

IN THE HIGH COURT OF SOUTH AFRICA
GAUTENG DIVISION, JOHANNESBURG



CASE NO: 36681/2017

- (1) Reportable: No
(2) Of interest to other Judges: No
(3) Revised: No

Hansen
Date: -11/05/2023

In the matter between:

TM obo DM

Plaintiff

and

THE MEC FOR HEALTH, GAUTENG PROVINCE

Defendant

Neutral Citation: TM obo DM v THE MEC FOR HEALTH, GAUTENG PROVINCE (Case No: 3681/2017) [2023] ZAG45 ZAGPHJHC (11 May 2023)

J U D G M E N T

MAIER-FRAWLEY J:

Introductory background

1. The plaintiff, acting in her personal and representative capacity on behalf of her minor child (DM) (hereinafter referred to as 'baby D' or 'the baby'), instituted a claim for delictual damages against the defendant as the employer of the nursing staff at the Hillbrow Community Health Centre (hereinafter referred to as the 'Hillbrow clinic'). The plaintiff's claim against the defendant was brought on the basis that the MEC was vicariously liable for the negligent conduct or omission of the employees of the defendant who attended to the plaintiff (and her then unborn child) at the Hillbrow clinic after her admission and/or who attended to baby D during the Plaintiff's labour process and the delivery of baby D on 9 July 2009. She claims that this negligence caused and culminated in baby D developing cerebral palsy as a consequence of a hypoxic-ischaemic event that resulted in irreversible brain damage being sustained by baby D.
2. The element of wrongfulness in the assessment of delictual liability was not in issue in the action. The other elements of delictual liability, namely, negligence and causation remained in dispute.
3. In her particulars of claim, the plaintiff pleaded that the defendant owed her and her and her unborn child a legal duty of care, amongst others, to monitor the plaintiff's labour with such skill, care and diligence as could reasonably be expected of medical practitioners and/or nursing staff with appropriate obstetric knowledge in similar circumstances, which duty was breached by employees of the defendant who were alleged to have been negligent in several respects, which for purposes of the judgment, in the main, included, amongst others, that they:

- 3.1. Failed to properly monitor the progress of the plaintiff's labour and the foetal well-being with sufficient regularity during the active phase of labour (i.e when the plaintiff's cervix had dilated 4cm or more);
 - 3.2. Failed to either appropriately, timeously and/or with sufficient frequency, monitor the plaintiff's labour;
 - 3.3. Failed to monitor the foetal heart rate appropriately, timeously or with sufficient frequency and/or at all and/or failed to detect that baby D was in foetal distress;
 - 3.4. Failed to appreciate and/or properly and/or adequately react to foetal distress;
 - 3.5. Failed to timeously or adequately institute appropriate or effective measures to prevent further distress in the foetus;
 - 3.6. Failed to maintain a proper and accurate partogram;
 - 3.7. Failed to summon, timeously or at all, for purposes assessment, advice and/or appropriate action, the assistance of a medical practitioner in circumstances where it was necessary and/or indicated to do so.
4. The defendant opposed the action and filed a plea, *inter alia*:
 - 4.1. denying that the employees of the defendant were negligent in any of the respects alleged or at all; and
 - 4.2. denying that there was any causal connection between the negligence (if any) of the staff at Hillbrow clinic and the cerebral palsy sustained by baby D.
5. No contributory negligence on the part of the plaintiff was averred in the plea.
6. The plaintiff called the following witnesses to testify at the trial:
 - 6.1. Mz 'TM' (plaintiff);
 - 6.2. Dr Alheit (Radiologist)
 - 6.3. Professor Anthony (Obstetrician & Gynaecologist);

- 6.4. Dr Kara (Paediatrician) and
- 6.5. Dr Pierce (Paediatric Neurologist)

7. The defendant called the following witnesses to testify at the trial:

- 7.1. Dr Mogashoa (Paediatric Neurologist)
- 7.2. Professor Bolton (Paediatrician)
- 7.3. Sr Moqhae (Midwife who delivered baby D);
- 7.4. Sr Mabanga (Midwife who assisted with resuscitation of baby D)
- 7.5. Dr Manthata-Cruywagen (Obstetrician and Gynaecologist);and
- 7.6. Dr Weinstein (Radiologist)

8. The expert reports filed of record by both parties were admitted into evidence at the trial as well as the available hospital and medical records on which all experts relied to formulate their respective opinions. The expertise and experience of all experts and midwives who testified at the trial was not in dispute.

Background Matrix

9. The background factual matrix is the following: The plaintiff received ongoing antenatal care and management of her pregnancy at the Jeppe Street Clinic. Baby D was the plaintiff's first pregnancy and is her firstborn child. The pregnancy was uneventful and progressed normally. The plaintiff experienced no illnesses and remained healthy throughout her pregnancy. She attended antenatal check-ups every month and was informed of the anticipated due date of baby D's birth. About 20 days before her due date, she was advised to attend at a hospital if she felt any pain. She initially attended at the Hillbrow clinic on 8 July 2012 after she noticed the presence of a discharge and blood, whilst experiencing abdominal pain. She was assessed as not being in labour and told that she should return home. Later that evening she returned to Hillbrow clinic as she perceived that liquid had been expelled from her vagina

whilst continuing to experience abdominal pain, including back pain. She was again assessed and admitted to what was ostensibly the labour ward at the clinic.

10. According to the medical records, between midnight and 4 am on 9 July 2012, the plaintiff's condition was monitored in the labour ward every hour at which times the foetal heart rate (FHR) was plotted on a partogram and recorded to be between 120 and 135 beats per minute (bpm), i.e., at 00h00; 01h00; 02h00; 03h00 and at 4h00. After 4h00, there were no further entries on the partogram.
11. At 0h00, the plaintiff's cervix was 4cm dilated and she had thus entered the first stage of the active phase of labour. A FHR of 120 bpm was recorded at this time. At 04h00, the plaintiff's cervix was 8cm dilated, at which time the medical records reflect that her membranes had ruptured and that she was draining clear liquor. A FHR of 133 bpm was recorded at this time. At 06h00 the plaintiff's cervix was 9cm dilated and a FHR of 120 bpm was recorded at this time. At 7h00 the plaintiff's cervix was fully dilated at 10 cm and the plaintiff had thus entered the second stage of active labour, however, by then, she was draining very thick (grade 3) meconium stained liquor and an abnormal foetal heart rate of 109 bpm was recorded. The plaintiff was thereupon taken to a section that was used as a delivery room where she gave birth to baby D by way of an unassisted natural vaginal delivery at 08h00 on 9 July 2012. Delivery of the placenta occurred at 8h15 on that day. After the delivery, the plaintiff was given 10 units of Syntocinon in a 100mls of Ringers Lactate intravenous infusion.
12. When baby D was delivered, he did not move or cry. He was put onto the plaintiff's chest and meconium was wiped manually from his mouth by the attending midwife. After the umbilical cord was cut, he was taken for

resuscitation and suctioning and given oxygen. Baby D's apgar scores at birth were 6/10 at 1 minute; 6/10 at 5 minutes and 6/10 at 10 minutes. Baby D was transferred to Charlotte Maxeke hospital for further care where he was admitted to the paediatric ICU.

Issues in contention at trial

13. The main controversy at the trial was in relation to the cause of the brain injury sustained by baby D. Whilst the experts (neuro-paediatricians and gynaecologist/Obstetricians) were agreed that the insult that led to the eventual brain injury occurred intrapartem (before delivery) and not after the birth of baby D, the dispute between the parties was whether it happened suddenly (without warning) and was so severe as to result in a total shut down of oxygen very quickly, i.e., in the space of between 5 to 15 minutes during the last 30 minutes of labour, so that no amount of monitoring of the foetal condition could have prevented it, or stated differently, so that inadequate monitoring of the foetal condition in fact made no difference, or whether it happened as a result of intermittent episodes of hypoxia that went undetected because of a lack of proper foetal monitoring and which was preventable. The further dispute centred around whether the negligence on the part of the attending staff caused baby D to suffer the consequences of such event.
14. Linked to the main controversy, was the question of whether the BGT¹ brain injury which baby D sustained can be caused by anything other than an acute profound total or near-total hypoxic ischaemic insult or whether it can be caused by intermittent or prolonged episodes of subacute/subthreshold hypoxic insults (interruption of the supply of blood to the brain).

¹ A BGT (or which is sometimes referred to as a PBGT injury in literature) is an injury to the basal-ganglia-thalamus deep nuclear structures including the periolandic area of the brain, being the metabolically active parts of the brain.

15. A further controversy which featured during the trial was whether or not the plaintiff had chewed a leafy substance called *khat* (also known as *Mira*) during her pregnancy, such that it had an adverse effect on her placenta and if so, whether an inadequate blood/oxygen supply to baby D caused by placental insufficiency/abnormality is what caused him to suffer the brain injury in question.

Agreements per experts' joint minutes

16. The agreements reached in the joint minutes of the various experts are extensive and are a matter of record. I therefore intend to only highlight the most salient of these for purposes of judgment.
17. The paediatricians agreed in their *addendum* joint minute that foetal bradycardia and late onset of grade 3 meconium liquor was detected an hour before birth. Baby D was transferred from Hillbrow clinic to Charlotte Maxeke hospital with an initial diagnosis of meconium aspiration. Baby D's birth weight, length and head size were in keeping with a baby born at term and there was no growth restriction or asymmetry. Baby D required resuscitation after birth.
18. The Gynaecologists/Obstetricians agreed in their joint minute of 22 November 2020, amongst others, that:
- (i) The abnormal fetal heart rate observed and the development of thick meconium stained liquor (at 07h00, an hour before birth) failed to elicit any response directed at more accurate assessment of fetal wellbeing from the nursing staff, which amounted to substandard care;
 - (ii) The commencement of the second stage of [active] labour lasted another hour beyond the 07h00 assessment and this may have added additional risk of injury to the *fetus that was hypoxic before the onset of the second stage of labour*; (emphasis added)

- (iii) The partogram was blank after 04h00 and during the remaining four hours of labour only two assessments were made and only two assessments of the fetal condition were recorded, which was substandard care. Partograms are required in the management of all parturients and failure to use a partogram correctly increases the likelihood of perinatal death and injury due to asphyxia;
- (iv) Baby D showed no signs of growth restriction at delivery and was described as having suffered from acute profound hypoxia in the perinatal period. The development of acute profound hypoxia may be attributable to a sentinel event in the perinatal period such as placental separation, cord prolapse or uterine rupture *but it may also develop during the course of what may otherwise be deemed to be normal labour*; (emphasis added)
- (v) The risks of fetal monitoring are well recognized and fetal monitoring in labour is universally recommended in order to detect the onset of fetal compromise even in a completely normal pregnancy. Protocols of fetal monitoring are universally endorsed and typically dictate that the fetal condition needs assessment every two hours in latent labour, every half hour in active labour and after every second contraction in the second stage of [active] labour. The changes in fetal heart rate precede injury and are markers of insult (which if unchecked) lead to injury. The purpose of fetal monitoring is the detection of patterns associated with hypoxia and acidosis which mark the occurrence of a compensated insult manifesting *before injury is sustained*; (emphasis added);
- (vi) The second stage of labour is the most dangerous time for the development of hypoxia and for that reason the monitoring of the fetal condition is by prescription at its most intense;

- (vii) The susceptibility of the foetus to injury during the second stage of labour varies depending on the extent to which compensatory mechanisms have already been deployed in the earlier part of labour and a baby that enters the second stage of labour with compensated acidosis may be more susceptible to injury in the second stage of labour than a child who has sustained normal metabolic activity on the basis of adequate oxygenation up to the start of the second stage of labour;
- (viii) A normal baby can sustain 10 to 15 minutes of complete hypoxia before damage ensues; however the susceptibility to injury and the time taken to cause injury will depend on the baseline oxygenation of the foetus. Where a gradual decline in oxygenation has occurred during labour and compensatory mechanisms have already been deployed, the capacity to withstand further hypoxic insult also declines creating the situation where seemingly unremarkable additional stresses may be the proverbial straw that breaks the camel's back. Thus, a baby who has suffered a progressive decline in oxygenation throughout the first stage of labour may not be able to sustain the same difficult second stage of labour without developing neurological injury;
- (ix) Avoidable injury is to be recognized where typical evidence exists of abnormal fetal heart rate patterns in the first or second stage of labour which provide evidence of the activation of reflex mechanisms characteristic of evolving fetal hypoxia and acidosis. Failure to recognize these patterns and failure to intervene early enough (while compensatory mechanisms can still sustain adequate perfusion of the brain) will have increased the susceptibility of the foetus to neurological injury the longer the labour progresses. The addition of *further insult* (such as the use of oxytocic drugs, the onset of a long second stage of labour *with the mother bearing down* and the use of fundal pressure during the second stage of labour) may provide the

straw that breaks the camel's back and may lead to limited or profound injury of the foetal brain;(emphasis added)

- (x) Injury is avoidable by early detection of evolving hypoxia;
- (xi) The object of foetal monitoring is to diagnose evolving foetal hypoxia *before* insult leads to injury;
- (xii) Where protocols of foetal heart rate monitoring have been breached and injury ensues there must be consideration of liability with the only exception to liability being the rare occurrence of a devastating and unpredictable sentinel event such as sudden antepartum haemorrhage due to placental separation before or during labour or the unprovoked development of a ruptured uterus – acute severe hypoxic ischaemic injury to the fetal brain may be avoidable and where abnormal fetal heart rate monitoring precedes the injury in the absence of a sentinel event, such assumptions are reasonable and sustainable;
- (xiii) Foetal monitoring in this case was inadequately performed during labour in conflict with the published national maternity guidelines;
- (xiv) Inadequate foetal monitoring precluded the identification of any abnormal fetal heart rate pattern;
- (xv) There is evidence that certain markers of potential foetal hypoxia emerged during the course of labour: (i) the development of thick meconium stained liquor; (ii) a slow foetal heart rate observed one hour before delivery and (iii) further foetal heart rate assessment did not take place before delivery;
- (xvi) In this case the markers of fetal hypoxia did not elicit an appropriate response in terms of immediate electronic fetal monitoring, intrauterine resuscitation or any attempt to expedite delivery;
- (xvii) Management [in labour] took place in breach of published guidelines and what evidence there is [in the clinical records]: (i) indicates the possibility of fetal hypoxia which either went unrecognized or may have

been ignored by the attending staff and (ii) supports the probability of an intrapartum cause for early onset neonatal encephalopathy;

(xviii) Consequently, neurological injury held to be associated with intrapartum hypoxia developed because the care provided was inadequate.

19. An amended joint minute by the Gynaecologists/Obstetricians was subsequently filed due to the fact that Dr Manthata-Cruywagen changed her opinion after receiving additional medical notes which queried the use of Mira/Khat by the plaintiff. Amendments to the initial joint minute were effected on 22 January 2022. In terms of the amended joint minute, Dr Manthata-Cruywagen raised the use of Khat/Mira by the plaintiff during her pregnancy as a causal factor that led to a total shutdown of oxygenated blood supply to the fetus 30 minutes before delivery. In her view, the use of Khat exposed the plaintiff's placenta to an 'amphetamine like substance' which has been postulated to have a deleterious effect on the placental function causing vasoconstriction and reducing fetal oxygenation. Put differently, the compromised placenta exacerbated the reduction in oxygen blood supply. Dr Manthata-Cruywagen recorded in the amended joint minute that '*A substance-exposed placenta will not be able to supply oxygenated blood to the fetus during labour adequately because during labour, contractions further exacerbate reduction of oxygenated blood supply. Ms [M...] acknowledged during the interview with the neuro development doctor Dr Blumberg in 2013 to using Mira/Khat during pregnancy.*' She therefore concluded that in the second stage of labour '*where a sub-optimally functioning placenta that has been exposed to an amphetamine like substance, further exacerbated the reduction in oxygen blood supply. This was the straw that broke the camel's back and led to a total shutdown of oxygenated blood supply to the fetus just a few minutes before delivery.*' Her earlier agreement recorded in sub-

paragraphs 17 (ix); (x) and (xii) above, was retracted with Dr Manthata-Cruywagen recording that '*Acute severe hypoxic ischemic injury to the fetal brain, implies either rapid total shutdown oxygenated blood (sic) (in which compensatory mechanisms have no time to become deployed)...*'.

20. Save for the issue of Khat and its postulated deleterious effect upon the mother's placenta, coupled with the suggested unavailability or non-preventability of the postulated inevitable consequences of an abnormally functioning placenta, the agreements recorded in the initial joint minute of the Gynaecologists/Obstetricians remained unchanged.
21. In their joint minute dated 10 May 2019, the radiologists initially agreed that the MRI pattern observed was in keeping with *acute profound hypoxic ischaemic* brain injury. The findings of the MRI study suggested that genetic disorders as a cause of baby D's brain damage were unlikely. There was no evidence of intracranial infection on the reviewed MRI. They further agreed that a review of the clinical and obstetrical records by pediatric, neurology and obstetrics experts was essential in determining the cause and probable timing of the hypoxic ischemic injury.
22. In a subsequent addendum joint minute dated 19 January 2022, Dr Alheit recorded that the term 'acute profound' hypoxic ischaemic injury of the brain '*is inappropriate as there is no history of an intrapartum sentinel event in this case. This statement is based on the article by Smith et al, which was published at the end of 2020 and the 2019 revision of the ACOG guidelines as well as the recently published article: Neuroimaging in the term newborn with neonatal encephalopathy...the terminology used for identifying the injury in this case should only describe the structural changes *PGBT/Central² hypoxic ischaemic*

² 'PGBT' refers to the Periolandic, Basal Ganglia and Thalamus structures.

injury of the brain.' Dr Weinstein remained resolute that the pattern observed was 'in keeping with a hypoxic ischemic event/s of an acute profound distribution (type).'

23. The Paediatric Neurologists agreed in their joint minute that baby D's condition was most likely the result of intrapartum hypoxia. As regards his condition, they agreed that he suffers from a mixed cerebral palsy (predominantly dystonic/dyskinetic). He is capable only of limited independent mobility and his co-morbidities include profound intellectual disability, microcephaly, strabismus, contractures, dislocated left hip and severe developmental delay.
24. Although they did not testify at the trial, the Paediatric/Genetic joint minute compiled by Dr Gerike and Dr Bhengu records their agreed observations of baby D's condition: he has no self-initiated movement; he has to be fed; wears nappies; he can see and hear and understand the most basic forms of communication but there is no expressive speech. Clinically, his lack of autonomous mobility makes for a diagnosis of GMFCS level v cerebral palsy, which indicates the most severe level of motor muscle disability.

Pre-trial admissions

25. Pursuant to a formal request for admissions and trial enquiries,³ the parties agreed that:
 - (i) The plaintiff did not suffer from complications during her pregnancy and her pregnancy progressed normally;
 - (ii) The Hillbrow Community Health Centre is fully equipped with foetal monitoring equipment and has a staff compliment that is fully trained in obstetric care and obstetric emergency management;

³ The request for admissions and trial enquiries are contained in a document at A11 to A14 on Caselines.

- (iii) A hypoxic ischaemic event in a term child can result from final circulatory collapse in a neonate exposed to subthreshold hypoxia over a period of time, however, the defendant stated that the facts of this case differ in that the hypoxic ischaemic event was caused by an acute profound injury;
- (iv) The late first and the second stages of labour require careful and continuous monitoring;
- (v) A prolonged labour may result in a lack of oxygen which can cause hypoxia;
- (vi) The purpose of foetal monitoring in labour is to detect hypoxia and to prevent asphyxia;
- (vii) Intrapartum hypoxia (foetal distress) can precipitate hypoxic ischaemic encephalopathy (HIE) in the neonate thereby leading to neonatal encephalopathy and cerebral palsy;
- (viii) Birth asphyxia may cause permanent neurological damage, expressed as cerebral palsy or mental deficiency or both; and
- (ix) Besides death, the most severe expression of birth asphyxia is the syndrome of Hypoxic Ischemic Encephalopathy (HIE).

Pre-trial enquiry

26. In terms of the plaintiff's list of pre-trial enquiries, amongst others, the following question was asked and answered:

"Does the defendant intend relying on any intrapartum (during labour and birth) cause for [baby D's] cerebral palsy? If so, the plaintiff requires the defendant, to indicate what intrapartum cause will be relied upon which caused [baby D's] cerebral palsy?

Answer: Yes. The hypoxic ischemic event was caused by an acute profound injury."

27. At the outset of the trial, the plaintiff placed on record that the defendant did not dispute that sub-standard care was rendered to the plaintiff (and by implication, also the unborn foetus) at the Hillbrow clinic during the labour process.

Evidence at trial

Plaintiff's witnesses

Plaintiff (Mother)

28. The plaintiff testified through the aid of an interpreter. She testified about her pregnancy having progressed normally, without complications, including the events leading up to her admission at Hillbrow clinic on 8 July 2012.⁴ She confirmed being 31 years old when she fell pregnant and that baby D was her first child. She hails from Ethiopia and is unable to speak or understand any African languages. She understands a little English.
29. According to the plaintiff, the nurse who checked on her at midnight and at 4am checked her blood pressure but did not use any instrument to check on baby D. When she screamed out for help because of the pain she was experiencing, the nurses did not heed her cries. She was aware of a changeover of staff after 6 am. One of the new nurses from the day shift, took her to a room that had a curtain where the nurse performed a vaginal examination. Then the nurse came with medical tools such as a needle, scissors and other instruments. The nurse told her to push for the baby to come out. She only started pushing once the nurse told her to push. The plaintiff said she was experiencing constant pain. At some point the nurse gave her an injection in her right thigh after which she felt intense pain and she screamed. The nurse swore at her and told her to keep quiet and to keep pushing until the baby came out. She could not remember for how long she

⁴ A summary of the salient events are recorded in paragraph 9 of the judgment.

was pushing before the baby came out. When all this was happening, no-one else came into the room to help the nurse who had delivered baby D.

30. After his delivery, the nurse put baby D on the plaintiff's chest. According to the plaintiff, baby D had a 'yellowish' blue colour. He did not cry or move and the nurse told her that the baby was dying. She could not recall how long the baby lay on her chest before he was taken away. She recalled falling asleep thereafter until she was woken up by another nurse and told to count her fingers. As the plaintiff was counting, she fell asleep again. This prompted the nurse to call for help. Other nurses then came into the room. One of the nurses put a drip into each of her hands and after a while they put an extra drip into her one hand. She recalled bleeding a lot after the birth of baby D and that she was feeling weak. She was transferred to Charlotte Maxeke hospital, where she was admitted, however, in the evening she was told that she could go home.

31. The following day she saw baby D at the hospital. He was lying inside an incubator and he was shaking or shivering and had medical tools attached to his body and head. The following day she was told to feed baby D but he was unable to suckle from her breast. The nurses told her to extract milk from her breasts and to bring it to the hospital inside a cup which they provided for this purpose. Baby D remained in hospital for 7 days. On the 7th day of admission, a doctor explained to her and her husband that baby D had suffered a shortage of oxygen and brain damage and that he would have a problem in the future in that he would not be able to speak or walk.

32. About 18 to 20 days after baby D's discharge from hospital, he was crying so much that the plaintiff took him to the clinic for a check-up. The baby was examined and the plaintiff was told to take him to the hospital. She attended at the emergency department at Charlotte Maxeke where a doctor examined

baby D and sent him for X-rays. She was later informed by a specialist doctor that the baby had an infection and a blood clot in his shoulder. He underwent an operation to remove the blood clot and remained hospitalised for about two months.

33. When baby D was about 6 months old, he developed a fever. She took him to hospital and it was then, when the examining doctor tried to sit him down, that she saw something was wrong with baby D, as he could not balance his head, which was 'wobbly'. She was referred to a 'brain doctor' who examined baby D and advised that the baby had suffered a lack of oxygen before birth.
34. The plaintiff was asked whether she used a substance known as khat (which is also known as 'Mira'). She admitted chewing it, however, not on a regular basis. In the Ethiopian culture, people chew khat at social gatherings as a cultural pleasure. She stated that she had last chewed it about one or two months before she fell pregnant and did not use Khat or any similar substance at all during her pregnancy.
35. During cross-examination she stated that she had not mentioned to any health worker that she had used khat during her pregnancy, nor had she informed any healthcare professional at the 'Johannesburg' hospital of her usage of khat. Prior to her pregnancy, she only chewed it once in a while, not regularly. When asked why she did not use it during her pregnancy, the plaintiff stated that it was because she did not feel like it.
36. During cross-examination the plaintiff confirmed that she was taken to the room where baby D was delivered by the new nurse, where she was told to lie down. Either the nurse or she (plaintiff) opened her legs. She was lying on her back and the nurse told her to push the baby out. The nurse was standing in front of her legs and she was pushing on the nurse's instruction.

37. During re-examination the plaintiff stated that from the time the nurse did the vaginal instruction until the birth of the baby, the nurse did not listen to baby D's heartbeat.

Dr Alheit (Radiologist)

38. Dr Alheit explained that the MRI scan performed on baby D's brain when he was 5 years and 6 months old reflected damage to the central part of the brain involving the basal ganglia thalamus structures of the brain and periolandic area, i.e., impacting the deep grey nuclei. This pattern of injury is ordinarily associated with an acute profound insult following the occurrence of an obstetric sentinel event.⁵
39. In his initial joint minute prepared with Dr Weinstein on 10 May 2019, he described the pattern of injury seen on baby D's MRI by its conventional nomenclature as '*Acute Profound Hypoxic Ischemic injury*'. 'Hypoxic' denotes too little oxygen whilst 'Ischemic' denotes too little blood pressure. Nomenclature such as 'Acute/Profound' gives a context of timing and severity, 'acute' denoting quick or sudden or abrupt cessation of blood supply or oxygen to the baby and 'profound' denoting how severe the insult is, i.e., a short severe process.
40. Thereafter, in November 2021, he came across an article published by Smith et al⁶ wherein the authors concluded that in the absence of a perinatal sentinel

⁵ Sentinel events being as defined by ACOG – 'Executive summary; neonatal encephalopathy and neurologic outcome, 2nd ed. Report of the American College of Obstetricians and Gynaecologists' Task Force on Neonatal encephalopathy 2014' – commonly referred to as the ACOG guidelines –examples include uterine rupture, uterine tear, placenta praevia, abruption placenta, umbilical cord prolapses, foeto maternal haemorrhage.

⁶ '*Intrapartum Basal-Ganglia-Thalamic Pattern Injury and Radiologically Termed "Acute Profound Hypoxic-Ischemic Brain Injury" are not synonymous*', published online in the American Journal of Perinatology in November 2020. The article is authored by eight medical experts from South Africa in the fields of obstetrics, paediatric neuroradiology, paediatric neurology and neonatology.

event, subacute or subthreshold prolonged or intermittent intrapartum hypoxic ischaemia may cause a BGT pattern brain injury but that warning signs in the form of non-reassuring foetal status, would be detectable by means of cardiotocograph (CTG) or auscultation monitoring up to a few hours before delivery. They thus concluded that the BGT pattern injury and radiologically termed 'acute profound' HI brain injury are not necessarily synonymous and that radiologists ought preferably to describe the pattern in relation to the areas of the brain that are damaged rather than to describe a causative mechanism of injury.⁷ In other words, it is inappropriate to describe the BGT pattern of injury as 'acute profound' in the absence of a sentinel event.

41. He testified that the 2019 revision of the ACOG guidelines distinguishes the description of an injury to the deep nuclear grey matter of the brain where a sentinel event has occurred from the description of an injury to the deep nuclear grey matter in the absence of evidence of a sentinel event. They describe an acute profound hypoxic ischaemic injury involving the deep nuclear grey matter in the context of an acute total hypoxic ischaemic insult, whereas they describe the BGT-HII injury, also involving the deep nuclear grey matter, in the context of severe partial insult of prolonged duration or a combined partial with profound terminal insult.⁸ In other words, ACOG 2019 recognises that the BGT pattern can occur in the absence of a sentinel event.

42. Thus there are two mechanisms that can result in the same injury pattern:

42.1. An injury resulting from a sentinel event;⁹ or

⁷ In the article, the authors point out that 'medicolegal lexicon often focus on descriptive radiological terminology which characterizes BGT pattern injury as an "acute profound HI brain injury." This has been taken to imply that such injury always occurs suddenly (acutely) and is so profound (severe and total) that very little, if anything, can be done to prevent fetal injury. This assumption is evident in earlier literature in which authors use "BGT pattern injury" and "acute profound HII" as synonymous.'

⁸ This is not the same as a partial prolonged hypoxic ischaemic event.

⁹ In an article published in the SA Journal of Radiology by Misser et al (Misser, Barkovich, Lotz Archary) in October 2020, titled 'A pictorial review of the pathophysiology and classification of the magnetic resonance imaging patterns of perinatal term hypoxic ischemic brain injury – What the radiologists need

- 42.2. Where no sentinel event occurred, an injury resulting from an insult that occurred over a prolonged period of either less than an hour or more than an hour.

By looking at an MRI image, radiologists cannot say which one of the said injuries occurred and one would need the clinical information to be in a position to say when and how the injury occurred. That is why he and Dr Weinstein agreed in their addendum joint minute that they (as radiologists) are unable to determine the cause/s or the time that the injury to the brain occurred and that a review of the clinical and obstetrical records by paediatric, neurology and Obstetrics experts is essential in determining the cause and probable timing of baby D's hypoxic ischemic injury.

43. Dr Alheit testified about a letter he addressed to the SA journal of radiology on the appropriate use of nomenclature when describing patterns of injuries.¹⁰ Dr Misser responded to this letter in his letter to the Editor of the SA Journal of Radiology and published therein, wherein he stated that in his co-authored article titled *'A pictorial review of the pathophysiology and classification of the magnetic resonance imaging patterns of perinatal term*

to know', the authors describe 'acute profound' ischemia as follows; *"In the setting of acute cessation of perfusion with rapid progression (eg in abruptio placentae), there is insufficient time for the cerebral autoregulatory mechanisms to adequately redirect blood flow to the high metabolic areas of the brain... The failure to protect these areas will result in a primarily central injury pattern with selective neuronal necrosis."*

¹⁰ The letter was published in the SA Journal of Radiology in May 2021 under the title *"Letter to the editor: Addressing radiological terminology of basal ganglia and thalamic injury in hypoxic ischaemic injury"*. A copy of this letter is to be found at V41 of Caselines. In the letter, Dr Alheit states, *inter alia*, as follows:

"Why is it important for us NOT to report the involvement of the deep nuclei in perinatal hypoxic ischaemic as an 'acute profound' pattern? Smith et al. draw attention to a medicolegal lexicon, which has evolved in South Africa specifically, where it is implied that 'acute profound HIBI is 'always' sudden (acute) and 'always' profound (severe and total). This view has fostered the belief in courts that very little could have 'ever' been done to arrest the process of neurological injury where that injury is reported as 'acute profound' on MRI... If there is no definitive confirmation of a preceding sentinel event, a radiologist is not in a position to deduce from the structural damage identified on MR images under what clinical or obstetrical conditions this type of injury occurred...the use of the term 'acute profound' remains valid, provided it is used in accordance with the ACOG 2014 definition of an acute profound injury and there is definitive obstetrical evidence of a sentinel event that preceded the insult... Radiologists should defer to clinical and obstetrical experts to advise on the clinical context, the probable causation, timing and severity of the insult."

hypoxic ischemic brain injury – What the radiologists need to know,¹¹ the cases therein described as ‘acute profound HIBI’ were in respect of children who had suffered sentinel events. Dr Misser pointed out that up until October 2020 (when the said article was published) radiological reporting of the central pattern of injury (the basal-ganglia, thalamus and periolandic injury), which they called acute profound injury, has previously been ascribed to an acute pathophysiology, but that cognizance was taken of the recent Smith et al article showing that a BGT pattern may also occur in the absence of sentinel events and is therefore not synonymous with acute profound HIBI. His group was therefore not opposed to the use of the anatomical description proposed, namely, to call this injury a BGT pattern where there is no correlative sentinel event documented.

44. In the definitive article titled ‘*Neuroimaging in the term newborn with neonatal encephalopathy*’ authored by highly esteemed medical professionals from a variety of fields,¹² published in October 2021, they describe the pattern of injury to the basal-ganglia thalamus and periolandic cortex (PBGT) of the brain as ‘Central/BGT’ whether caused by sentinel events or whether caused by severe partial asphyxia with prolonged duration or a combination of partial and near-total asphyxia. In other words, the latest literature does not use the term ‘acute profound’ to describe a central brain injury involving the PBGT structures of the brain.
45. Dr Alheit was asked whether an infection such as that sustained by baby D some twenty days after birth and which according to the hospital records, resulted in arthritis in his shoulder, would be visible on an MRI. He replied that a remote infection – if it is septicaemia- could get to the brain if it eventually

¹¹ Cited in fn 9 above.

¹² The article is co-authored by 9 international medical experts (including the renowned ‘doyan’ of radiology - A. James Barkovich) in the fields of radiology, paediatric neuroradiology and paediatric neurology and neonatology, who individually hail from either the USA, Canada, United Kingdom or the Netherlands.

forms a brain abscess. There was however no evidence of a brain abscess on baby D's MRI.

46. During cross-examination Dr Alheit testified that you cannot get the BGT injury without hypoxia. It is not possible to say how long it took for the injury to occur, as no-one knows. The only way is to postulate through foetal monitoring - if foetal distress is detected through monitoring, then one can postulate that the hypoxic insult probably started at that time. The central brain injury is due to a combination of hypoxia and ischaemia. Where there is no sentinel event, no-one can say when it started and when it concluded. But what is now known is that in the absence of a sentinel event, the injury does not happen suddenly – it can take place over a period of time.

Professor Anthony (Gynaecologist/Obstetrician)

47. Prof Anthony testified that the process of labour is a dangerous journey, particularly during the active phase of labour when contractions increase in frequency. During a contraction there is a reduction in blood supply and a decrease in oxygen content in the foetal blood. Generally, if there is sufficient time between contractions, the baby will be able to recover from a lack of oxygen experienced during contractions. If the contractions become too frequent or prolonged, the baby may not be able to maintain a baseline oxygenation as the mother's labour progresses.
48. Foetal monitoring is employed to detect the evolution of foetal hypoxia where the baby does not have enough oxygen due to contractions. Where the baby does not receive enough oxygen there will be a change in the acid content of the blood. This is detected in foetal circulation and these are translated into changes in foetal heart rate. The start of hypoxia is an indication of a potential insult which if sustained and severe may lead to injury. Therefore one looks at any change in the foetal heart rate pattern to detect any insult.

49. In order to determine from the foetal heart rate whether the baby is coping with the demands of labour, one would have to listen to the baby's heartbeat before, during and after uterine contractions. An attempt to measure the foetal heart rate without reference to contractions means that the nurse will not be able to ascertain or deduce the wellbeing of the foetus with certainty.
50. From a perusal of the maternity records, there is no evidence that the nurse/s monitored the foetal heart rate at the required intervals (every half hour in the active stage of labour, precisely because a hypoxic insult may occur) or in the correct manner (auscultation before, during and after contractions) as prescribed in the 2007 Maternity Guidelines. As the labour progresses, so the frequency of monitoring increases because it is important to know the state of the foetus so that timeous intervention can occur in the event that foetal distress is suspected or diagnosed.
51. In the case of baby D, prior to 7am on 9 July 2012, his recorded heart rate which was recorded, fell within the normal baseline range between 120 and 133 bpm. However, at each assessment, only a single heartbeat was recorded. The recorded heartbeat may or may not represent a foetal heart rate before or after contractions. So for example, at 04h00 the labour record reflects the foetal condition as 'satisfactory at 133bpm'. However, such a record denotes no more than that the baby was alive because one cannot determine foetal wellbeing without auscultating the foetal heart rate.
52. At 7 am, the records reflect the presence of thick meconium and an abnormal heart rate of 109bpm.¹³ At this point there ought to have been a real suspicion of foetal distress and that the baby was becoming or had become hypoxic. The maternity guidelines prescribe CTG tracing to evaluate the foetal condition

¹³ According to the maternity guidelines, a normal heart rate falls between 110 and 160 bpm.

more accurately in a case of suspected foetal distress but if that is not available, the foetal condition could be assessed by means of auscultation every five minutes or after every second contraction with a stethoscope. Intervention is required to manage foetal distress and it is vitally important to take appropriate steps to alleviate the lack of oxygen the baby would be experiencing. The correct protocol is to administer oxygen to the mother in order to increase oxygen in the circulation, and to have the mother lay on her left lateral side. Further steps include attenuating the contractions as best one can, so as to try and let the baby catch up on oxygen intake between contractions, e.g., by the use of tocolytic drugs to reduce the frequency and duration of the contractions in order to restore oxygenation to the baby, and performing intrauterine resuscitation 'until you can get the baby out.' However, if delivery is imminent in that the cervix is fully dilated *and* the baby's head has descended onto the perineum of the pelvic floor, then expedited delivery by means of vacuum extraction or the use of forceps to pull the baby out of the birth canal ought to be performed. The maternity guidelines allow for one hour from the time a decision is made that delivery is necessary, to the time that the delivery occurs. In the case of baby D, there was no way that the staff at Hillbrow clinic could have foreseen that delivery would take place naturally within one hour.

53. If the mother is encouraged to start pushing at a time when thick meconium is sighted (the presence of thick meconium being compatible with a diagnosis of foetal hypoxia), by pushing, she will thereby increase her intrauterine pressure, which decreases the placenta perfusion and oxygen supply to the baby. The mother is usually lying on her back when she is pushing. In the context of probable foetal distress when meconium was sighted, immediate intervention was required to manage the foetal distress by restoring oxygen to the baby until such time as delivery could be expedited.

54. Foetal monitoring at 5 minute intervals or after every second contraction is necessary during the second stage of active labour because contractions tend to cause a reduction of oxygen to the foetal blood and the baby's vulnerability to foetal distress is at its greatest during this stage of labour. When the mother is lying on her back she pumps less blood through the peripheral circulation at a time when it is most needed.
55. It is common practice for a nurse to administer oxytocin drugs whilst the baby is in the process of being born in order to get the mother's uterus to contract after delivery. However, if oxytocin was given to the plaintiff before the baby D was being delivered, it would have stimulated foetal contractions, thereby worsening any underlying hypoxia.
56. In the case of baby D, what was done by the nurse in the second stage of active labour was inappropriate. The baby was showing signs of distress due to not having enough oxygen available, as evidenced by the baby's abnormal heart rate and the presence of thick meconium at 7am. The presence of thick meconium at 7 am means it probably developed prior to 7 am. What the nurse ought to have done is to *first* address the foetal distress by alleviating the lack of oxygen to the baby and then to expedite delivery. No foetal resuscitation was carried out on baby D and oxygen was also not given to the mother. No attempt was either made to expedite delivery. Instead, labour was allowed to continue normally for another hour after 7 am instead of definitive attempts being made to restore oxygen supply to the baby and to deliver by means of assisted delivery.
57. During the period between 4 to 6 am and between 6 and 7 am there is no record evidencing any monitoring whatsoever of the foetal condition. This was in breach of the maternity guidelines. It is thus possible that baby D could have

become hypoxic and that signs of foetal distress existed prior to 07h00 on 9 July 2012 without this being detected and diagnosed.

58. In the case of baby D, there was a lapse in monitoring and a failure to assess the foetal condition through auscultation at the required intervals in accordance with the standard of care required in terms of the maternity guidelines. In addition, the nurse's response to the evidence of foetal distress at and after 7am was inappropriate. The partogram used in the case of baby D was not completed properly either. No observations of the foetal condition or contractions were plotted on the partogram after 4 am. The nursing staff at Hillbrow clinic staff would have been blind as to the condition of the foetus between 4 and 7 am. The partogram is used to document the process of labour – it is a pictorial graph that shows what happens during labour over time. When used and completed correctly, a nurse would be able to detect abnormalities in labour so that care and intervention can be provided *before* there is an adverse outcome to the baby. This all cumulatively amounted to substandard care which culminated in an adverse outcome in this case.
59. Baby D's apgar scores of 6/10 at 1, 5 and 10 minutes indicated that the baby was not a normal/well baby. Baby D required resuscitation after delivery. The neonatal records indicate that baby D developed convulsions (seizures) and was diagnosed with HI encephalopathy about two days after birth. Baby D was also hospitalised after birth. A baby who is well would not require hospital care
60. Foetal monitoring is designed to detect a hypoxic insult so that intervention can take place before an injury occurs to the baby. Obstetricians do not know for how long the baby can endure foetal insult or when precisely an injury occurs. All that is known is that where there is evidence of hypoxia, immediate intervention is needed to restore oxygenation to the baby and if foetal hypoxia remains, to expedite delivery before injury occurs.

61. Prof Anthony was asked to comment on the defendant's version, namely that the kind of central brain injury Baby D sustained occurred suddenly or unpredictably and was so severe (the conventionally described 'acute profound' HI injury) that nothing could have been done to prevent it. According to Prof Anthony, Neuro-Radiologists have conflated the process giving rise to the injury, with the injury itself. A sentinel event means an alerting or overt event that one cannot help but observe and which would have to be noted in the obstetric records. It is always overt and seen. A sentinel event is an acute profound event that gives rise to the ultimate brain injury. There is no evidence that a BGT injury can *only* take place in the presence of a sentinel event. There is substantive expert opinion that indicates that a rapid shutdown of oxygenated blood is not the only mechanism by which this BGT injury can occur. So the presumption that with a BGT injury, the mechanism is an acute profound obstetric insult (characterised by rapid total shutdown of oxygenated blood in which compensatory mechanisms have no time to become deployed) is no longer correct.
62. Where there is evidence of foetal distress/hypoxia and even if the only feasible action is to deliver the baby, then protocol still requires that whilst taking steps to expedite delivery, actions that may exacerbate the lack of oxygen to the baby are to be avoided and interventions such as administering oxygen to the mother; avoiding having the mother start pushing so as to allow the baby to recover from a lack of oxygen during contractions, and carrying out foetal resuscitation is called for. In other words, there is therefore still a process to be followed and active steps to be taken to prevent or mitigate final injury.
63. In this case, there is no evidence that a sentinel event occurred. Had it presented, it would no doubt have been noted in the obstetric records.

64. Any problem with the mother's placenta, i.e., some form of placental disease, would usually lead to growth restriction. Based on his birth weight of 3.999kg, baby D was not growth restricted.
65. Dr Mantha-Crywagen's opinion in the amended joint minute, namely that there was no evidence of foetal distress in the first stage of active labour prior to full cervical dilation is true only to the extent that what was recorded as the foetal heart rate and that it was 'satisfactory' did not demonstrate the presence of foetal hypoxia, however, FHR monitoring was inadequate and not performed in accordance with the maternity guidelines and hence any foetal distress would not have been detected.
66. Management of foetal distress is provided for in the maternity guidelines. It is not just managed by expedited delivery. Expedited delivery is a proponent, but it is not the first thing that has to be done. The first thing is to give the baby the oxygen he/she needs so that the baby survives. Intervention would include lying the mother in the left lateral position; give oxygen to the mother by face mask at 6L per minute; start an intravenous infusion of ringers lactate to run at 240ml per hour (this is a non-colloidal infusion which assists in mopping up acid in the mother's bloodstream); perform a vaginal examination to exclude cord prolapse; if delivery is imminent, then deliver immediately by vacuum extraction or forceps. If delivery is not imminent, give a tocolytic drug to stop contractions and prepare for a caesarean delivery and urgent transfer to a hospital. If at the time that a problem comes to light, i.e., foetal distress is noted, the baby's head is already deep into the mother's pelvis, then it would be permissible to deliver by vacuum extraction.
67. In this case, an abnormal heart rate and presence of thick meconium liquor was sighted at 7 am. A finding of meconium reasonably implies that it was present for an unknown period of time before being sighted because of

inadequate monitoring. Baby D was born one hour later at 8am. This was not a rapid extraction or expedited delivery. By 7am it was not foreseeable that the baby would be delivered naturally at 8 am. The guidelines allow for 2 hours after full cervical dilation (10cm) for the descent of the baby's head and delivery. Whilst the guidelines prescribe that delivery must occur within one hour of noting some sort of abnormality, in this case the nurse allowed a natural birth to proceed for an entire hour in circumstances where there was no way to predict how long the second stage of labour would take or that an unassisted vaginal delivery would take place within that hour. The guidelines state if delivery is imminent, deliver *immediately*. This implies a high degree of urgency once foetal distress is suspected or diagnosed - it is not a licence to wait for up to one hour before delivering. Once foetal distress is noted one cannot just let labour continue naturally. This would be prejudicial to an already hypoxic baby. The midwife is required to deliver immediately if delivery is imminent and that means intervening to deliver quicker than the one hour allowed for delivery. The process of management of foetal distress has to do with rescuing the baby by restoring oxygen supply to the baby and by foetal resuscitation. This was not done in the case of baby D.

Dr Kara (Paediatrician))

68. Dr Kara's evidence is on record and only a few salient aspects of his testimony need be referred to for purposes of judgment.
69. Dr Kara stated that the medical records confirmed that there was evidence of Hypoxic Ischemic Encephalopathy at birth – the baby had an abnormal tone, did not cry, experienced respiratory distress at birth requiring resuscitation, was lethargic, could not suckle and also experienced convulsions after birth.
70. From what is contained in the medical records, after 4 am there are only two records of monitoring, namely, at 6am; and at 7am at which time thick

meconium was observed and an abnormal heart rate was detected. Between 4am and 7am something happened, because at 7am, thick meconium was *already* present when it was detected. The presence of thick meconium is a sign of foetal compromise. In his opinion, an insult (evidenced by the recorded deceleration in baby D's heart rate and presence of thick meconium) most likely occurred sometime between 4am and 7pm, which insult led to the brain injury that occurred thereafter but before baby D was delivered.

71. Baby D's apgar scores remained at 6/10 at 1 minute and 5 minutes and 10 minutes. That means that 9 minutes later, the baby was unresponsive to resuscitation. A score under 7/10 is a sub-standard score, meaning that baby D was not in a good condition but was compromised. Usually a baby's apgar scores will improve after some stimulation and resuscitation. A score of 6/10 at 10 minutes shows that resuscitation either did not improve the baby's condition or it was improperly performed. The need to transfer baby D to a neonatal ICU at Charlotte Maxeke hospital after birth supports the conclusion that he had neurological depression or compromise at birth. Two to three weeks after birth, baby D was hospitalised and diagnosed with septic arthritis. Arthritis could be caused by an infection but such infection was only present long after the diagnosis of 'Hypoxic ischaemic encephalopathy grade II' some two days after birth.

72. Dr Kara supported Prof Anthony's opinion that in the face of poor monitoring of the foetal heart rate between 4am and 7am, the presence of thick meconium foetal bradycardia (slow heart rate) at 7 am and constituted enough evidence of foetal distress.

73. Nobody is able to tell from MRI imaging exactly when an injury occurred. One can only deduce that there has been a sudden unanticipated injury that occurred *if* proper foetal monitoring took place and which evidenced normal

signs until suddenly something happened from whence abnormal signs were noted.

74. Some experts have suggested that the 'acute profound' pattern of injury on a MRI scan means that the brain injury would have occurred in the last 30 to 60 minutes of labour (otherwise the baby would be dead). Dr Kara stated that this may not be correct, for as stated by Professor Volpe in his textbook 'Neurology of the Newborn'¹⁴: "80-90% of cases of infants with hypoxic ischaemic disease in which [an] overt sentinel event is not present, the uncertainty in timing is often measured in hours, often many hours or more and not minutes."

75. Dr Kara confirmed what was stated in his report, namely that:

"Volpe quotes 4 studies involving foetal lambs that suggest that "the severe terminal insult that results in injury to deep nuclear structures especially, may be likely to occur after brief repeated hypoxic ischaemic insults first cause a cumulative deleterious effect on cardiovascular function that presumably then can result in a severe late insult." This means that it is probable that effective foetal monitoring would be able to identify the "at risk" foetus as the heart rate would alter (and be detected), followed by cardiac compromise and then reduced blood supply to the brain. This may allow for intervention before brain injury can manifest, noting that it is stated that repeated brief hypoxic insults occur before cardiac compromise occurs.

In this case, we have no means to identify exactly when in the labour the injury occurred but it probably occurred over hours and not minutes, (with a sudden decompensation that led to basal ganglia and thalamic injury). If it did occur over a process of hours, there would have been opportunity to detect warning signs of potential catastrophic brain injury had there been good foetal monitoring.

The available records do not confirm foetal well being in labour (from 04.00 onward in particular as there was poor foetal monitoring), It then cannot be stated that there was a sudden deterioration in the foetal condition late in labour. It is likely that the injury could have been anticipated and possibly prevented if the foetal monitoring was done according to [maternity] guidelines." (emphasis added)

¹⁴ J J Volpe *Neurology of the Newborn* 6 ed (2018) at 503.

76. The importance of foetal monitoring is to arrest the abnormal situation from worsening. There could be a hypoxic insult that progresses or increases over time, i.e., a gradual deterioration of oxygen and blood supply to the baby and if the insult is detected, injury could be prevented if the baby were to be delivered before any injury occurs. In other words, if the labour is monitored appropriately, there is a good probability that the attending medical practitioner will detect signs of foetal compromise and be able to act/intervene before the catastrophic injury occurs
77. By the time baby D developed septic arthritis a few weeks after birth, according to the obstetric records, neonatal encephalopathy had already been diagnosed weeks before. In this case, it has already been determined that baby D's cerebral palsy was caused by a hypoxic ischaemic injury. Thus Baby D's brain was already damaged by the time the septic arthritis arose. There is nothing in the records to support a conclusion that either the use of Khat or the development of septic arthritis some weeks after birth was a cause of the cerebral palsy. In particular, there is no record that postnatally, baby D went into postnatal septic shock, which if it occurs can lead to HI injury.

Dr Pierce (Paediatric Neurologist)

78. Dr Pierce testified that it was common cause between herself and Dr Mogashoa the timing of the insult and injury is that it occurred in the intrapartum phase given that there was no record or evidence of an identified sentinel event in this case.

Defendant's witnesses

Dr Mogashoa (Paediatric Neurologist)

79. Dr Mogashoa confirmed that baby D's birth weight of 3.999 kg was normal and evidenced that no growth restriction was present. Likewise, his length was normal and not suggestive of any abnormality.

80. Dr Mogoashoa confirmed the contents of her expert report and her conclusions therein. She testified that the injury to baby D's brain was caused by hypoxia during the intrapartum period. This is borne out by the labour records in that after birth, baby D was unwell and had to be transferred to a hospital for management and care. Apgar scores of 6/10 at 5 and 10 minutes after birth indicate that baby D was depressed at birth.
81. As highlighted in her report, the definition of neonatal encephalopathy, according to ACOG, is that it is a clinically defined syndrome of disturbed neurologic function in the earliest days of life in an infant born at or beyond 35 weeks of gestation, manifested by a subnormal level of consciousness or seizures, and often accompanied by difficulty with initiating or maintaining respiration.
82. Placental insufficiency is a lack of adequate supply of blood or oxygenation to the foetus.
83. At 7am on the morning of 9 July 2012 the foetal heart beat was compromised and meconium stained liquor was visible. Dr Mogashoa agreed with Prof Anthony that there may have been signs of foetal distress before 7am, which went undetected, because the 6.30 am assessment was not performed.
84. Under cross-examination, Dr Mogashoa stated that the clinical picture found in respect of baby D is that he suffers from dystonic or diskietic cerebral palsy, being a signal of a particular brain injury referred to either as acute profound or BGT injury. She is aware of the debate as to whether radiologists should refer to the pattern of injury as acute profound HI injury in the absence of a sentinel event. In her view, acute profound suggests a particular mechanism and timing of the injury i.e., how and when it occurred, and radiologists cannot

determine this from looking at an image on a MRI scan, therefore it makes logical sense to describe only the anatomical areas of damage in the brain.

85. With the use of the term 'acute profound' the conventional/traditional thinking has been that there is a sudden severe insult which is not compensated for and which will result in damage to the most metaphysically active areas of the brain such as the PBGT. In other words, if it is not sudden, the baby will try and compensate for a lack of oxygen/blood supply in order to spare the central brain from being injured. If oxygen/blood supply is suddenly totally cut off, then the GBT is not spared and is affected. But having heard Prof Anthony's evidence, Dr Mogashoa stated that she accepts that if there is another way that the same BGT injury can be caused, namely by evolving sub-threshold hypoxia. In other words, if there is no evidence of a sentinel event that results in a sudden, total cut off of oxygen and blood supply, then one cannot attribute the mechanism of the injury to a sudden total cut off of oxygen and blood supply to the baby, as denoted by the words 'acute profound'.

Professor Bolton (Paediatrician)

86. Prof Bolton confirmed his amended report which he prepared after receipt of further records in the matter.
87. In his evidence in chief, he confirmed that it is essential to suction meconium from the baby's mouth and before the baby takes his first breath so that the baby does not inhale it into his lungs and develop respiratory distress. Baby D had respiratory distress at birth, requiring admission to hospital.
88. "Asphyxia" means a state of the baby at birth –a sub-optimal neurological condition due to hypoxia. In other words, because of hypoxia, the baby does not breathe adequately after birth and has low apgar scores. "Hypoxia" is

inadequate oxygen circulation in the baby's blood and the brain is the organ in the body that needs it most.

89. All babies become hypoxic during labour with each contraction but are usually able to recover from it. It becomes problematic when the baby is unable to continue to manage the deprivation of oxygen during contractions. The foetal heart rate during a contraction is always slow. One therefore looks for a sustained heart rate dip after the contraction. A baby can become hypoxic systematically and be unable to cope, which constitutes foetal distress. We call it foetal distress when there is a delay in recovery from a lack of oxygen during a contraction, which is marked by a persistent low foetal heart rate after the contraction.
90. In the case of baby D, at 7am on 9 July 2012, the simultaneous passage of meconium and drop in foetal heart rate would suggest that there was foetal distress detected at that point.
91. According to the 2014 Acog consensus statement, before ascribing an outcome of HI encephalopathy and cerebral palsy, one must consider all the possible causes, because multiple causes can lead to brain injury in term infants, not just oxygen deprivation around the time of birth.¹⁵ Possible causes are from intrapartum events, a positive HIV status, severe neonatal infection within the perinatal period or inadequate placental function. Here, meconium aspiration at birth, the mother's use of khat (which when taken during pregnancy has been shown to have a deleterious effect on foetal outcome) and the baby's infection 18 days after birth were factors Prof Bolton stated

¹⁵ . The ACOG task force states that a comprehensive, multidimensional assessment should be performed. It is to include the neonatal status and all contributing factors including the mother's medical history, prior obstetric history, factors such as foetal heart rate monitoring results, any delivery issues and placental pathology.

that he considered in this case as possible causes of Baby D's adverse outcome.

92. When reviewing the neurodevelopmental records from Charlotte Maxeke hospital, Prof Bolton testified that he came across an inscription made by the attending doctor under the heading 'Birth history', which queried the possible use of Khat/Mira by baby D's mother. The note read: "*? chewed tobacco – occasionally (called Mira)*" He interpreted the note as a query concerning what the mother had chewed – ie., tobacco or mira. He further testified that according to some literature, the effects of khat are said to be intrauterine growth restriction, congenital abnormalities and it has also been associated with foetal distress and low Apgar scores at birth and post-partem haemorrhage. In this case, no growth retardation was evident and no congenital abnormalities were found. Nor was there evidence of inadequate placental function, thus he could not say in this case that there was inadequate placental function.
93. An acute profound injury to the PBGT structures is due to an acute (sudden) profound (severe) drop in oxygen and blood supply to the brain. The timing of the injury cannot be determined from a MRI scan performed years later.
94. There are many causes besides hypoxic ischaemia for neonatal encephalopathy, one of which is meconium aspiration. According to the Hillbrow clinic delivery records, baby D was transferred to the hospital for meconium aspiration. However it is possible that a severe systematic infection at day 18 after birth could have caused the damage as babies born with asphyxia are predisposed to later infection. In this case, baby D was diagnosed with sepsis 20 days after birth.

95. The common cause for bradycardia is due to hypoxia to the foetus. If it is sustained after a contraction it suggests a failure to compensate for the deprivation of oxygen and is reflective of persistent hypoxia.
96. During cross-examination Prof Bolton made significant concessions. These included that:
- 96.1. There is a high degree of probability that the insult occurred in the intrapartum phase although Prof Bolton was not willing exclude other causes;
 - 96.2. He agreed that the objective medical records reflect that a medical doctor (Dr J Groenewald at Charlotte Maxeke hospital) had diagnosed baby D with grade 2 HI encephalopathy two days after his birth;
 - 96.3. The medical record from the neurodevelopmental clinic at Charlotte Maxeke hospital does not refer to the plaintiff as having chewed Khat during her pregnancy;
 - 96.4. In so far as his amended report recorded that the plaintiff had *admitted* to having chewed Khat during her pregnancy, this was incorrect and could not either be assumed;
 - 96.5. Since he has been unable to locate certain additional source documents that he relied on for his opinions in his amended report, and without such source documents having been discovered at the trial, his opinions in his amended report were unsustainable;
 - 96.6. His opinion regarding other causes of neonatal encephalopathy (use of khat during pregnancy and the infection some weeks after birth) and the timing and mechanism of the injury sustained by baby D could likewise not be supported or sustained.

Sister Moqhae (midwife who delivered baby D)

97. The witness testified that she obtained a diploma in advanced midwifery in 2011. On 9 July 2012 she was on duty at Hillbrow clinic. She was part of the

day shift that took over from the night shift with her duties commencing at 7am. She confirmed that she was familiar with the 2007 guidelines for maternity care during July 2012.

98. When she assessed baby D's mother at 7am, she was 10 cm dilated and therefore the plaintiff was moved to the delivery room that is located inside the labour ward. The mother was restless and screaming and had told Sr Moqhae that she had an urge to 'do a number 2' i.e., pass a bowel movement, hence Sr Moqhae recorded in the obstetric records that the plaintiff had an urge to push. When a mother is fully dilated she would usually experience 4 to 5 contractions in the space of ten minutes. Nurses use a foetoscope at Hillbrow clinic to check the foetal heart rate, and do not make use of CTG tracings as they do not have a CTG machine at Hillbrow clinic. There is however a stethoscope available for use at Hillbrow clinic. At the 7am assessment, she recorded the FHR as 109 bpm. This was the heart rate that was measured after the plaintiff's contraction. She was unable to recall what the FHR was before the contraction, but stated that she checked the FHR both before and after contractions. The plaintiff's liquor was stained with meconium and was very thick and she thus graded it as grade 3. That meant that the foetus was in distress at the time. At 7am she also performed a vaginal examination at which time the baby's head was not visible on the perineum of the pelvis but 'was still high up.' When performing a vaginal examination she is able to tell how far the baby's head is from the perineum.
99. Since the plaintiff was fully dilated with an urge to push, she encouraged her to push 'as pushing encourages the baby's head to descend.'¹⁶ As the plaintiff

¹⁶ The maternity guidelines prescribe as follows

: "From the time that full dilatation of the cervix is first noted, up to 2 hours may pass before the mother starts to bear down. **Time can only be allowed for the head to descend onto the pelvic floor if fetal distress and cephalopelvic disproportion have been ruled out.** The bladder should be emptied, using a catheter if necessary. The observations of the first stage of labour should continue. **Efforts at bearing down are only encouraged when the fetal head starts to distend the perineum and the mother has an urge to push.**" (emphasis added)

could not understand much English, she demonstrated to the plaintiff with gestures on how to push. The plaintiff only started pushing at 7h30am. At this time the baby's head was still not visible although she believed that it was 'near'. She checked the plaintiff every 15 minutes whilst she was bearing down, however she stated that she was unable to record her findings anywhere. She screamed for Sister Mabanga to assist her as she did not want to leave the plaintiff alone.

100. Baby D was delivered at 8am, a half an hour after the plaintiff started to push. Before the baby's full body came out, she 'tried to wipe meconium from the baby's mouth'. After the baby came out she put the baby onto the mother's chest and cut the umbilical cord. She then suctioned his mouth because the baby was not crying. According to the witness, when the baby came out he had meconium aspiration which blocked his airways. Thereafter she and sister Mabanga started resuscitation and used a bag and mask to administer oxygen to the baby as he was struggling to breathe and also put up a drip to administer fluid to the baby intravenously. Baby D's heart rate was 140 bpm after delivery and his respiratory effort was graded as 1/10. Sister Mabanga was present when the baby was delivered and Sister Pasha also came to assist because it was an emergency. They notified the hospital that they had a baby who had aspirated meconium and who was in distress and that they needed to transfer him for further management.

101. Sister Pasha took over management of the plaintiff. She was bleeding profusely and therefore fluids (ringers lactate and oxytocin) were administered to her intravenously by means of a drip in an attempt to control the post partem haemorrhaging.

102. Sr Moqhae was asked how she managed the foetal distress she had observed at 7am. She initially stated that she had reassured the plaintiff and had

encouraged her to bear down so that the baby could be delivered. After being referred to the maternity guidelines on how to manage foetal distress,¹⁷ she stated that whenever she asked the plaintiff to bear down, the plaintiff would lie on her left lateral side, however, she would also change sides and therefore did not always lie in a supine position. Later during cross-examination she stated that the plaintiff would turn whenever she did not have contractions. Because she believed that delivery was imminent (as the plaintiff was fully dilated) she 'tried to deliver the baby immediately'. They did not have vacuum extraction equipment at the Hillbrow clinic.

103. During cross-examination the witness confirmed that the partogram that was used by the night staff between the hours of 0h00 and 04h00 was inadequately completed. It only showed the foetal heart rate but did not reflect if there were decelerations after contractions. The plaintiff's contractions were not plotted, and the rate of descent of the foetal head was also not recorded. She agreed with the proposition put to her, namely, that if

¹⁷ According to the maternity guidelines, foetal distress is suspected when certain signs are observed. These include, amongst others, a baseline foetal heartrate of less than 110 bpm and late decelerations of the foetal heart rate.

Management of foetal distress is as follows:

1. Explain the problem to the mother.
2. Lie the mother in a left lateral position.
3. Give oxygen by face mask at 6 L/minute.
4. Start an intravenous infusion of Ringer-Lactate to run at 240 mL/hour.
5. Do a vaginal examination for cervical dilation and to exclude cord prolapse..

If vaginal delivery is imminent (cervix fully dilated), deliver immediately, by vacuum extraction if necessary.

If vaginal delivery is not imminent, give hexoprenaline 10 micrograms IV and prepare for immediate caesarean section. Arrange urgent transfer from a community health centre to hospital.

the partogram only reflects the foetal heart rate without reflecting if there were decelerations, the nurse would not know if the child was coping with contractions and thus there is a strong possibility that abnormalities in FHR would be missed. Sr Moqhae also stated that it is important to plot the rate of deceleration of the FHR as it enables the nursing professional to intervene if problems manifest.

104. At 0h00 on 9 July 2012 the plaintiff's cervix was 4cm dilated which meant that she had entered the first stage of the active phase of labour at which stage, according to the witness, the FHR ought to have been checked every 2 hours.¹⁸ In this case, the witness stated that it was not checked at the required intervals. For example, no assessment took place at 2am. After 4 am until delivery, nothing at all was plotted on the partogram.

Sister Mabanga (Midwife)

105. Sr Mabanga confirmed that she is trained as an advanced midwife. She was in charge of managing the maternity department at Hillbrow clinic in July 2012 in the capacity of operational manager and team leader. On 9 July 2012 there were five midwives on duty (including herself) during the day shift, which started at 7am.
106. She was asked what management she would give if she were to find a patient where signs of foetal distress were observed, such as draining of thick meconium stained liquor and where the foetal hear beat was 109 bpm. She replied that she would question where the baby's head was in relation to the pelvic rim (perineum). She would also assess the rate of contractions as contractions usually intensify and tend to be expulsive when the baby's head has descended. If the head has descended and is already on the perineum and

¹⁸ The maternity guidelines prescribe that when the cervix is 4 cm dilated, the FHR ought to be checked **half-hourly before, during and after contractions**, using a hand held Doppler instrument.

the mother is fully dilated at 10 cm, this would determine if delivery is imminent or not. In that event, she would take steps to deliver the baby immediately.

107. She testified that she was called to assist with possible resuscitation when delivery of baby D was 'happening'.

108. At Hillbrow clinic the midwives use a foetoscope to determine the FHR. The clinic did not have a hand held Doppler in July 2012. She started ordering hand held Dopplers subsequent thereto. She indicated that she was more comfortable in using a foetoscope as opposed to a Doppler or stethoscope, as midwives are not trained to use stethoscopes to auscultate FHB. They are only trained to listen to the chest of the mother with a stethoscope.

109. She confirmed that midwives are trained to determine the FHR before, during and after contractions. The FHR measured before and after contractions must be recorded. However, she stated that the midwife tends not to get an accurate reading of the FHR when using a foetoscope as a tool or instrument. The midwife would have to have a good timing of the end of a contraction to note the FHR. It's a tricky situation to time when the contraction has subsided. If a woman has a contraction the uterus is hard and intense pain is experienced by the mother, which makes it very difficult to put the foetoscope on the contracted abdomen because the mother tends to be very restless and is moving around, i.e., is not lying in a supine position all the time. Therefore it happens that by the time the midwife starts feeling for the FHR, the contraction has stopped a long time ago. Midwives use the mother's contraction and the FHR to determine whether any deceleration is late or early. It is not a common practice to record the FHR before, during and after contractions.

110. A normal FHR is between 110 and 160 bpm. If it drops below 110 bpm you would want to know the duration of such deceleration.
111. According to the labour records, at 4am the plaintiff's membranes had ruptured and she was draining clear liquor. At 6am, the mother was 9cm dilated. There was no record of any liquor draining at 6am. The descent of the baby's head was also not recorded.
112. The known principle in nursing is that what was not recorded, was not done.
113. At 7am the plaintiff was draining thick meconium stained liquor. The records reflect only one recording of the FHR at 109bpm. One reading cannot give you confirmation of bradycardia but because of the presence of meconium stained liquor (grade 3), there was foetal bradycardia or foetal distress. The correct protocol would have been to insert a drip of ringers lactate and to administer oxygen to the mother.
114. If a midwife decides to transfer a patient to a hospital, the process is as follows; (i) phone the labour ward and locate the obstetrician on call. Phone the doctor on call and discuss the patient's history, how far she is dilated, state of contractions, foetal distress. If the doctor accepts the patient, then (ii) call the emergency medical services control to request an ambulance. It depends if an ambulance is available as to how soon the ambulance will arrive. At the hospital the patient is taken to maternity admission and admitted to the labour ward. In most cases the patient will be escorted by the midwife in the ambulance in case she delivers in transit. The doctor would examine the patient and decide if an emergency caesarean has to be performed or whether to deliver by vacuum extraction or by way of normal vaginal delivery. If a caesarean has to be performed in hospital, the patient's consent has to be

obtained and other preparations would have to be done before the patient is taken to theatre. Between 7 to 8 am all this was unlikely to be achieved.

115. Under cross-examination the witness stated that although the delivery record reflects that she assisted in the delivery of baby D, her only independent recall is that she took part in the resuscitation after delivery.

116. She further confirmed that information plotted on a partogram is to provide a pictorial view of the progress of labour from admission until delivery. It is used to alert any medical practitioner of any pathological development during the course of labour. In this case, the partogram was not completed properly from 0h00 to 4h00 on 12 July 2012 in that the FHR before, during and after contractions was not plotted. The rate of descent of the baby's head was also not recorded at all.

117. In a situation where the mother is draining meconium stained liquor and the foetal heart rate has dropped below 110bpm, the witness stated that she would not have doubted that the foetus was in distress. In such a situation she agreed with the proposition put to her, namely, that the first obligation would be to relieve the baby's distress. As there was no record of the descent of the head at 7am at all, one could not determine that delivery was imminent, more particularly, as there was no record of the descent of the head in relation to the pelvic brim. Delivery is imminent if the patient is fully dilated *and* the head has descended onto the perineum of the cervix. In the event that delivery was indeed imminent, the guidelines require immediate delivery, by vacuum extraction, if necessary. The witness agreed with the proposition put to her that the priority in the face of foetal distress is to get the baby in a good enough condition to deliver without injury or further injury to the baby. She also agreed that if the only route is to deliver because the baby's condition is deteriorating rapidly, then one should expedite the delivery eg by using

forceps, although the witness qualified this by stating that midwives 'can't do vacuum extraction' and given that she herself last did vacuum extraction in 2008, her competency level could not be guaranteed.

118. Based on incomplete records, the witness conceded that it could not have been known at 7 am that delivery was imminent because the rate of descent of the head was not recorded and the position of the head in relation to the pelvic brim was not recorded. The witness agreed that the guidelines require that contractions be slowed down or stopped and tocolytic drugs administered to the mother in order to buy time for the foetus to recover between contractions and for the mother to be transferred to a higher level of care. On the records as they stand, the mother was not given ringers lactate intravenously, the foetal heart rate was not assessed at all after 7 am and administration of a tocolytic drug to inhibit contractions was not considered.
119. The witness also agreed that it cannot be determined with certainty how long it would take for the child to be delivered once the mother's cervix has fully dilated – it could be anytime from 30 minutes to two hours before delivery would take place and therefore, in the face of foetal distress, the midwife might have had time to make arrangements to transfer the plaintiff to a hospital for higher care. She further agreed that on the available records, there was a lapse in how labour and delivery was managed in relation to the condition of the mother and foetus, and sub-standard care was given during the labour process. In this case, baby D's adverse outcome was most probably due to sub-standard care administered by the midwives.

Dr Manthata-Cruywagen (gynaecologist/obstetrician)

120. Dr Manthata-Cruywagen confirmed that she prepared an addendum report in which she changed her opinion as to the likely cause of baby D's brain injury that resulted from hypoxia. Initially she agreed on all aspects reflected in the

first joint minute signed by her and Prof Anthony. The amended joint minute sets out her amended opinion that the neurological outcome suffered by baby D was likely caused by placental compromise (where the placenta does not function as it is supposed to function) due to the mother's use of Khat during the pregnancy. The use of Khat during the pregnancy caused some form of placental pathology which only manifested in the last thirty minutes of the second stage of active labour.

121. Proper monitoring is used to detect foetal distress so that timeous intervention can take place to prevent an injury from occurring. The failure to monitor adequately does not cause the injury. Something else causes the hypoxia that culminates in injury. In this case it was most likely caused by compromised placental function due to the mother's use of khat during pregnancy. With a compromised placenta, in utero blood flow to the foetus is compromised. In the second stage of active labour contractions intensify in regularity. The baby cannot perfuse because the placenta is not functioning properly and will therefore not be able to compensate for the lack of oxygen experienced during increased contractions.
122. After receiving records pertaining to Charlotte Maxeke hospital's neurodevelopmental clinic, the witness noted what the attending doctor had recorded under the heading 'Birth History', namely: "*? Chewed tobacco-occasionally (called Mira)*" which she testified meant that the mother had admitted to using khat when she was pregnant. Because anything pertaining to 'birth history' relates to the mother's pregnancy and the plaintiff would have told the attending doctor who completed the record that she had chewed khat during her pregnancy. It is recorded that the mother used Khat (mira) on occasions, which means that she could have used it on the occasion of labour.

123. According to the witness, foetal monitoring is used to detect hypoxia but it does not prevent or cause the hypoxia. A lack of oxygen supply is caused by something else, be it a sentinel event or placental abnormality or something else such as the mother's heart or labour contractions that would inhibit oxygen/blood supply to the foetus. A foetal heart rate below 110 bpm signifies that the foetus is hypoxic i.e., there is reduced oxygen supply to the baby.
124. In her view, one cannot administer tocolytic drugs to a fully dilated mother. She reasoned as follows: 'why stop contractions in a woman who is going to deliver?' According to the witness, delivery is imminent when the mother has an urge to push. As the baby's head descends, it presses on the pelvic floor and it causes what is known as the 'ferguson' reflex so that the mother has the urge to push. The urge is there because the baby's head is there. Delivery is not imminent where the mother is not fully dilated and does not have the urge to push. If the head is not engaged in the pelvis then one can stop contractions but if there is an urge to push, delivery is imminent. If the head was higher than the pelvic brim then the mother would not have an urge to push.
125. In the second stage of active labour the guidelines prescribe that the FHR should be monitored after every second contraction, which could equate to every 5 or 6 minutes.
126. In her opinion, delivery could have been expedited by means of assisted delivery with an episiotomy in order to get the baby out as quickly as possible. However, the guidelines state that delivery must take place within one hour of a diagnosis of foetal distress. In this case, delivery in fact occurred within one hour of the diagnosis of foetal distress.
127. In her opinion, the injury to baby D's brain occurred within the last 30 minutes of labour when the uterine expulsive contractions were pushing the baby out

and that the injury resulted from a total sudden shut down of oxygen supply to the baby.

128. She disagreed with Prof Anthony that the cause of the injury was due to evolving hypoxia because 'acute profound' implies a rapid total shut down of oxygen supply to the foetus and therefore it cannot be evolving. Had it evolved and occurred over a long period of time, the pattern on the MRI would not have been that of 'acute profound' but of 'partial prolonged'.

129. In her view, evolving hypoxia means that the hypoxia (deprivation of oxygen supply) was 'going on and foetal distress was continuing' while the mother was pushing. In this case there was evidence (in the form of Khat usage by the plaintiff) of an abnormally functioning placenta, which the literature suggests could have predisposed the foetus to foetal distress. Monitoring is required to identify foetal distress and the guidelines specify how to manage the foetal distress. The management of foetal distress is to expedite delivery. The guidelines allow for one hour in which to deliver after diagnosing foetal distress. In this case the delivery occurred within an hour of foetal distress having been diagnosed. With an acute profound injury, it means that the injury occurred within the last 30 minutes of delivery.

130. She agreed with Prof Anthony that injury is avoidable by early detection of evolving hypoxia, however, in her view, the records reflect that there was no foetal distress during the period from 0h00 until 6h00. Had the FHR not been satisfactory (i.e., between 110 and 160 bpm) the midwife would have recorded this. Since baby D's FHR was not assessed at 6h30 am, she was unable to say that all was well with baby D at 6h30 am. Midwives normally record the FHB taken after a contraction. In labour, when there is a reduction in oxygenation, the baby tries to conserve energy and his/her heart slows down. That is when a deceleration happens, A late deceleration which comes

after the contraction is a sign that the baby is not getting enough oxygen, in which event the heart rate goes down.

131. During cross-examination the witness agreed that Charlotte Maxeke hospital is situated approximately 2 km from the Hillbrow clinic. In her estimation, it was located about 8 to 10 minutes away from the Hillbrow clinic.
132. She agreed with Prof Anthony that maternity monitoring in this case was not at the required standard, and that the FHR had to be monitored up until delivery, which was not done in this case, which amounted to sub-standard care. Furthermore there is no record of the descent of the foetal head, which also amounts to sub-standard care.
133. She also agreed that even if the mother is fully dilated but the baby's head has not sufficiently descended, delivery would not be imminent. As the position of the baby's head was not known at 7 am, the midwife could not have determined that delivery was imminent. It is when the baby's head reaches the pelvic floor that the 'ferguson' reflex kicks in so that the mother starts pushing involuntarily. This causes more contractions but the head still has to descend further until it reaches the perineum of the cervix. So at 7 am the head was in the pelvis but it still had to descend gradually.
134. She conceded during cross-examination that given the signs of foetal distress at 7 am and given her evidence that delivery ought to have been expedited by means of assisted delivery, it was not an option for the midwife to just stand by and let labour continue naturally – there needed to be an intervention. Later during cross-examination she reverted to the contrary view that foetal distress was managed well, as delivery was imminent and baby D was delivered within one hour of the diagnosis of foetal distress.

135. Dr Manthata-Cruywagen referred to an article authored by Janet Rennie and Lewis Roosenbloom titled '*Review How long have we got to get the baby out? A review of the effects of acute and profound intrapartum hypoxia and ischaemia*'¹⁹ in which the authors conclude that damage to a human baby begins to accrue after 10 minutes of an acute profound hypoxic ischaemic insult. It was put to Dr Manthata-Cruywagen that if it is accepted that damage can occur within 10 minutes of an insult, then all the more reason when discovering foetal distress *not* to wait for an hour to deliver. The midwife must do something to alleviate the foetal distress if delivery is not imminent, or if it is imminent, she must deliver immediately to avoid the insult from developing into an injury. In other words, the midwife must intervene immediately and cannot simply wait for an hour whilst waiting for delivery to happen naturally. Dr Manthata-Cruywagen remained resolute that since delivery occurred within the time frame permitted by the guidelines, therefore the period that elapsed was not improper.

136. During re-examination Dr Manthata-Cruywagen testified about the known causes of foetal distress, namely, uterine contractions (especially in the second stage of labour); placental dysfunction or compromise (which could be caused by infection, high blood pressure or exposure to 'amphetamine-like substances' such as Khat during pregnancy); cord prolapse; and sentinel events. With placental compromise, the blood vessels in the placenta become constricted. They are not open enough to take in oxygen from the mother to supply to the baby.

Dr Weinstein (Radiologist)

137. Dr Weinstein confirmed that he studied an MRI scan taken of Baby D's brain when he was 5 years and ten months old. He confirmed the anatomical areas

¹⁹ The article is to be found at S42 on caselines. Significantly, the authors recognize that '

that were damaged, namely the PGBT, being the metabolically active areas of the foetal brain. According to Dr Weinstein, the damage was consistent with an acute profound pattern of injury. These areas get damaged from a short episode (insult) whatever the episode may be. What damages the metabolically active areas of the foetal brain is, *inter alia*, a cut-off of blood supply to these areas. If blood supply is mildly cut off for a long time then the other areas of the brain will suffer damage. An acute profound injury is caused by a short, sudden, severe event, although the radiologist cannot tell from the MRI image when precisely it happened. The obstetricians have to determine how and when it occurred

138. In this case although the clinical records mention that baby D suffered septic arthritis some weeks after birth, there is no evidence that sepsis got to the brain.

139. In regard to the addendum joint minute he complied with Dr Alheit and wherein Dr Alheit expressed the view that it is inappropriate to use the conventional terminology 'acute profound' in the absence of a sentinel event, Dr Weinstein stated that 'acute profound' terminology has been used for the past 20 to 30 years and it has not been refuted that the metabolically active areas of the brain get damaged by a short, severe event (ranging between 15 to 35 minutes). If the baby was in a poor condition before the event, then it will take a shorter time for damage to occur.

140. The Smith et al article that suggests that the intrapartum BGT pattern injury and radiologically termed 'acute profound HI brain injury' are not necessarily synonymous goes against the convention of 20 to 30 years of understanding. In his view, the pattern is the same – one could call it a BGT pattern or an 'acute profound' pattern as long as it is understood that the injury arises from a sudden and severe insult that causes the auto-regulatory response 'to go off'

and not protect the metabolically active areas of the brain, so that they become damaged.

141. In support of his testimony that an acute profound pattern of injury means that the injury occurred suddenly and was severe, Dr Weinstein relied on an article that was published in the SA Journal of Radiology in October 2020, which was co-authored by esteemed radiologists, Misser SK, Barkovich AJ, Lotz JW and Archery M, titled "*A pictorial review of the pathophysiology and classification of the magnetic resonance imaging patterns of perinatal term hypoxic ischemic brain injury – What the radiologist needs to know...*"²⁰ Dr Weinstein went on to say that he agrees with the Misser article - the nature of the injury is such that it happens suddenly. Misser et al explain the pathophysiology of the acute profound ischemic injury and do not merely explain the pattern of injury seen on MRI images in relation to the anatomical features of the brain, which is what Dr Alheit contends for.
142. If the severe injury happens for longer than 10 minutes there will be damage to the central areas of the brain. If it takes longer than 15 to 25 minutes then the whole brain will be impacted and the pattern will be acute profound partial prolonged, which is called a mixed pattern. In the case of baby D, there was damage to the basal ganglia thalamic areas and the periolandic strip, as well as the vermis, so one could also call it a PBGT pattern (or by its shortened name, being a BGT pattern).

²⁰ The article appears at V24 of the papers. Under the heading 'Acute profound ischemia', the authors state the following: "*In the setting of acute cessation of perfusion with rapid progression (e.g. in abruptio placentae) there is insufficient time for the cerebral autoregulatory mechanisms to adequately redirect blood flow to the high metabolic areas of the brain...The failure to protect these areas will result in a primarily central injury pattern with selective neuronal necrosis. This pattern of injury...involves the deep basal nuclei.*"

The authors distinguish 'Partial prolonged ischemia' which is explained as follows: "*When there is mild or moderate hypoxia (e.g. in occult cord prolapse or placental insufficiency), there is sufficient time available for the cerebral auto-regulatory mechanisms to redirect blood flow to the high metabolic areas of the brain, many of which have a greater proportion of NMDA receptors. This results in sparing of these high metabolic areas at the expense of the watershed areas of the cerebral hemispheres between the major arterial territories, especially at the borders between perfused zones...*"

143. During cross-examination, Dr Weinstein maintained the stance that he was trained in conventional radiology to report things in a conventional way as 'acute profound' and that the BGT pattern of injury is synonymous with acute profound pattern of injury as both refer to the same areas in the brain that are damaged.
144. During cross-examination Dr Weinstein made the following important concessions:
- 144.1. When using terminology to describe a brain injury pattern observed on MRI imaging it is important that the appropriate clinical context accompany the use of such terminology;
 - 144.2. The BGT injury is described in the 2019 ACOG consensus statement as a '*cerebral – deep nuclear neuronal injury*' impacting the deep nuclear gray matter in the brain and is associated with *severe partial insult of prolonged duration or a combined partial with profound terminal insult*; however Dr Weinstein questioned 'how long is 'prolonged' in the BGT injury?' He reiterated that if the insult persists for longer than 40 to 50 minutes, then a mixed pattern would result. If the insult is longer than 10 minutes (allowing for up to 20 to 40 minutes), a BGT pattern would result.
 - 144.3. Dr Weinstein agreed that ACOG 2019 distinguishes the BGT injury from cases where there is a complete shut-down of blood supply, which ACOG describes as a '*deep nuclear gray – brain stem*' which is associated with *acute total hypoxic ischemic insult*. In this case, baby D's brainstem was not damaged.
145. Dr Weinstein was referred to an article titled '*Neuroimaging in the term newborn with neonatal encephalopathy*' that appeared in the publication 'Seminars in Fetal and Neonatal Medicine' and which was published online on

29 October 2021,²¹ in which article the authors identify three pathophysiology's that may cause the BGT pattern of injury to the BGT and periolandic cortex, namely, (i) sentinel events; or (ii) severe partial asphyxia with prolonged duration; or (iii) combination of partial and near-total asphyxia. For ease of reference, I will refer to this article as 'Barkovich et al'. It was thus put to Dr Weinstein that the description 'acute profound' is no longer used in relation to the BGT injury and that the BGT injury is also not only caused by a short severe event (such as a sentinel event) as Dr Weinstein testified in-chief, but can also be caused by a severe partial insult of prolonged duration. Dr Weinstein responded that he uses the terminology 'acute profound' in its classic sense to indicate where the damage in the brain occurred (PGBT) and one can call it BGT or 'acute profound' because the pathophysiology is the same.

146. Dr Weinstein was referred to an article authored by Volpe titled '*Hypoxic Ischemic Injury in the Term Infant: Pathophysiology*' where Volpe discusses a deep nuclear brainstem injury caused by total asphyxia.²² According to Volpe, total asphyxia will take approximately between 10 to 15 minutes to result in brain injury in a human foetus. In other words, a human foetus that is subjected to total asphyxia, for example, with cord prolapse or uterine rupture [i.e., (a sentinel event)] will suffer brain damage within approximately 10 to 15 minutes. Volpe also points out that in one carefully documented human study, this neuropathology was identified by neuroimaging *after such sentinel events occurring* generally from 10 to 46 minutes before delivery. Dr Weinstein did not disagree with what is documented by Volpe. On the same page, Volpe records that '*in the 80% or 90% of cases with hypoxic ischemic disease in which*

²¹ The article was co-authored by world renowned radiologists such as Wisnowski JL and Barkovich AJ and others together with medical experts in the fields of paediatric neurology, and neonatology. Barkovich AJ is the same person who co-authored the Misser article which is cited in para 141 of the judgment.

²² Volpe ____ chapter 19 at p503.

an overt fetal sentinel event is not present, the uncertainty in timing is often measured in hours and not minutes.' Dr Weinstein was asked to comment thereon. He replied stating that as Volpe was dealing with obstetrics, therefore he could not comment thereon.

Discussion

147. Upon a consideration of the full conspectus of evidence, the following indisputable and irrefutable facts were established in evidence:

147.1. Foetal heart rate monitoring during the plaintiff's first phase of the active stage of labour was not carried out with the required frequency (every half hour) or in the required manner by auscultation before, during and after contractions, as stipulated in the maternity guidelines and without auscultation in relation to contractions, foetal well-being could not be established. The defendant conceded that the failure to properly monitor the FHR amounted to sub-standard care;

147.2. During the second stage of active labour until baby D's delivery, the FHR was not monitored at all and the foetal distress diagnosed at 7 am was not arrested by intervention in terms of the guidelines pertaining to foetal distress;

147.3. As from 7 am at which time when the plaintiff's cervix was fully dilated and the second stage of active labour commenced, the FHR was required to be monitored even more frequently, that is, after every second contraction, as this is the period during which an upsurge in foetal hypoxia would be expected during contractions, which increase in frequency and strength;

147.4. The partogram that was utilised only from 0h00 to 0h04 during the first stage of active labour was not completed correctly nor did it record the required information in that the nurses failed, amongst others, to plot the rate of descent of baby D's head and failed to plot

plaintiff's contractions properly, more specifically, the variability of accelerations and decelerations in relation to the contractions;

147.5. The midwives who monitored the plaintiff's labour did not keep proper records and by the time the staff took over the care of the plaintiff at 7am, they would have been in the dark as to the true condition of baby D or the extent or duration of the foetal distress diagnosed at 07h00, given that no partogram had been utilised after 04h00 and that which had been used was incomplete, and given that there was no record of auscultation of the FHR before, during and after contractions;

147.6. The care that the plaintiff (and her unborn child) received from the midwives at Hillbrow clinic during the plaintiff's active stage of labour until delivery of baby D amounted to sub-standard care in so far as the conduct of the midwives failed to comply with the maternity guidelines in the various respects as highlighted in the testimony of Prof Anthony and other experts;

147.7. At 7am, the attending midwife had not factual basis to conclude that delivery was imminent given that the descent of the baby's head was unknown.

148. The obstetricians including the midwives who testified all agreed that the purpose of monitoring of the FHR is to detect foetal distress so that immediate intervention can take place to prevent injury or further injury to the foetus. Stated differently, if the FHR is not monitored properly and with the required frequency, any abnormalities in FHR would not be detected and if abnormalities go undetected, the nurses would not be able to respond adequately or immediately to arrest foetal distress.

149. It was common cause between the obstetricians and the paediatric neurologists and radiologists who testified that the insult that led to Baby D's

irreversible brain damage occurred intrapartum, i.e., before birth, the cause of which was hypoxia/ischemia (where the baby is deprived of sufficient oxygen). Prof Bolton was a lone voice in postulating that the brain damage may have been caused by sepsis that developed 20 days after baby D's birth, however, such postulation was conceded by him to be unsupportable by any proven facts. In any event, such theory is irreconcilable with the diagnosis of hypoxic ischaemic encephalopathy grade II some two days after birth, which was based on established facts such as baby D's abnormal tone, the fact that he did not cry, that he experienced respiratory distress at birth requiring resuscitation, which respiratory distress was sustained despite continued resuscitation, the fact that baby D was lethargic, could not suckle and also experienced seizures after birth.²³

150. In *AN v MEC for Health, Eastern Cape*,²⁴ the court explained that a sudden, total, persistent interruption to the blood supply [and hence oxygen supply] in the prepartum period is usually caused by a perinatal sentinel event such as placental abruption, uterine rupture, umbilical cord prolapse, shoulder dystocia, maternal collapse or compression of the cord (which totally interrupts blood supply for a period long enough to cause damage) (a total, persistent interruption). Each of these (with the exception of a cord compression) can be verified afterwards because they leave a footprint.²⁵
151. In the present case, the evidence did not establish that any such markers were present at birth to denote that any sentinel event had occurred. There was

²³ Acog's definition of neonatal encephalopathy is articulated in para 81 in the judgment. What baby D experienced in the earliest days of life, fits in with Acog's definition.

²⁴ *AN v MEC for Health, Eastern Cape* (585/2018) [2019] ZASCA 102 (15 August 2019), para [16]. In that case the court explained that 'acute' means 'sudden' as opposed to developing over time. 'Ischaemia' is a restriction in blood/oxygen supply. Blood supplies oxygen to the brain. A restriction in blood supply leads to a lack of oxygen supply. 'Hypoxia' results from sustained reduction in the supply of oxygen to the brain. This is a form of neurological dysfunction.

²⁵ This is consistent with Prof Anthony's testimony.

also no evidence that cord compression had likely occurred. Likewise, there was no evidence of congenital, metabolic, infectious or genetic causes for baby D's brain injury.

152. It should be noted at the outset that the defendant denied that the sub-standard care provided to the plaintiff in the management of her labour until delivery of baby D (evidenced, *inter alia*, by a lack of proper monitoring of the FHR throughout the active phase of labour until delivery; the inability to timeously detect the onset of foetal distress through the failure to monitor the FHR correctly or at all at the required intervals; the midwife's inappropriate response to diagnosed foetal distress when detected; her inappropriate response to the meconium stained liquor observed at 7 am; her failure to monitor the FHR at all between the period 7am to delivery; her failure to intervene appropriately once foetal distress was detected; her failure to suction meconium from Baby D's mouth before his trunk was delivered) amounted to negligence on the part of the relevant midwives in all the attendant circumstances.

153. The defendant disputed causation – the contention being that there was no causal connection between the negligence alleged and the cerebral palsy suffered by Baby D. This was premised on the addendum reports of Dr Manthata-Cruywagen and Professor Bolton and the report of Dr Weinstein. These experts raised the following two issues to deny causation:

- (i) That the plaintiff used the substance called Khat (also called Mira) during her pregnancy which had an adverse effect on her placenta and caused placental insufficiency, thereby causing the foetus to suffer a hypoxic ischaemic injury in the last 30 minutes of labour prior to delivery; and
- (ii) The injury pattern identified on the MRI scan was of an acute profound hypoxic ischaemic injury which suggests that the insult which caused

the child to sustain brain damage occurred suddenly (acutely) and was of a severe nature (profound) as is denoted by the term 'acute profound'.

154. In *A M and Another v MEC for Health, Western Cape*,²⁶ the Supreme Court of Appeal had occasion to reiterate the role of experts and how their evidence is to be approached. Wallis JA put it thus:

[17] ...The functions of an expert witness are threefold. First, where they have themselves observed relevant facts that evidence will be evidence of fact and admissible as such. Second, they provide the court with abstract or general knowledge concerning their discipline that is necessary to enable the court to understand the issues arising in the litigation. This includes evidence of the current state of knowledge and generally accepted practice in the field in question. Although such evidence can only be given by an expert qualified in the relevant field, it remains, at the end of the day, essentially evidence of fact on which the court will have to make factual findings. It is necessary to enable the court to assess the validity of opinions that they express. Third, they give evidence concerning their own inferences and opinions on the issues in the case and the grounds for drawing those inferences and expressing those conclusions.

[20] The need for clarity as to the facts on which an expert's opinion is based has been stressed in a number of cases. In *PriceWaterhouseCoopers v National Potato Co-operative Ltd* the following passage from a Canadian judgment was cited with approval:

'[326] "Before any weight can be given to an expert's opinion, the facts upon which the opinion is based must be found to exist"

[327] "As long as there is some admissible evidence on which the expert's testimony is based it cannot be ignored; but it follows that the more an expert relies on facts not in evidence, the weight given to his opinion will diminish".

[328] An opinion based on facts not in evidence has no value for the Court.'

[21] The opinions of expert witnesses involve the drawing of inferences from facts. The inferences must be reasonably capable of being drawn from those facts. If they are tenuous, or far-fetched, they cannot form the foundation for the court to make any finding of fact.

²⁶ *A M and Another v MEC for Health, Western Cape* (1258/2018) [2020] ZASCA 89 (31 July 2020) at paras 17 and 19-21.

Furthermore, in any process of reasoning the drawing of inferences from the facts must be based on admitted or proven facts and not matters of speculation. As Lord Wright said in his speech in *Caswell v Powell Duffryn Associated Collieries Ltd*:

‘Inference must be carefully distinguished from conjecture or speculation. There can be no inference unless there are objective facts from which to infer the other facts which it is sought to establish ... But if there are no positive proved facts from which the inference can be made, the method of inference fails and what is left is mere speculation or conjecture.’

(emphasis added) (footnotes omitted)

155. In regard to the factual evidence presented at trial, being that of the plaintiff and the two midwives, I bear in mind the principles that I am to apply in the face of conflicting evidence as set out in *Stellenbosch Farmers' Winery Group Ltd v Martell et Cie*, [2003 (1) SA 11 (SCA)] at paras 14–15

156. In written argument presented on behalf of the defendant, it was submitted that the plaintiff was not a truthful or credible witness due to certain external contradictions between her testimony concerning what the nurses did or did not do when monitoring the plaintiff and her unborn baby at 0h00, 04h00; and 06h00, and that which was in fact done, as reflected in the labour records, including an internal contradiction concerning the language barrier that impeded communication between the plaintiff and the midwives. As I understand the argument, on the one hand, the plaintiff's testimony was that she was not conversant in the English language and that she used a person to help her when she was asking for help, whilst on the other hand, she testified that she told the nurse that she was feeling pain and that the nurse told her that she must try to push so that the baby can come out. The defendant's submission was that *‘based on the contradictions highlighted above, the plaintiff was not a truthful and credible witness as she had serious language barriers and could therefore not have comprehended what the nurses were*

telling her. This appears from the evidence of Sister Moqhae that the plaintiff did not follow instructions. The plaintiff's evidence regarding the events of the 08th to 9th July 2012 is therefore not probable, The only reliable evidence regarding the events of the said dates is that of the midwives, Moqhae and Mbanga.'

157. I cannot, with respect, agree with the defendant's submission that the plaintiff was not a truthful or credible witness or that sister Moqhae was a reliable witness regarding the events. The plaintiff's evidence was that she was in abject pain from the time of her admission to Hillbrow labour ward on 8 July 2012. There were times when she cried out for help when the pain was really bad, but the nurses did not heed her calls. As a first time mother who had never before endured labour pains, one would expect that the plaintiff may have needed comfort and reassurance at times when the pain became unbearable. In my view, it cannot be said that the plaintiff was not honestly mistaken as to how she and baby D were assessed prior to 7 am on 9 July 2012. Stated differently, I cannot find that the plaintiff was deliberately lying about the conduct of the midwives, whether before or after 7 am. In other words, being honestly mistaken does not necessarily translate to willfully lying or being deliberately dishonest. My impression of the plaintiff was that she did not seek to embellish her evidence, nor did she seek to exaggerate how the midwives treated her until the delivery of Baby D. On her usage of Khat prior to falling pregnant, her evidence was consistent – she did not partake in it regularly, but only occasionally. She had last used it one or two months before falling pregnant. The plaintiff remained steadfast, despite vigorous questioning, about the fact that she did not use Khat at all during her pregnancy, which pregnancy, as was not refuted in evidence, had progressed normally and without complications.

158. Sister Moqhae denied having sworn or shouted at the plaintiff after she took over the management of the plaintiff's labour when commencing her duties at 7 am on 9 July 2012. As she had only commenced duties at 7 am on that date, she was understandably not able to comment on how the night shift midwife may have treated the plaintiff. In contradistinction to the plaintiff's evidence, Sr Moqhae's evidence was in my view tailored to exculpate herself from the consequences of failing to adhere to the maternity guidelines, not only as regards her failure to properly intervene in the face of foetal distress or to manage or react to the presence of meconium stained liquor, but also as regards the failure by the night shift midwife to monitor the FHR at the correct frequency and in the correct manner and her own failure to monitor the FHR at all after examining the plaintiff at 7am until delivery.

159. It is worth repeating what the maternity guidelines require when foetal distress and the presence of meconium is detected. The following intervention for foetal distress is mandated:

Explain the problem to the mother;

Lie the mother in a left lateral position;

Give oxygen by face mask at 6 L/minute; (this was not done)

Start an intravenous infusion of Ringer-Lactate to run at 240 mL/hour;

Do a vaginal examination for cervical dilation and to exclude cord prolapse.

If vaginal delivery is imminent (cervix fully dilated), **deliver immediately**, by vacuum extraction if necessary.

If vaginal delivery is not imminent, give hexoprenaline 10 micrograms IV and prepare for immediate caesarean section. **Arrange urgent transfer from a community health centre to hospital.** (emphasis added)

Where thick meconium is present the following steps must be taken:

Transfer the patient from a community health centre to hospital unless delivery is imminent;

Monitor the foetus with a cardiotocograph (CTG) if available;

When the head extends at delivery, **thoroughly suction the infant's mouth and then nose before delivering the trunk.** (emphasis added)

160. The evidence irrefutably established, which Sr Moqhae was constrained to concede, that:

160.1. She did not explain the problem of foetal distress to the plaintiff. She sought to excuse doing so by either blaming the plaintiff for not listening and/or because the plaintiff was restless and screaming and/or an inability to do so because of the language barrier that impeded communication and the lack of anyone present to interpret;

160.2. She did not give the plaintiff oxygen by face mask at 6 L/minute. Her excuse was that delivery was imminent;

160.3. She did not start an intravenous infusion of Ringer-Lactate to run at 240 mL/hour. Her excuse was that delivery was imminent;

160.4. She did not give the plaintiff a drug (hexoprenaline 10 micrograms IV) to slow down contractions. Again, her excuse was that delivery was imminent and in any event, once the cervix is 10 cm dilated, it is not viable to slow down contractions;

160.5. She did not even consider the need for transfer to a hospital, let alone attempt to arrange an urgent transfer from the Hillbrow clinic to the hospital, despite the undisputed and unrefuted evidence that Charlotte Maxeke was no further than a 10 minute drive away. Here the excuse was that none of this could have been achieved in the space of one hour, being the time period that elapsed before baby D was delivered;

160.6. When baby D's head extended at delivery, she did not thoroughly suction his mouth and then his nose before delivering the trunk. Her excuse was that it was not possible to do so because the plaintiff kept

having contractions when the baby's head came out – a proposition I deal with below;

160.7. As regards Sr Moqhae lying the plaintiff in the left lateral position, the probabilities support a conclusion that Sr Moqhae did *not* do this whether for purposes of managing the foetal distress or at all, firstly, because Sr Moqhae considered that delivery was imminent and that is precisely why the only thing she did was to encourage the plaintiff to push in an attempt to get baby D's head to descend in order to get the baby out. Secondly, because her evidence fluctuated between the plaintiff just lying on her side prior to bearing down at 7h30 am, to the plaintiff herself changing sides so that she was not always in a supine position. Thirdly, because the undisputed evidence of Prof Anthony was that the mother is usually in a supine position when bearing down. Fourthly, the Plaintiff's evidence was that she lay in the supine position and that she began pushing after being told to do so. Her undisputed evidence was and that Sr Moqhae remained positioned in front of her legs whilst encouraging her to push for purposes of delivering baby D.

161. There were several unsatisfactory aspects to Sister Moghae's evidence. I need only mention a few. One was that she considered that the guidelines only require full dilation (10cm) for delivery to be considered imminent, whereas, none of the expert witnesses supported such interpretation because it is known that delivery is only imminent when the cervix is 10 cm dilated *and* the baby's head extends to the perineum, albeit that Dr Manthata-Cruywagen testified that delivery is imminent as soon as the mother has the urge to push and the urge to push only occurs when the baby's head has extended to the pelvic brim. I agree with Prof Anthony that Sr Moqhae could not have known

at 7am how long it would take for the head to descend, given that the guidelines allow for up to two hours for the head to descend.

162. Another unsatisfactory aspect is that she claimed that because delivery was imminent, she tried to deliver the baby *immediately*, which was belied by the fact that she had spent an hour doing nothing other than encouraging the plaintiff to bear down (push) before baby D was delivered by way of unassisted natural vaginal birth instead of intervening immediately to alleviate baby D's distress and then expediting delivery by way of assisted birth (be it by means of episiotomy or the use of forceps or preparing the plaintiff for a c-section and arranging transfer to a hospital or by vacuum suction).
163. A further unsatisfactory aspect is that whilst Sr Moqhae agreed that the purpose of auscultation of the FHR is to determine if there are late or sustained decelerations in the foetal heart beat after contractions in order to detect foetal distress and that the only way to do this is by measuring the FHR before, during and after contractions, she sought to justify the failure of the night shift midwife to do so (i) because at Hillbrow clinic, they use a foetoscope notwithstanding that, according to Sr Moqhae, one cannot determine decelerations with a foetoscope as a foetoscope can only tell one if there is tachycardia or bradycardia and (ii) the evidence was that Hillbrow clinic is equipped with a stethoscope which does permit auscultation of the FHR, which leads me to the inescapable and necessary inference that the midwives did not make use of such instrument by their own choosing. Later during cross-examination Sr Moqhae sought to mould her evidence, ostensibly in an attempt to justify her own failure to auscultate the FHR at 7 am or thereafter, by stating that the plaintiff would not allow her to put the foetoscope on her (plaintiff's) abdomen and that during contractions the plaintiff would change positions. She again sought to mould her evidence after being asked during cross-examination why the staff including Sr Moqhae, having realized that

communication with the plaintiff was problematic, failed to take any steps to arrange for the plaintiff's husband to be present so that he could assist in interpreting what was being said. Her answer was that the situation (leading up to delivery) was chaotic, and they were trying to save baby D. It was then put to Sr Moqhae that the situation was becoming increasingly chaotic because she was not taking the correct steps. She then stated, in conflict with her evidence-in-chief, that she was encouraging the plaintiff to push only during contractions and that she listened to the FHR whenever the plaintiff allowed her to, ostensibly to portend that she knew what the condition of baby D was.

164. Yet a further unsatisfactory aspect is in relation to the astonishing explanation Sr Moqhae provided for not following the guidelines in relation to the management of meconium, amongst others, to suction the baby's mouth and nose *before* the trunk is delivered. Her explanation was to the effect that the plaintiff was restless as she was pushing and they could not understand each other and that is why she merely wiped baby D's face because she could only put a suction into his mouth if the plaintiff stopped pushing and Sr Moqhae could not get the plaintiff to stop pushing.

165. Having regard to all the issues raised above, I am not persuaded that the quality, integrity and independence of Sr Moqhae's recall of the events (more specifically, in regard to how she managed the maternal and foetal condition between 7 am and 8am) is such that Sr Moqhae's version of events can be considered reliable or credible in so far as it is in conflict with the plaintiff's evidence of the events preceding delivery or in so far as it is in conflict with Prof Anthony's evidence²⁷ discussed below. The plaintiff testified that that Sr

²⁷ Conflicts pertained to, amongst others, whether delivery was imminent or not at 7 am; whether encouraging the plaintiff to push was inappropriate in circumstances where overt signs of foetal distress required immediate intervention as prescribed by the maternity guidelines; that *inadequate* monitoring of the foetal condition probably led to abnormalities in the FHR being undetected prior to 7 am; that the approach whereby the plaintiff was encouraged to push even though there was no basis to conclude

Moqhae injected something into her thigh after which she experienced intensified contraction pain and very shortly thereafter Baby D was born. What the substance was that was injected was not identified in evidence and therefore I need not consider it further.

166. Before dealing with Prof Anthony's evidence, it convenient to first deal with Dr Manthata-Cruywagen's evidence. In relation to her conclusion in her addendum report that the brain injury that baby D sustained was attributable to placental insufficiency or placental abnormality caused by the plaintiff's use of khat during pregnancy, suffice it to say that her opinion was based on an assumption of placental pathology in the absence of any factual evidence of placental pathology. In her testimony she confirmed having relied on a cryptic note that went no further than to raise a query as to whether the plaintiff had chewed khat occasionally. Dr Manthata-Cruywagen testified that she interpreted the note to mean that the plaintiff admitted to using khat during her pregnancy, an interpretation that was not at all discernable or sustainable from text of the query itself and which was entirely lacking in factual foundation. The plaintiff's evidence, namely, that she did *not* chew Khat during her pregnancy, remained unrefuted. In any event, the doctor who completed the note was not called to testify on behalf of the defendant and as such the note constituted inadmissible hearsay. In short, since the inference that Dr Manthata-Cruywagen sought to draw from the cryptic note lacked factual foundation, as such amounts to no more than inadmissible supposition or conjecture founded upon illogical or irrational reasoning that is speculative in the extreme, and as such, her opinion falls to be rejected in relation to the use of khat as a causative basis for the foetal hypoxia sustained by baby D. Dr Manthata-Cruywagen's propensity to couch her opinions as statements of fact

that delivery was imminent, served to exacerbate baby D's existing hypoxia to the point where he lacked the ability to compensate for a lack of oxygenation during the Plaintiff's increased contractions in the second stage of labour.

but which are based entirely on speculation, is perhaps more aptly illustrated by her most shocking evidence to the effect that the plaintiff probably chewed khat *during* labour, which the nurses probably just did not see.

167. The rejection of Dr Manthata-Cruywagen's opinion has the result that Prof Anthony's evidence to the effect that the hypoxia that led to the brain injury suffered by baby D was, on the available evidence and in the absence of a obstetric sentinel event, most likely caused by (i) the failure of the night midwife to detect the onset of foetal hypoxia due to the lack of monitoring of the FHR at the correct intervals and in the correct manner and which in turn led to an inability to be in a position to intervene immediately during the first stage of active labour and (ii) the concomitant failure of Sr Moqhae to implement the correct steps prescribed by the guidelines to arrest foetal distress in the second stage of labour, which remained unrefuted in evidence.
168. The plaintiff argued that Dr Manthata-Cruywagen was a biased witness in favour of the defendant, in that she lacked objectivity and went to great lengths to create a baseless defence for the defendant which could not be substantiated. Regrettably, I am inclined to agree. Her revised opinion lacked evidential foundation and was not defensible on the proven facts. Moreover, she based her entire opinion that the injury sustained by baby D occurred in the last thirty minutes of labour on the postulation that the injury resulted from an acute profound event, i.e., a short sudden or unpredictable event premised on placental abnormality, which, irrespective of inadequate monitoring on the part of the midwives, resulted in a total shutdown of blood flow and hence oxygen to the foetus only in the last 30 minutes before birth so that there was nothing that the midwives could have done to prevent the

adverse outcome which ensued. As was pointed out in Lord Justice Brooks in *Ratcliffe*:²⁸

"...the judge should not be diverted away from the inference of negligence dictated by the plaintiff's evidence by mere theoretical possibilities of how that outcome might have occurred without negligence: the defendants' hypothesis must have the ring of plausibility about it."

169. In my view, Dr Manthata-Cruywagen's hypotheses, not being founded on proven facts, lack plausibility. It should be noted that the plaintiff's placenta was not tested after birth and therefore there is no medical evidence to confirm the supposed placental insufficiency. Moreover, the objective evidence established that baby D was not growth restricted as would have eventuated if there had been placental insufficiency.
170. Prof Anthony's theory, on the other hand, was supported by proven facts and was based on logical reasoning. Firstly, Dr Mogashoa, Dr Kara and even Sr Moqhae agreed with Dr Anthony that foetal distress was likely present *before* 7 am when it was first detected, as proper monitoring of the FHR had not taken place prior thereto so that an abnormal foetal heart rate may well have arisen prior to 7am which went undetected. There was consensus amongst the experts, including the midwives, that the presence of meconium is a sure sign of foetal distress. It seems to me to be logical that meconium would have developed or existed *before* it manifested. Since meconium was *already* present at 7 am, it is probable that it arose before 7am but went undetected precisely because the 6h30 am assessment of the foetal condition was not performed. Even Dr Manthata-Cruywagen was constrained to conclude that it could not be said that the baby D was well before 7 am, given that monitoring was missed at 6h30 am.

²⁸ *Ratcliffe v Plymouth and Torbay Health Authority* (1998) EWCA Civ 2000.

171. Prof Anthony's theory regarding a BGT injury occurring in the absence of a sentinel event through evolving hypoxia over a longer period is, as was pointed out in evidence, not a novel notion. It finds support in the 2019 Acog consensus statement, and in Volpe,²⁹ both of which were published prior to publication of the the Smith et al article, as well as the Barkovich et al review, which unequivocally endorses it. In the Barkovich et al review, which I referred to earlier in the judgement, the authors state concerning the central/BGT injury pattern: *"Clinically, the central/BGT pattern is commonly observed following perinatal sentinel events and moderate to severe relatively prolonged insult, while experimental studies in primates suggest that this pattern results from a combination of anoxia (e.g., near-total asphyxia) and hypoxia (e.g., partial asphyxia) which may be incurred during a single event or across serial events."*

172. The theory propounded by Prof Anthony and his co-authors in the Smith et al article (on which he based his evidence), has also been endorsed by the Supreme Court of Appeal in *MEC for Health, Limpopo v L W M obo D M* where the following was said:³⁰

"Furthermore, a lack of general acceptance of his theory cannot, without more, warrant a rejection of his theory, as it is backed up by a case-study. Clearly, there is no basis in law for rejecting Prof Smith's theory. The 10 cases on their own demonstrate that a series of partial intermittent, subacute/subthreshold hypoxic insults can cause an injury to the BGT deep nuclear structures including the periorlandic area with a pattern like that revealed by D M's MRI scan. Moreover, Prof Smith's conclusions were not based exclusively on animal experiments. It was also based on his experience and that of his co-authors over many years, involving human cases. His views find material support in Volpe's textbook, where the following is stated in relation to the injuries arising from an insult to the deep nuclear-brain stem, in which the insult is severe and abrupt:

²⁹ Volpe, in his work titled 'Neurology of the Newborn' 5th ed, chapter 19 at page :

³⁰ *MEC for Health, Limpopo v L W M obo D M* (502/2021) [2022] ZASCA 146 (27 October 2022) at para 36.

'In the more prolonged and less severe insults, the diversion of blood to deep nuclear structures occurs at least to a degree, and thus the cerebral regions are more likely to be affected. Studies in the near-term fetal lamb indicate that the severe terminal insult that results in injury to deep nuclear structures especially may be likely to occur after brief, repeated hypoxic-ischaemic insults *first* cause a cumulative deleterious effect on cardiovascular function that presumably *then* can result in a severe late insult.' "

173. The plaintiff argued that 'the dogmatic and sometimes frighteningly illogical evidence given by Dr Weinstein, coupled with his combative or argumentative, evasive and condescending demeanour is clear proof of his bias in favour of the defendant. The submission is not entirely without substance. Dr Weinstein certainly behaved in an eccentric manner in the witness box. He was also highly argumentative and emotive, assuming the role of advocate at times.³¹ My impression was that he was determined not to concede the cogency of scientific review of research that has been published by a host of international medical professionals in which they support the finding that in some instances (e.g., in the absence of a sentinel event) a BGT pattern injury can result from hypoxia which develops over time during the labour process (which is preventable by early detection of the hypoxic insult before it leads to injury) as opposed to only resulting from sudden (acute) or severe and total (profound) event such as a sentinel event, which is unexpected and unpredictable, occurring during a short window period before birth, and which is thus unpreventable.³² All being said, Dr Weinstein's evidence did nothing to refute Dr Alheit's evidence, which was both logical and defensible on the proven facts, namely, that because there was no evidence that a sentinel event had occurred in this case, the use of historical nomenclature such as

³¹ In *Schneider v Aspelting* [2010] 3 ALL SA 332 (WCC) the court cautioned that an expert should not assume the role of an advocate, nor give evidence which goes beyond the logic which is dictated by the scientific knowledge which the expert possesses. The expert is expected to provide the court with an objective and unbiased opinion, based on his or her expertise.

³² Put differently, there is now substantive expert opinion that indicates that a rapid (sudden) total shut down of oxygenated blood (i.e., the mechanism of an acute profound Hypoxic Ischaemic injury) is *not* the only mechanism by which a BGT pattern injury can occur.

'acute profound' (which connotes a sudden, unpredictable, severe and total event that leads to fetal neurological injury) was inappropriate.

174. Not much needs to be said about Prof Bolton's theories regarding the causative basis of baby D's brain injury and cerebral palsy. Suffice it to say that the foundation on which his theories were based, dissipated like mist before a the sun during cross-examination. Prof Bolotn was however a honest witness who made the necessary concessions during cross-examination. In so far as Dr Kara's evidence supported Prof Anthony's theory as to the cause of baby D's injury, his evidence was logical, rational and defensible on the proven facts.

175. This brings me to the question of whether the sub-standard care the plaintiff and her unborn child received at the hands of the staff at Hillbrow clinic amounted to negligence.

176. The proper approach for establishing the existence or otherwise of negligence was formulated by Holmes JA in *Kruger v Coetzee* 1966 (2) SA 428 (A) at 430 E-G where the following was said:

"For the purposes of liability culpa arises if—

(a) a diligens paterfamilias in the position of the defendant—

(i) would foresee the reasonable possibility of his conduct injuring another in his person or property and causing him patrimonial loss; and

(ii) would take reasonable steps to guard against such occurrence; and

(b) the defendant failed to take such steps.

...Whether a diligens paterfamilias in the position of the person concerned would take any guarding steps at all and, if so, what steps would be reasonable, must always depend upon the particular circumstances of each case. No hard and fast basis can be laid down."³³

³³ See too: *Sea Harvest Corporation (Pty) Ltd and Another v Duncan Dock Cold Storage (Pty) Ltd and Another* [1999] ZASCA 87; 2000 (1) SA 827 at [19], where the following was said:

"It should not be overlooked that in the ultimate analysis the true criterion for determining negligence is whether in the particular circumstances the conduct complained of falls short of the standard of the reasonable person. Dividing the inquiry into various stages, however useful, is no more than an aid or

177. It was common cause in this case that the purpose of foetal monitoring in labour is to detect hypoxia and to prevent asphyxia. In my view, the failure by the midwives who were tasked for caring for baby D ought reasonably to have foreseen that (i) failure to properly monitor by auscultation of the FHR in relation to the plaintiff's contractions at the correct intervals during the first and second stages of active labour would result in foetal distress being undetected and thus not being managed in terms of the required treatment protocols and (ii) failure to respond to diagnosed foetal distress by intervening to first arrest such distress in accordance with the steps required by the maternity guidelines and if distress persists, to expedite delivery, amounted to negligence in the circumstances of this case. On the established facts, it was entirely inappropriate for the midwife to encourage the plaintiff to bear down without first arresting the foetal distress, as it remained unrefuted and undisputed that bearing down efforts served only to deprive baby D of oxygen when he needed it the most. The injury that befell baby D was in my view both reasonably foreseeable and preventable by correct monitoring designed to detect foetal distress and correct intervention to alleviate such distress before an insult leads to injury, neither of which steps were taken to guard against the injury occurring.

178. The next question is whether the failure to adhere to the necessary treatment protocols caused the HIE and resultant cerebral palsy sustained by baby D.

guideline for resolving this issue... It is probably so that there can be no universally applicable formula which will prove to be appropriate in every case... [I]t has been recognised that while the precise or exact manner in which the harm occurs need not be foreseeable, the general manner of its occurrence must indeed be reasonably foreseeable."

And

Pitzer v Eskom [2012] ZASCA 44; JOL [2012] 29007 (SCA) at [24] where the court stated:

"What is or is not reasonably foreseeable in any particular case is a fact bound enquiry...Where questions that fall to be answered are fact bound there is seldom any assistance to be had from other cases that do not share all the same facts."

179. Prof Anthony's evidence that baby D's injury was, on the available evidence, likely caused by intermittent (undetected) episodes of sub-threshold hypoxia so that baby D entered the second stage of active labour already hypoxic, which distress was not alleviated at all by appropriate intervention but was exacerbated by allowing the labour to progress naturally with the baby being exposed to further hypoxia during more regular contractions and by encouraging the mother to bear down so that even further deprivation of oxygen occurred. In this context, there was already evidence of declining foetal oxygenation which, through lack of appropriate intervention, became acutely aggravated by intense second-stage uterine contractions so that the baby eventually lacked compensatory mechanisms to prevent injury.
180. There can be no doubt that further exposure to a deprivation of oxygen at a critical time during which baby D needed it the most and which could have been provided in accordance with a basic treatment protocol designed to alleviate foetal distress, fell dismally short of the standard of care that was required to be provided to baby D by the attending staff who were responsible for monitoring his condition.
181. In *AN v Mec for Health, Eastern Cape*, supra, the test for causation was stated as follows:
"The test for factual causation is whether the act of omission of the defendant has been proved to have caused or materially contributed to the harm suffered. Where the defendant has negligently breached a legal duty and the plaintiff has suffered harm, it must still be proved that the breach is what caused the harm suffered."
182. In *Minister of Safety and Security v Van Duivenboden* 2002 (6) SA 431 (SCA) at [25], the court observed as follows:
"A plaintiff is not required to establish the causal link with certainty but only to establish that the wrongful conduct was probably a cause of the loss, which calls for a sensible

retrospective analysis of what would probably have occurred, based upon the evidence and what can be expected to occur in the ordinary course of human affairs rather than an exercise in metaphysics.”

183. In *Minister of Finance and Others v Gore NO 2007 (1) SA 111(SCA)* at [33] the SCA held that:

“Application of the ‘but-for’ test is not based on mathematics, pure science or philosophy. It is a matter of common sense, based on the practical way in which the ordinary person’s mind works against the background of everyday life experiences.”

184. In my view, on a conspectus of the expert testimony, the plaintiff has established that it was the deprivation of oxygen during the labour process (which hypoxia, on the probabilities, went undetected during the first stage of active labour as a result of a failure to properly monitor the foetal condition at the required intervals) and which was acutely aggravated by intensifying hypoxia related to intense second-stage uterine contractions (which the evidence established, was allowed to increase or intensify by the failure of the attending midwife intervene appropriately for purposes of restoring oxygenation to the foetus until such time as delivery could be expedited by assisted delivery or the plaintiff could be transferred to a hospital for higher care) which caused baby D to sustain the HIE injury which ultimately resulted in the cerebral palsy from which he suffers.

185. I am persuaded that the plaintiff has established that the defendant is 100% liable for any damages sustained by her as may be proven or agreed to as a result of the negligence of the defendant’s employees which led directly to the resultant injury to baby D. It is not in dispute that the defendant is vicariously liable for their conduct.

186. The general rule is that costs follow the result. I see no reason to depart therefrom.

187. In the circumstances, the following order is granted:

ORDER

187.1. The defendant is liable for any damages that are proved or agreed to be due to the plaintiff in her capacity as parent and natural guardian of DM.

187.2. The plaintiff's costs in respect of the determination of the issue of liability are to be borne by the defendant.

A. MAIER-FRAWLEY

JUDGE OF THE HIGH COURT

GAUTENG DIVISION OF THE HIGH COURT, JOHANNESBURG

This judgement was prepared and authored by the Judge whose name is reflected and is handed down electronically by circulation to the Parties/their legal representatives by email and by uploading it to the electronic file of this matter on CaseLines. The date for hand-down is deemed to be 14h00 on 11 May 2023.

Dates of virtual hearing:	24 January 2022 to 11 February 2022; & 18 July 2022 to 22 July 2022 & 4 August 2022.
Judgment delivered:	28 September 2021 11 May 2023

APPEARANCES:

Counsel for Applicant:	Mr BD Molojwa
Attorneys for Applicant:	Jerry Nkeli & Associates Inc

Counsel for respondent:	Mr DJ Joubert SC
Attorney for Respondent:	State Attorney (Johannesburg)

the resultant injury to baby D. It is not in dispute that the defendant is vicariously liable for their conduct.

186. The general rule is that costs follow the result. I see no reason to depart therefrom.

187. In the circumstances, the following order is granted:

ORDER

187.1. The defendant is liable for any damages that are proved or agreed to be due to the plaintiff in her capacity as parent and natural guardian of DM.

187.2. The plaintiff's costs in respect of the determination of the issue of liability are to be borne by the defendant.


A. MAIER-FRAWLEY

JUDGE OF THE HIGH COURT

GAUTENG DIVISION OF THE HIGH COURT, JOHANNESBURG

This judgement was prepared and authored by the Judge whose name is reflected and is handed down electronically by circulation to the Parties/their legal representatives by email and by uploading it to the electronic file of this matter on CaseLines. The date for hand-down is deemed to be 14h00 on 11 May 2023.

Dates of virtual hearing:	24 January 2022 to 11 February 2022; & 18 July 2022 to 22 July 2022 & 4 August 2022.
Judgment delivered:	28 September 2021 11 May 2023

APPEARANCES:

Counsel for Applicant:	Mr BD Molojoa
Attorneys for Applicant:	Jerry Nkeli & Associates Inc

Counsel for respondent:	Mr DJ Joubert SC
-------------------------	------------------