating clinicians to give opinion evidence was unfair where, as in the present case, they had not provided reports and the pursuer's representatives had not been permitted to precognosce them unsupervised; there had been no prior notice of their evidence. Third, the treating clinicians had not prepared themselves to give evidence by applying their minds to the particular role of medico-legal expert providing expert opinion evidence and taking the steps which were necessary for the carrying out of that exercise. These steps would include (1) reading all the medical records, not just those which concern

their involvement in the case or those from their particular department, (2) writing a report which complies with the professional requirements imposed by the General Medical Council in order to ensure objectivity including taking account of alternative points of view, and (3) reading all the relevant expert reports in the case before giving evidence, again to ensure objectivity. A consequence of that was that the treating clinicians had not seen all the records (a point raised by the Lord Ordinary *ex proprio motu* at MS 2395) and they had not had the opportunity to consider the opinions given by the medico-legal experts. It was no answer to suggest that the treating clinicians might be well placed to provide an opinion because they had been there at the relevant time (that is where they had been there; in many instances the treating doctors were not actually in attendance at the events as to which they were asked to give opinions). Clinicians could not be expected to recall the details of their cases after a period of years; all they can do is work from the notes in the same way as the medico-legal experts do (cf Dr Pearse - MS 3842).

[304] We see force in the pursuer's submission. We may have reached or may be about to reach a stage in the development of our civil procedure where prior to the leading of evidence parties are required formally to nominate such witnesses as they wish to rely on as their forensic experts in the expectation that their evidence will be given in a more structured way and more under the direction of the court than has formerly been the case. In cases of clinical negligence that can be achieved through operation of the case management provisions of chapter 42A of the Rules of Court. However, chapter 42A was only introduced with effect from 1 May 2013 and as far as the present case is concerned we were not advised of any orders in relation to expert witnesses having been made in the Outer House (in any future case of this sort we would expect a very different course to be followed indeed, one of the consequences of the approach taken in this case has been the

provision of more detailed guidance as to how such cases should be conducted, in PN No 5 of 2017). In the absence of such orders, it is difficult to see how any successful objection could be made to the admissibility of evidence of opinion given by any of the treating clinicians and relating to their own particular area of skill. Indeed, it might be necessary for them to express opinions when elucidating what are essentially matters of fact. Nevertheless, it seems clear from the Lord Ordinary's opinion that he recognised those listed in paragraph [4] of that opinion as being the experts put forward by the parties as authoritative on the obstetrical, neurological, radioneurological, neonatal and midwifery issues (what we have referred to as the medico-legal or forensic experts) and that the Lord Ordinary treated them accordingly. That being so we see it as appropriate to give their evidence, and particularly their opinion evidence, a weight which reflects the role given to them by parties and which was recognised by the Lord Ordinary. Similarly, when considering expressions of opinion by the treating clinicians we will have regard to their context and the expectation that the witness will not have had much in the way of opportunity to reflect on the matter he or she is asked to give an opinion about. Again that goes to weight. However, there is also the question of fair notice, as is illustrated by the discussion about the evidence on the significance of the measurement of blood gases two hours after delivery. The expectation is that where a party intends to lead a witness in order to give expert opinion that party will instruct a report from the expert which will indicate what the opinion is and a reasoned explanation of why it has been arrived at. Once lodged, the report gives the other party notice of the expert's position in a way that cannot always be done with pleadings. The other party can plan its response accordingly. This should focus the issues and make for a rational approach to their resolution. That structure is disrupted where a perhaps entirely well-qualified treating clinician, led to speak to the facts and who

has accordingly not provided a report, is allowed to offer opinion evidence on a matter on which the parties are at issue.

[305] The second matter raised in submission can be identified by the expression "the multidisciplinary approach" but clearly parties take very different views as to its salient features and why it is of importance. For the pursuer, it is a virtue and it strengthens her case. For the defender, it is a vice and it undermines the pursuer's case.

[306] What is referred to as the multidisciplinary approach relates to the preparation and presentation of the pursuer's case and in particular the collaboration among the pursuer's forensic experts at the preparation stage. Counsel for the pursuer commended the multidisciplinary approach. A collaborative approach had been adopted by the experts led for the pursuer in addressing the issue as to whether LW's cerebral palsy was the result of his experiencing an AHII. It reflected what is done in clinical practice where experts from different specialisms frequently work together in searching for the cause or causes of cerebral palsy in an individual child, as was explained in evidence by the pursuer's experts, Drs Kendall, Connolly and Pearse and acknowledged by Professors Chiswick and Murphy who had been led for the defender. No one person is an expert in all facets of the condition. In a medico-legal context a variety of evidence-based opinions from a range of specialists may be needed to elucidate the possible causes of the cerebral palsy in the case before the court (MS 12620 to 12621).

[307] The defender's perspective is different. It contends that the way in which the case was prepared by the pursuer's experts significantly detracts from the weight of their testimony taken as a whole. The defender submits that in order for the pursuer to be able to prove her case, it is essential that she provides the court with carefully considered and truly independent expert evidence on the relative issues, in accordance with the principles

recognised in the leading authority of *National Justice Compania Naviera SA* v *Prudential Assurance Co Ltd (The Ikarian Reefe*r) *(No 1)* [1993] 2 Lloyd's Rep 68 at 81 and adopted for Scotland by the United Kingdom Supreme Court in *Kennedy* v *Cordia (Services) LLP*. According to the defender the pursuer's experts had collaborated to the point where each came to the court ready to give evidence in support of the entirety of the pursuer's single insult theory (as the defender described the pursuer's case), rather than on the aspects which fell within his or her specific remit. In fitting the different disciplines together, the pursuer's overall presentation had fallen foul of gaps between the disciplines, misunderstandings about the opinions of the other experts and a frequent willingness on the part of her experts to step beyond their fields. The fitting together of the expert evidence is a matter for the court. As Professor Murphy had explained, the "collaborative" approach which the pursuer's experts had purported to take, was in fact not that at all. In her opinion, the pursuer's expert witnesses had been misguided by an over-reliance on the contents of Dr Kendall's report (Dr Connolly, for example, accepted that he had taken all the facts of the case from Dr Kendall – report 6/138p1, MS 9728), a report which was not the entire answer but only a part of it and which did not, in any event, turn out to represent Dr Kendall's views as presented in evidence. A proper multidisciplinary approach requires an assessment of what each discipline should reasonably be expected to contribute to it. The pursuer treats the radiology as decisive. In contrast, the evidence of the defender's experts constitutes a detailed analysis, within each expert's area of expertise. It is not an attempt to make the facts fit a preconceived notion about the case or to make them fit the subjective perceptions about what one sees on the MRI scan (MS 13989).

[308] What this difference of opinion appears to be about is collaboration. In particular, it is about the process of putting together a case on behalf of the pursuer, the initial impetus of

which was provided by a reading of the MRI scan. While we would not consider the point as remarkable as counsel's insistent repetition of it might suggest, we would accept that the case for a particular causal connection is made stronger where it is supported by evidence from different interlinking perspectives. How her attendance at consultations may have coloured the pursuer's recollection of the events of October 1996 is a different point but, without seeing the need for going into details, we consider there to be nothing untoward in a party developing her case from a working hypothesis with a particular evidential base by seeking to recruit experts from other disciplines to support it. We would suppose that, *mutatis mutandis*, that is how litigation is often conducted. We suspect that this is not the same as a multidisciplinary approach to a clinical problem where we would expect the perspective to be continuously sceptical and questioning. In a clinical context the objective will be to determine the correct diagnosis and to find an effective treatment. That is likely to involve adopting an initial hypothesis or initial hypotheses and then testing it or them against the available evidence as it emerges, applying insights from all relevant and available disciplines. We would suppose clinicians always to be ready to abandon a hypothesis when the evidence or the insights of colleagues suggest a more robust alternative. That is not how litigation is conducted. Having identified and pled a case the objective will be to strengthen it and that may involve laying aside doubts. It may also involve casting a wide net in the search for supportive expertise. Thus, while one might use the expression "multidisciplinary approach" in a forensic context as well as in a clinical context, we would see the meaning of the expression in one context as being different from its meaning in the other. Accordingly, simply because it was generally accepted that there is virtue in adopting a multidisciplinary approach in a clinical context it does not follow that there is exactly the same virtue in adopting a multidisciplinary approach in a forensic

context. All that said, we find little of substance in the criticisms put forward by the defender. The defender refers to *The Ikarian Reefer*, with the consequent implication that either the pursuer or her experts in some way failed in their duties to the court. The point is not however developed. We accept that the evidence on what appears on the MRI scan is only part of a larger picture and we accept that the expert witnesses can only be authoritative within their particular areas of expertise. We also recognise that the witnesses (for both parties) tended to interpret the clinical evidence in the light of their preferred hypotheses but we have found nothing to suggest that those led for the pursuer are any less experts or less "truly independent" than those led for the defender.

[309] Our final general observation is perhaps somewhat hackneyed but it reflects our approach to the expert evidence. We have already referred to what was said at paragraph 48 of *Kennedy* v *Cordia (Services) LLP*. Put short, the Supreme Court there endorsed what had been said by other courts about the need for opinion evidence to be supported by reasoning if it is to be given weight.

[310] While we have not seen it to be reasonable entirely to reject every incidental expression of opinion over 51 days of evidence which was not accompanied by a supporting argument, where it has been material to the parties' respective cases we have looked for reasoning rather than simply for conclusions and in the absence of reasoning we have been less inclined to adopt conclusions. This applies to assessments of probability. As we have already explained, where there is no direct evidence of a past event it is for the judge and not the witness to determine what is likely to have happened. An expert witness may offer his view as to the likelihood of a past event. If he gives reasons for his view then that might assist the judge in making his decision, but an opinion as to likelihood without an explanation of the reasons why it was arrived at, like any other such opinion, is of little or no

help. We stress this because in the parties' respective submissions there was at least a tendency to treat statements of what witnesses thought was probable as the equivalent of direct evidence of fact. A judge's view of what was probable becomes established fact. A witness's view of what was probable can never be more than an expression of opinion.

Assessment of and decision on the pursuer's case in relation to the second causation issue

[311] The pursuer's case is built on a broad framework of what we regard as uncontroversial primary facts and generally accepted medical knowledge. LW has a variety of disabilities. These are: (1) a number of movement disorders which can properly be described as cerebral palsy; (2) severe learning difficulties or cognitive dysfunction; and (3) certain behavioural problems. These have all been apparent from an early age. These disabilities are such as, in any particular case, may be the result of the reduced functionality of the brain by reason of permanent damage to brain tissue consequent upon a period of deprivation of oxygen (hypoxia). LW has suffered damage to brain tissue as demonstrated by MRI scanning, very specifically in relation to the putamina and less specifically in relation to the deep cerebral white matter. He may have suffered other brain damage but interpretation of the MRI scans differed. LW experienced a six-minute period of bradycardia immediately following his delivery by caesarean section. For the purposes of the case that period of bradycardia can be taken as a period of consequential hypoxia (MS 7507). Prior to delivery LW's mother suffered at least one but probably two placental abruptions. From that broad framework the pursuer develops her case that LW's condition was caused by brain injuries sustained as the result of hypoxic ischaemia, secondary to circulatory collapse (an AHII) which began some 20 or so minutes before he was born and which ended when he was about six minutes old, the trigger for the AHII being the

placental abruption, albeit that the abruption may have occurred some time before the circulatory collapse.

[312] While there is what we have described as a broad framework underpinning the case, when one examines the detail of the evidence led in support and in contradiction of the pursuer's case, matters become much more uncertain. We shall consider these uncertainties below but would first mention four general points which bear on the context in which the pursuer's case falls to be considered.

[313] First, we reiterate that the case that the pursuer sets out to prove is that LW suffered a damaging event immediately prior to delivery. That case assumes that up to that point, notwithstanding his history of severely restricted intra-uterine growth probably consequent upon placental insufficiency LW was undamaged in the sense that he had the potential for entirely normal development free from any of the disabilities spoken to in the evidence, and capable of a period of resistance to the effects of an hypoxic event. However, on the evidence that is at best a possibility. There are others which were fully canvassed in the evidence.

[314] The second general point is that it is possible that rather than all flowing from one acute event, LW's disabilities have more than one cause. This is recognised by the pursuer when making her secondary case of material contribution which we consider further below.

[315] The third point relates to statistics as to general medical experience. The pursuer expressly concedes that there are myriad causes of cerebral palsy; an AHII is only one possibility among many others. As has already been touched on, there was some evidence of the general statistical association as between cerebral palsy and AHIIs. We have been inclined to treat it with extreme caution. As is very familiar, we are concerned with a particular case and not the generality of cases. Moreover, this particular case has features

which may take it out of the generality. These features included a history of severe IUGR, delivery very significantly preterm and delivery by caesarean section. Further, even if it is meaningful to apply statistics drawn from population studies with a view to coming to a preliminary view of probability in the individual case, such a preliminary view must shift in response to the leading of evidence which is specific to the individual case. All that said, the pursuer accepts that the hypothesis put forward on her behalf is of the occurrence of a relatively rare event and accordingly in considering the evidence we have seen it as relevant to have regard to the fact that rare events happen rarely.

[316] The fourth point is that the defender makes something (MS 13077) of the coincidence of the commencement of the "dark period" and the incidence of the posited circulatory collapse which is an essential element of the pursuer's case. We are not persuaded that this of itself has any significance. It is uncontroversial that LW was in a state of bradycardia immediately after delivery at 1652 hours on 8 October and it is uncontroversial that he was not in a state of bradycardia when the CTG trace was discontinued at 1639. Therefore it is the pursuer's case that a critical event, the onset of circulatory collapse, occurred within a period of thirteen minutes when LW's heart rate was not being monitored. That means that the pursuer's case is a highly specific one and one that might give rise to the question of what it was in that thirteen-minute period which led to the circulatory collapse. That seems to us to be all that can be said. Coincidences occur.

[317] We turn then to what we see as the uncertainties and anomalies emerging from our review of the evidence.

[318] The pursuer attaches considerable weight to the fact that brain damage consistent with an AHII is demonstrated on the MRI scans carried out when LW was six years of age. That is indeed a fact but MRI scans require to be interpreted and agreement between the

pursuer's two neuroradiologists, Drs Kendall and Connolly, is limited to the identification of damage to the putamina and within the cerebral deep white matter. They differ as to whether the scans demonstrate damage to the thalamus and the pre and postcentral gyri. Dr Connolly tentatively identified damage to the thalamus, again a potentially vulnerable area, but Dr Kendall did not. Dr Kendall identified damage to the pre and postcentral gyri, but Dr Connolly did not. Thus, while it is true to say that the MRI scans are indicative of LW's brain having been damaged, there was only a limited consensus on the precise nature of that damage. While the location and nature of the damage as to which there was consensus indicated hypoxia as the cause, the consensus as to location did not comprehend all of the elements within what Dr Connolly described as the "classic triad" of areas which are particularly susceptible to hypoxic damage. Moreover, the experts were agreed that the scans did not show damage to a number of areas which are recognised as vulnerable in the event of deprivation of oxygen. These were the head of the caudate nucleus, the medial temporal lobes, the superior vermis, the subthalamic nuclei, hippocampus, and the optic radiations. No explanation was offered for this. As Dr Connolly had to accept, this was not a typical presentation, particularly of a case of cerebral palsy in an infant born preterm. That assessment reflects those of both Dr Ferrie and Dr Kirkpatrick, from their perspective as neurologists.

[319] We have understood the neuroradiological evidence to be that the MRI scans show damage to the deep white matter which could be explained by either chronic or acute hypoxia and that that damage could explain LW's cognitive defects. However, when it came to LW's movement disorders matters were more complicated. Dr Connolly accepted that the appearance of the MRI scans was not entirely consistent with LW's physical disabilities. LW's movement disorders include dyskinesia (lack of coordination). That is not

to be expected in the absence of damage to the subthalamic nucleus. Dr Connolly did not identify such damage. There is a further issue in relation to LW's cerebral palsy. On the basis that LW was to be assessed as suffering from dystonic quadresis (motor dysfunction significantly involving four limbs) Dr Connolly expressed the opinion that the features on the MRI scan are in keeping with an acute profound asphyxia. However, the significant involvement of four limbs, whether described as quadresis or quadriplegia, is of importance. The pursuer's case is one of damage caused by an acute event. As was apparent from the evidence of Drs Ferrie and Kirkpatrick, and also the pathway documents, quadriplegia is indeed considered to be consistent with a particular cerebral palsy being the result of an acute hypoxic event. However, diplegia (the significant involvement of only two limbs) is indicative of a more chronic hypoxic event, as postulated by the defender. Whether LW is to be assessed as quadriplegic or diplegic is a question on which Drs Ferrie and Kirkpatrick differ. This court has no material which would allow it to form a view as to whether Dr Ferrie or Dr Kirkpatrick is to be preferred on this question (although we note that Dr Zuberi's assessment of spastic dystonic cerebral palsy mainly affecting LW's lower limbs looks to be close to that of Dr Kirkpatrick and therefore supportive of the defender's case). However, without departing from her reliance on Dr Ferrie's assessment of LW's condition, the pursuer specifically concedes that, taking his disabilities as a whole they are not the most typical presentation for a child whose cerebral palsy has been caused by an AHII (12724). We consider that, on the evidence, this concession could not have been withheld. Thus, whether one concentrates on the neuroradiology or on the neurology, this is not a case which the pursuer's expert witnesses were able to say conformed to the pattern of what they would regard as that of similar cases. We consider this to be of significance. Where a particular instance exhibits features conforming to the pattern of features exhibited by a

paradigm then it may be easy to conclude that the instance is an example of the paradigm. However where it does not then it becomes more difficult to do so.

[320] Quite a lot was said by the experts about the time that it would take for brain damage of the sort demonstrated on the MRI scans to result from an AHII (taking that to involve a complete deprivation of oxygen). This is important because if that time may be assessed, then a view can be taken as to whether that time supports the pursuer's case of an AHII beginning in the thirteen-minute "dark period" and continuing until LW was resuscitated six minutes after delivery or whether the damage could be explained by deprivation of oxygen in the six minutes post-delivery. The generally accepted understanding about the response of a foetus to hypoxia *in utero* is informed by the monkey studies discussed by Myers, although we assume it to be at least consistent with clinical experience. It is to the effect that a previously healthy foetus in the course of vaginal delivery at term will be able to sustain itself for a period of about 10 minutes of hypoxia through a process of anaerobic metabolism before its brain cells begin to die off. That process is discriminating as to the areas of the brain affected and will continue for perhaps 15 minutes before the foetus dies. As can be seen, that sort of timescale would fit with the pursuer's case in that the "dark period" plus the six minutes of post-delivery bradycardia is long enough to accommodate that process. It provides an explanation of why LW did not die, in that the posited period of hypoxia could not have exceeded 19 minutes and may well have been a bit shorter depending upon when in the "dark period" the circulatory collapse, which is part of the pursuer's case, might have occurred. It also would appear to exclude the possibility canvassed by Professor Chiswick that the damage to the putamina evidenced by the MRI scans occurred within the six minutes of post-delivery bradycardia. There is however difficulty in applying the generally accepted understanding of foetal response to

hypoxia to the case of LW. LW was very far from being a previously healthy foetus delivered vaginally at term. He was delivered by caesarean section some seven weeks prior to term. He was very severely growth restricted, his size being equivalent to that of a 26 or 27 week old foetus, rather than his chronological 32 weeks. Significantly, as the pursuer accepted, his IUGR was due to a history of placental insufficiency the consequence of which was inadequate nutrition and oxygenation. Dr Pearse, one of the pursuer's expert witnesses, described LW in the period leading up to delivery as "teetering". That might be regarded as an optimistic assessment. In so far as it is meant to indicate that the foetus was under stress but had not yet succumbed to material damage, it is no more than supposition. It is not supported by any evidence other than the fact that LW was born in a state which allowed him to be resuscitated notwithstanding a six minute bradycardia. What is clear, and as was accepted by witnesses for the pursuer, is that it must be doubtful whether prior to delivery LW had the reserves of glycogen (or anything else which may be relevant) to allow him to be resilient in the event of any further deprivation of oxygen. In the absence of resilience LW was susceptible to damage immediately upon the commencement of any hypoxic episode. A consequence of that is to open up the possibility, supported by Professor Chiswick and not excluded by either Dr Ferrie or Dr Connolly, of the damage to the putamina demonstrated on MRI scan being due to hypoxia in the six-minute period post-delivery and, associated with that, the explanation of LW's bradycardia during that period being the stress of delivery by caesarean section.

[321] An element of the pursuer's case is a circulatory collapse beginning in the "dark period". There is nothing to support the occurrence of such an event other than the post-delivery bradycardia, which, as we have already noted, is open to a different explanation. Against there having been such a collapse is the evidence that LW was cyanosed on delivery

and the interpretation of cyanosis by Dr Coutts as indicative of LW having peripheral circulation at delivery. Having peripheral circulation is not consistent with a state of circulatory collapse.

[322] LW's history of placental insufficiency includes at least one partial placental abruption, and probably two. The significance of an abruption for the pursuer is that it provides the "sentinel event" precipitating a sudden circulatory collapse which is an essential element in her case. The uncertainty in relation to the abruption is when it occurred. This is simply not known but the evidence suggests a time prior to the pursuer's admission to hospital and therefore more than four hours prior to when the pursuer submits that LW suffered a circulatory collapse. While we have not found any detailed discussion of the mechanisms that might be involved, we suppose it may be possible that a sufficiently severe partial abruption with a consequent reduction in the effectiveness of the placenta in supplying the foetal circulation with oxygen might lead to foetal ischaemia and, as a result, heart failure. We further suppose it possible that the two events might be separated in time. However, as it seems to us in the absence of specific evidence, the longer the period between the event (the abruption) and its alleged consequence (the collapse) the more difficult it would be to infer a relationship of cause and effect as between one and the other, particularly where, as is contemplated by Professor Walker, a foetus may survive a partial abruption. Putting the matter shortly, while there is no doubt that there was a partial placental abruption it is a doubtful candidate for a sentinel event.

[323] We have considered the evidence led under reference to the pathway documents. We repeat that, whatever may have been the intention of their framers, we do not consider that either of the documents should be regarded as a check list of criteria which have to be met before the pursuer can succeed. That would be so even if we were concerned with an

infant delivered at term, which we are not. However, what we have taken from this chapter of the evidence is that while it does not exclude the possibility of a perinatal acute hypoxic insult it does little or nothing to support such a possibility.

[324] A further difficulty in accepting the pursuer's case that it was only in the thirteen or so minutes before and the six minutes after delivery that LW sustained the injury which caused his disabilities, lies in the essentially uncontroversial primary evidence as to what may be taken to have happened in the weeks prior to that short perinatal period. As we have observed, LW was very severely growth restricted. That meant he had been unable to develop normally *in utero* over the final five or six weeks of his 32 weeks of gestation, and according to Professor Walker probably longer. A mechanism for that was identified. The placenta was not functioning as it should with the consequence that the foetus was not being supplied with sufficient oxygen and essential nutrients. One measure of that was the Doppler readings carried out on 8 October, but the extent of IUGR demonstrated that the pathology was of long duration. The biophysical profile recorded by Dr Crichton and the subsequent CTG trace indicate that LW's antenatal condition was very poor. These measurements relate to the four hours preceding delivery but it is the pursuer's case that she had been experiencing reduced foetal movements from the time of her review on 19 September. While it may be, as the pursuer submits, that IUGR and prematurity do not necessarily mean that an infant so affected cannot develop into a healthy adult, the fact remains that for a lengthy period *in utero* LW was receiving inadequate nutrition and (at best) a suboptimal supply of oxygen. On the basis of Dr Ferrie's evidence, the pursuer argues that the absence of evidence of PVL on the MRI scans and a distribution of LW's cerebral palsy interpreted as quadriplegic point away from brain damage having been caused by chronic hypoxia. However, Dr Connolly's evidence undercuts the significance of

PVL not being identified given that LW was at 32 weeks gestation and Dr Kirkpatrick disputes Dr Ferrie's assessment of the nature of LW's cerebral palsy. On any view, it would seem to be indisputable that *in utero* LW's supply of oxygen was restricted and that over a long period, in other words he was exposed to chronic hypoxia. While the extent of the restriction of the oxygen supply to the foetus is unknown it must qualify as a possible explanation for LW's brain being damaged or not properly developed. That is the explanation favoured by Professors Murphy and Walker.

[325] Where, as here, a pursuer has to prove a particular causal relationship, all she need do is to meet the standard of marginal probability. However if she fails to do that her claim falls to be dismissed. It is not necessary for that to happen that the court has been able to make the further step and find that an alternative explanation is more likely: *The Popi M.* Nevertheless, the fact that alternatives can be put forward which are supported by evidence as possible explanations for the relevant outcome, as is the case here, is of obvious relevance to the question as to whether the court can be satisfied that the pursuer's explanation meets the standard of marginal probability (cf *La Compania Martiartu* v *Royal Exchange Assurance Corporation* [1923] 1 KB 650, Scrutton LJ at 657). Having given the pursuer's case as careful consideration as we have been able to give it, we consider that she has not met her *probandum*. There are simply too many uncertainties and inconsistencies within her case and too many doubts raised by the clear evidence of placental insufficiency, consequent IUGR and likely postnatal hypoxia.

The pursuer's material contribution case

[326] The pursuer's primary case on causation was that the negligence of the midwives caused all of LW's disabilities. As a secondary case, the pursuer submitted that even if the negligence of the midwives was not the sole cause of LW's disabilities, it made a material

contribution to them. The submissions to that effect (MS 12782) focus, in factual terms, on the occurrence of an acute hypoxic event, and interpretation of the radiological evidence which establishes that at least some of LW's disabilities may be attributed to such an event. There is a separate argument based on an assumption that it has been established that delivery was delayed by about a day as a result of the negligence. The material contribution case was advanced under reference to *Bailey* v *The Ministry of Defence* [2009] 1 WLR 1052 and *Popple* v *Birmingham Women's NHS Foundation Trust* [2013] Med LR 47. It was argued that the defender had failed to show that LW would have suffered his disabilities if the negligence had not occurred, and that accordingly, the pursuer was entitled as a matter of law to recover damages for all of the disabilities. The primary argument was that even if all the disabilities could not be attributed to an AHII, some of them could, and the evidence being unable to establish between those which might be so attributed, and those with a non-negligent cause, the pursuer should recover damages for all of them.

[327] The defender's submissions (MS13459 to 13464) were that material contribution had no application to the present case. *Bailey* and *Popple* could be distinguished on their facts, as cases where there was a known single cause for a single harm and the issue for the court was the extent to which negligence contributed to that cause and that harm. That was very different from the present case where the difficulty for the pursuer was in showing which, if any, of many possible causes made any contribution at all to each of LW's many and distinct disabilities (*c.f., Wilsher* v *Essex Area Health Authority* [1988] AC 1074).

Discussion

[328] The essential proposition of the pursuer's argument is that, even if the negligence of the midwives was not the sole cause of LW's disabilities, it made a material non-negligible contribution to them (POHS, Ch. 11, MS12779). An immediate difficulty for the pursuer is

that, like her primary case, her material contribution case is in the first place predicated on her establishing that damage (in this case, material, non-negligible damage) to LW occurred in an AHII in the period beginning in the "dark period" and ending at six minutes following LW's delivery. A subsidiary argument, based on the court's being able to find that there was a neonatal event which contributed to the disabilities, is that the negligence led to delay in delivery and thus contributed to the disabilities. This would require consideration of the fact that if any event were precipitated by the caesarean section it would have followed in any event, and of the issues of resilience and the extent to which the pursuer could point to evidence that this would have deteriorated in the hours prior to delivery. However, standing our conclusions at paragraph [297] onwards in respect of the pursuer's primary causation case, and our findings at paragraph [134] in respect of whether delivery was delayed as a result of the negligence, there would be a short answer to these arguments.

[329] In any event, we do not consider that the cases of *Bailey* and *Popple* have any application to the circumstances of the present case. Under reference to these cases, the pursuer is seeking to depart from an application of the conventional "but for" test. Her argument appears to be simply that if the negligence of the midwives made more than a negligible contribution to the damage, on the authority of *Bailey*, she is entitled to recover for all of LW's disabilities. In our view that is not the import of the decision in *Bailey*.

[330] As the defender pointed out, the difficulty for the pursuer lies in showing which, if any, of the many possible causes made any contribution at all to each of the many distinct disabilities. It is akin to the situation in *Wilsher*, which, as described in *Bailey*, was a case "where there were different distinct causes which operated in a different way and might have caused the injury and where the claimant could not establish which cause either "caused or contributed" to his injury" (*Bailey*, paragraph 44). The extent to which *Bailey*

provided for a departure from the "but for" test, related to the uncertainties of medical science. It was a policy-based decision, which stated that in a case where medical science could not establish the probability that "but for" an act of negligence the injury would not have happened, but could establish that the contribution of the negligent cause was more than negligible, the "but for" test was to be modified, and the claimant would succeed. In the present case, there was no argument that the pursuer was unable to prove the mechanism of damage contended for by her because of the inadequacies of medical science. In a manner similar to cases such as *Chappel* v *Hart*, as discussed at paragraphs [63] and [64] above, the *Bailey* line of authority is a restricted, policy-based exception to "but for" causation in cases where inadequacies in medical science result in the pursuer being unable to prove the full extent of the causative mechanism. The issue in this case is whether or not, on the balance of probabilities, the evidence establishes that the damage for which the pursuer sues was caused in an AHII commencing in the "dark period" and ending at six minutes following LW's birth. It clearly falls within the "but for" test, and no issue of the kind described in *Bailey* arises.

[331] For all the reasons which we have given, both the reclaiming motion and the cross-appeal fall to be dismissed.

APPENDIX OF EXPERT WITNESSES

Pursuer's Experts

1. Jean McConville, Midwife, PGDips, BSc(Hons), DPSN, RM, RGN.

2. Sandra Tranter, Midwife, MSc, HV, RM, RGN.

3. Professor Timothy Draycott, Consultant Obstetrician, BSc, MBBS, MRCOG, MD.

4. Professor Michael de Swiet, Emeritus Professor of Obstetric Medicine at Imperial College School of Medicine, London, MD, FRCP, FRCOG; retired consultant physician of obstetrics.

5. Dr Brian Kendall, Consultant Neuro-radiologist at the London Imaging Centre and HCA Wellington Hospital and Emeritus Consultant Neuro-radiologist at the Hospital for Sick Children and the National Hospital for Neurology and Neurosurgery, London, FRCR, FRCP, FRCS.

6. Dr Colin Ferrie, Consultant Paediatric Neurologist, Leeds General Infirmary, BSc(Hons), MD, MRCP, FRCPCH, MEWI.

7. Dr R G Pearse, Consultant Neonatal Paediatrician at North Trent Regional Neonatal Intensive Care Unit at the Jessop Wing in Sheffield Teaching Hospitals NHS Foundation Trust, MA, MB, BChir (Cantab.), FRCP, FRCPCH.

8. Dr Daniel J A Connolly, Consultant Paediatric Neuro-radiologist, Sheffield Children's Hospital and Consultant Neuro-radiologist, Royal Hallamshire Hospital, Sheffield Teaching Hospitals Trust, BSc(Hons), MBChB, MRCP, FRCR.

9. Dr Allan G Howatson, retired Consultant Paediatric and Perinatal Pathologist and Head of the Department of Paediatric Pathology at the Royal Hospital for Sick Children, Yorkhill, Glasgow, Batchelor of Science (with Honours in Pathology), Batchelor of Medicine and Surgery, Fellow of the Royal College of Surgeons in Edinburgh, Fellow of the Royal College of Pathologists, Diploma in Forensic Medicine.

Defender's Experts

10. Dr Julia Sanders, Consultant Midwife, Cardiff and Vale University Health Board, Lead Clinician for Midwifery Led Unit and Clinical Research Fellow, Cardiff University. Registered General Nurse, Registered Midwife, Advanced Diploma in Midwifery, Master of Laws (Legal Aspects of Medical Practice), Master of Public Health, Doctor of Philosophy.

11. Professor Deirdre Murphy, Professor of Obstetrics, Trinity College, University of Dublin, Consultant Obstetrician with lead responsibility for the labour ward at Coombe Women and Infants University Hospital, MB, BCh, BAO, BA, Diploma in Epidemiology, MRCOG, MD, CCST, FRCOG, MSc in Healthcare Leadership.

12. Professor Malcolm L Chiswick, retired consultant paediatrician, Honorary Professor of Neonatal Paediatrics at the University of Manchester, and Honorary Consultant Neonatal Paediatrician with Central Manchester and Manchester University Hospitals NHS Foundation Trust. Lead Governor of Central Manchester and Manchester University Hospitals NHS Foundation Trust, MD, FRCP[Lond], FRCPCH, FRCOG, DCH.

13. Dr Martin Kirkpatrick, Consultant Neurologist and Honorary Senior Lecturer, Tayside Children's Hospital, Ninewells Hospital and Medical School, Dundee, MBBS, FRCPCH, FRCPE, DCH.

14. Professor James Walker, Professor of Obstetrics and Gynaecology, St James University Hospital, Leeds, and Honorary Consultant in Obstetrics and Gynaecology, United Leeds Teaching Hospital Trust, MB ChB, MRCP, MRCOG, FRCPS, MD, FRCOG, FRCP.

15. Dr Maeve McPhillips, Consultant Paediatric Radiologist, Royal Hospital for Sick Children, Edinburgh, MB BCh BAO (NUI), FRCR, Fellow, Diagnostic Imaging (University Hospital, Zurich), Fellow, Diagnostic Imaging (Chinese University, Hong Kong), Fellow, Paediatric Radiology (Montreal Children's Hospital).