IN THE HIGH COURT OF SOUTH AFRICA

GAUTENG DIVISION, PRETORIA

CASE NO: 34666/2018

(1) REPORTABLE: NO

(2) OF INTEREST TO OTHERS JUDGES: NO

(3) REVISED

DATE: 2025/06/26

SIGNATURE:

In the matter between:

L.S.W. **(O.B.O. F.B.W.)**	**FIRST PLAINTIFF**
F.B.W. **(O.B.O. F.B.W.)**	**SECOND PLAINTIFF**

and

THE PREMIER, GAUTENG PROVINCE	**FIRST DEFENDANT**
THE MEC FOR HEALTH AND SOCIAL DEVELOPMENT, GAUTENG PROVINCE	**SECOND DEFENDANT**

JUDGMENT

MOTHA, J:

Introduction

(1) F.B.W. was born on 27 February 2012, and due to intrauterine severe oxygen deprivation during his delivery, he suffered a serious brain injury that manifested in cerebral palsy (CP). Following an unopposed application in terms of Rule 33(4) of the Uniform Rules of Court, the issue of liability was separated from quantum, which was postponed *sine die.* Accordingly, the issues for determination are those of negligence and causation.

(2) Since some of the medical experts of the plaintiff hail from Cape Town, it was agreed that the trial would be in a hybrid format, part physical and part virtual. Several medical experts testified on behalf of the parties, six to be exact. In addition, the first plaintiff was the sole factual witness regarding her treatment at the Tshwane District Hospital ("TDH"). Neither the doctor(s) nor any nursing staff identified in the hospital records were called to testify. The parties agreed that there was no need to traverse the joint minutes of the specialist physicians because that evidence was relevant to the issue of quantum. Therefore, the evidence to be examined was that of the Radiologists, Neurologists, Genetists, Obstetrician Gynaecologists, Paediatricians and Midwives.

(3) The issues were considered by all the aforementioned experts, each of whom had a counterpart, and resulted in the compilation of joint minutes. These joint minutes significantly narrowed the issues and eliminated the need to call those experts without disagreements recorded in their joint minutes.

The parties

(4) The first plaintiff is L.S.W., an adult female born on 23 August 1991, who acts in her personal and representative capacity as the mother of the patient, F.B.W.

(5) The second plaintiff is B.F.W., an adult male born on 15 January 1983, who acts in his personal and representative capacity as the father of the patient, F.B.W.

(6) The first defendant is the Premier of Gauteng Province, who is cited in his official capacity as the head of the Department of Health of the Provincial Government of Gauteng Province.

(7) The second defendant is the MEC for Health, Gauteng Province, who is cited in her official capacity as the Head of the Department of Health of the Provincial Government of Gauteng Province.

The plaintiffs' case

(8) The first plaintiff testified that she currently resides in Eersterus Township and was 19 years old at the time of her pregnancy. On 26 February 2012, she experienced labour pains at around 04h00 and presented herself at Tshwane District Hospital (TDH) at approximately 06h00. She was advised that her cervix was 1 cm dilated and since the pain was not severe, she decided to go back home. When her contractions increased and became stronger, she returned to TDH between 10h00 and 11h00 Upon the assessment of her cervix, she was admitted with a cervical dilation of between 2 and 3 centimeters.

(9) To increase the cervical dilation, she was instructed to walk around and a heart machine was attached to her to monitor the heartbeat of the baby. At approximately 20h00, she felt her contractions intensifying and communicated to the nurses that she felt as though the baby wanted to come out. A drip was administered to increase the contractions, and a heart machine was reapplied to her. After returning from the ladies' room, she accidentally pulled out the drip, which resulted in her bed being soaked in blood. Consequently, they switched her to another bed, and shortly thereafter, she lost consciousness. Between 00h00 and 00h30, she was awakened, moved to the labor ward, and informed that it was time.

(10) It was her testimony that two nurses attended to her, one on her right side and the other at the end of her feet, at the labour ward. The nurse on her right side placed her hands below her breast and pushed down on her stomach, she stated. This is what the experts refer to as fundal pressure. She estimated that this process endured for about 20 minutes, and the baby was delivered, neither crying nor breathing, at 02h30. She mentioned that she overheard the doctor telling the nurses that she should have delivered a long time ago. She was discharged later that afternoon.

Cross-examination

(11) Under cross-examination, she testified that a cardiotocography (CTG) machine was not on her at all times, save from 20h00 until she switched beds. Her cross-examination was uneventful, as her testimony was unchallenged.

(12) In response to the court's questions, she mentioned that she stopped the consumption of alcohol when she became aware of her pregnancy, and reduced her cigarette intake to approximately 2 to 3 cigarettes per day.

(13) Overall, her testimony was reliable and candid. She was credible and did not shy away from taking the court into her confidence. For example, she openly admitted to smoking during her pregnancy.

The uncontested joint minutes

(14) The Geneticists, Neurologists, and Radiologists reduced their testimonies into joint minutes, which were submitted as uncontested evidence and accepted as common cause between the parties[1]. In the main, their agreements were recorded as follows:

The Radiologists [Dr. T. Kamolane for the defendant and Prof.Andronikou for the plaintiff]

[1] Defendant's heads of argument para 5.2.3

(15) These experts agreed that: -

The MRI demonstrated a basal-ganglia-thalamus [BGT] pattern of hypoxic-ischemic injury in the chronic stage of evolution. TK submitted that the features were consistent with acute profound hypoxic brain injury in a term brain, now in the stage of evolution. They also agreed that there were no features to suggest a congenital infection or malformation.

Neurologists [Drs. D. Pearce, for the plaintiff and V.R. Mogashoa for the defendant]

(16) The neurologists agreed as follows:

"4. We agree that F has an asymmetrical mixed-type cerebral palsy, predominantly dystonic. He is classified as GMFCS V, (Gross motor functional classification scale) and CFCSV (Communication functional classification scale)

5. We agree that F is incapable of independent mobility and his co-morbidities include profound intellectual disability, early contractures, microcephaly, pseudobulbar palsy, previous epilepsy (according to the records from Steve Biko), possible subluxation of his hips, wasting and nutritional concerns, and severe developmental delay.

6. We agree that the MRI which was performed on 15/06/2018 reveals the following features: The MRI study identifies features that constitute an acute profound hypoxic-ischemic injury of a term brain at a chronic stage of evolution. There are no MRI features to suggest intracranial: congenital infections, congenital anomalies, metabolic disorders, inflammatory conditions, or haemorrhage.

7. We agree that the clinical examination concluded by Dr. Pearce and Dr. Mogashoa of F is in keeping with the aforesaid MRI findings.

<u>THE AETIOLOGY OF F'S NEUROLOGICAL DISABILITY</u>

8. We agree that F fulfils the criteria for the diagnosis of a Grade II-III neonatal encephalopathy (According to Samat classification)

9. In determining the aetiology of F's neonatal encephalopathy we have excluded, as far as possible, the following aetiologies: congenital brain abnormalities, intrauterine growth restriction, intracranial haemorrhage, neonatal infection, genetic disorders, inborn errors of metabolism and acquired metabolic causes.

9.1. We have deferred maternal medication and placental insufficiency to expert obstetric opinion.

10. Having regard to ACOG 2003 and 2019 we are of the opinion that when considering intrapartum hypoxia and the aetiology of cerebral palsy, F fulfils sufficient criteria.

10.1 metabolic acidosis – pH 7.06/pC02 21/BE-22.3/ HC03 6.1

Severe metabolic acidosis. Gas taken 90 minutes after delivery. – fulfils criteria

10.2 early onset of neonatal encephalopathy (HIE II-III) – fulfils criteria

10.3 cerebral palsy of mixed type, predominantly dystonic - fulfills criteria

10.4 exclusion of other identifiable causes -excluded as far as possible provided based on records available.

10.5 APGAR score of 2 and 4 at 1 and 5 minutes respectively. - fulfills criteria

10.6 Multisystem involvement: Evidence of respiratory, autonomic and neurological dysfunction noted

10.7 early imaging of acute non-focal cerebral abnormality - cranial sonar showed inhomogeneous parenchyma

10.8 sentinel hypoxic event/deterioration in foetal heart rate following the event - defer obstetric opinion

11. We agree that the timing of the insult is most likely intrapartum.

12. We agree that having regard of ACOG 2019, and based on available records, intrapartum hypoxia is the most probable cause of the neonatal encephalopathy in this child.

13. We therefore agree that F's condition is most likely the result of intrapartum hypoxia.

14. Possible risk factors include maternal nicotine exposure, prolonged labour, meconium-stained liquor and fetal distress. We defer the management of these and their possible contribution to the expert obstetrician.

Geneticists [Drs. G.S.Gericke for the plaintiff and L. Bhengu for the defendants]

(17) In a nutshell, these experts' agreement can be summed up as follows:

F.B.W.'s birthweight was 2700 g, length 48cm, head circumference 34cm. These measures are in keeping with the gestational age at delivery and thus it is clear that there was no intrauterine growth retardation (IUGI) which would have been indicative of an intrauterine/antenatal factor influencing the delivery outcome. Furthermore, the normal head circumference at birth, with microcephaly subsequently developing would be in keeping with the effects of intrapartum hypoxic ischemic on the postnatal evolving brain.

In summary, none of the available facts are as yet contradictory to the outcome of a clinical genetic investigation, which indicates the absence of a genetic contributor/underlying hereditary predisposing factor for EM to have suffered birth asphyxia and/or to develop cerebral palsy associated with global developmental delay. No genetic syndromic features were found clinically or with MRI neuroimaging, and no further investigations or laboratory testing appear to be warranted under the present circumstances.

The findings in this case comply with the baby having suffered intrapartum birth asphyxia and neonatal encephalopathy. The presentation of a mixed cerebral with no syndromic features, even without relevant MRI findings of hypoxic ischemic injury, suggests a linked chain from an intrapartum injury to the current neurological presentation.

With regard to commenting on labour, neonatal management, and cause(s) considered to have resulted in the adverse neurological outcome, they defer to obstetric, neonatal, and paediatric neurological experts.

The second witness for the plaintiff was Professor Edward John Coetzee, an Obstetrician and Gynaecologist.

(18) His opening remarks were that neither alcohol nor smoking could be linked to the subsequent condition of neonatal encephalopathy and mixed-type cerebral palsy suffered by F.B.W.

(19) Commencing his testimony, he focused on the CTG tracings and said he had sight of five CTG tracings, to wit: 06h20, 17h30, 00h19, 00h50, and 01h20 CTG tracings. He explained that the normal foetal heart rate (FHR) is placed at 110 beats per minute (bpm) in some textbooks, but he placed it at 160 bpm. When interpreting a CTG tracing, one would observe short-term variability of at least 5 bpm, as the foetal heart rate was constantly oscillating, he said. Further, he clarified that accelerations were defined as increases in the foetal heartbeat of at least 15 bpm lasting for a minimum of 15 seconds, while decelerations referred to the foetal heart rate decreasing by at least 15 bpm sustained over at least 15 seconds.

(20) When interpreting the 00H19 CTG tracing, he stated that it showed that there was reduced short-term variability and demonstrated five decelerations that lasted for more than 15 bpm, and some lasted for 3 minutes. This, he said, was a long time for a baby's heart rate to go down to that low level.

(21) Commenting on the 00h50 CTG tracing, he stated that it was difficult to interpret as the mother was restless, and it was recorded so on the trace report. Following some probing questions, he said that he saw a few decelerations. He, then, focused on the 01h20 CTG tracing and described the decelerations as subtle.

(22) Finally, he commented on the 17h30 CTG tracing, which he regarded as reassuring. This tracing was recorded approximately 5 hours and 40 minutes after the plaintiff's admission at TDH.

(23) Unpacking and explaining the science behind CTGs, he stated that the deceleration can be compartmentalised into two: (1) Early decelerations which are

caused by the contractions on the baby's head which cause the slowing of the heartbeat, but the moment the contraction relaxes, the pressure on the head also relaxes, and the baby's heart rate goes up again. That is a nerve reaction to the contraction. (2) Late deceleration, which is the delay that lasts longer than the contraction. This is more likely to be indicative of hypoxia. Of paramount importance was his statement that there was nothing absolute about CTG or FHR; rather, its value lay in suggesting the possibility of hypoxia and indicating that the foetus might not be coping with the stress of labour.

(24) He emphasised that the court needed to understand that, during the contractions in labour, the baby virtually gets no oxygen from the mother, as the oxygen supply is suddenly cut off. Once the contraction ends, oxygen flow resumes. Therefore, a baby is a little hypoxic with every contraction. However, if the baby fails to recover adequately between contractions, it usually signifies that the hypoxia persists beyond the contractions.

(25) Referring to the 00h19 CTG tracing as horrendous, he opined that the preparation for a Caesarean section (C-section) should have been initiated at that time. Given the severity of the CTG tracing, he insisted that a C-section should have been done within 30 minutes, and certainly within an hour.

(26) To the question that Dr. Mbokota only saw one deceleration at 00h19, he firmly disagreed that one could see one deceleration.

(27) He lamented the absence of CTG tracings from 01h20 and expressed doubt about the swift cervical dilation of the plaintiff from 3 cm to 9cm within approximately 30 minutes. Furthermore, he criticised the quality of the midwife's clinical notes and the partogram, describing them as extremely poor and lacking in essential information. This was in direct contravention of the Maternal Guidelines[2] , which required that the FHR be recorded hourly, and during active labour, at least every half hour or after every second or third contraction; none of this was done, he stated.

[2] guidelines for maternal care in South Africa Department of Health Republic of South Africa 2007 third edition

(28) Responding to a question about the fundal pressure, he said that it was not recommended, and given that the uterus was contracting, it was most unusual. He interpreted this as an indication that the nurses were worried and questioned the decision to resort to episiotomy. At the time of this labour, episiotomy was frowned upon and only performed if it was evident that the baby could not make it out of the mother's perineum without assistance.

(29) He queried the APGAR score of 2, which is given for a heart rate higher than 100 bpm. Considering that the baby's heart rate was 40 bpm upon arrival at Steve Biko Hospital (SBH) and seeing that it was hypoxic, not breathing, and its colour was blue, he doubted the score. He, however, on this score, deferred to the neonatologists. On the weight of the baby, he deferred to the Paediatricians and Prof Smith and said it was a borderline case. To determine IUGI, he mentioned that one of the most important organs was the placenta. To him, the weight issue was irrelevant because when the mother arrived at TDH, her CTG was normal, with a well-oxygenated baby.

(30) Responding to the question about the administration of pethidine and atarax, he testified that the attending staff ought to have been concerned about the preparation for a C-section, rather than administering medicine that would depress the baby's respiration after birth. With regard to the Neurologists' joint minutes, he deferred to them.

(31) Before dealing with the issues of smoking and birthweight, it bears mentioning that counsel for the plaintiff mentioned that alcohol and cigarettes were not issues for determination by this court, but an issue between the OBSTETRICIAN AND GYNACOLOGISTS (My emphasis). When he was asked to comment on those issues, he testified that smoking two to five cigarettes a day did not lead to cerebral palsy (CP). He added that smaller mothers tended to have smaller babies, and there was no IUGR in this case. Instead, he testified, the abnormal CTG tracing pointed to an intrapartum event as the likely cause of the CP. He remarked that the placenta was neither weighted nor examined, with the only recorded observation being that it was flat and round.

Cross-examination

(32) In response to the question about his opinion that a C-section should have been performed following what he described as a "horrendously abnormal CTG pattern soon after 00h19 when she was only 3cm dilated, with deep prolonged FHR decelerations[3]," as recorded in his report, he reaffirmed his position and added that the presence of meconium stain liquor (MSL) should not be overlooked. He recorded that the doctor was over an hour late.

(33) He recanted his statement that the baby was small for gestational age and said the 10th percentile does not reach small for gestational age and deferred to Prof Smith.

(34) He was confronted with the contents of paragraph 8 of his report, which read:

> "Of note is the fact that this newborn only weighed 2.7 kg at almost 39 weeks, which makes him small for gestational age (see Prof Smith's notes). This could have been due to placental insufficiency (mother was a smoker), which was not detected, and this could explain why the fetal condition deteriorated so rapidly."

(35) He testified that something was supposed to have been done when they realized that the baby was hypoxic, but nothing was done. He insisted that when the mother arrived at the hospital, the baby was healthy.

(36) Referring to his statement: "However, despite the rapid 2nd stage mother needed an episiotomy because she struggled to push," he said he did not understand because this appeared to have been an easy delivery, yet fungal pressure was applied.

(37) Asked about the Pediatricians' agreement that the normal red blood cell count "(NRBC) was strong evidence against a prolonged (> 6 hours) exposure to

[3] Medicolegal report page 8.

intrauterine hypoxia"[4], he agreed with the Pediatricians that the hypoxia was not prolonged, as "there was a hypoxic incident at least 00.00 on 27/2/2012 till birth."[5] He stated that this fit in with his argument that this hypoxia happened late in labour.

(38) When confronted with the Neurologist's agreement that the possible risk factors for F's condition - which most likely result from intrapartum hypoxia - included maternal nicotine exposure, prolonged labour, meconium-stained liquor, and fetal distress, he said he could not be expected to answer what these experts said.

(39) Whilst he agreed with the midwife's initiation of intrauterine resuscitation, he insisted that the decision to perform a C-section should have been made two hours earlier. Consequently, he did not agree with the proposition that since delivery occurred within 25 minutes after the full dilation, a call for a C-section was not justified. He asserted that by the time the full dilation occurred, a C-section would have been underway.

(40) He was questioned on his opinion that neither alcohol nor smoking could be linked to the subsequent condition. He disagreed with Dr. Mbokota's reading of the 00h20 CTG tracing and added that the nurses, too, did not agree with Dr. Mbokota. Hence, they instituted intrauterine resuscitation, and after the said resuscitation, FHR was better but never reassuring, he stated. The fact that the doctor told the nurse to monitor for repeat decelerations meant that there were other decelerations before, he asserted.

(41) He testified that, scientifically, he could not explain why Dr. Mbokota interpreted the 00h19 CTG tracing differently. He acknowledged that short-term variability could be open to differences of opinion, especially in interpreting oscillations, which is less than five; others might see it as above 5 or 6 or 7, but when it came to decelerations, there was no ambiguity. Decelerations are clear by anyone's definition, decelerations are decelerations, and in all the literature, there is an agreement on what a deceleration is, he said. To be courteous to his colleague,

---

[4] Joint minutes of Pediatricians para 1.19
[5] Supra para 7

he wrote that “all the differences in opinion about the interpretation of CTG are based on our individual professional opinion”[6],

(42) Further, he testified that a single reading of FHR was insufficient, as the midwife should have stated the FHR before and after the contraction. A single reading means nothing, he emphasised.

(43) He disagreed with Dr. Mbokota that this was a precipitous delivery. In interacting with the court, he explained that, at present, science cannot measure oxygen supply *in vitro*; doctors deduce the foetal supply of oxygen from what is happening to the FHR, as it is known that when a baby is starved of oxygen, the heart rate goes down. He agreed that the active phase started at about 02h00. Had the timely medical intervention (C-section) occurred, the catastrophic results could probably have been prevented, he said. The court suggested that it would have taken just as long to prepare for a C-section; he answered that the C-section could have been prepared within an hour, and that would have still made a difference.

(44) On further interaction with the court on the issue of a C-section, he clarified that level 1 hospitals are staffed primarily by midwives; level 2 hospitals have both midwives and general doctors, who might not be specialists; and level 3 hospitals are fully equipped, including the presence of specialists such as Obstetricians. It is common cause that the TDH is a level 1 hospital. After explaining MSL, he dealt with the issue of smoking and said 10 and 20 cigarettes would be heavy, but in this case, that did not play a role. He said there is no proper study on that. On engagement with the court, he ruled out nicotine as the cause of low birth weight.

(45) He testified that due to the limited research his opinion on smoking was based on decades of his clinical experience, dating back to 1973. He stated that he dealt with hundreds of cases involving women who smoked during pregnancy, and there is no evidence that smoking causes cerebral palsy.

---

[6] Joint minutes of Obstetricians gynecologists page 5

(46) For the court's benefit, he explained the physiological changes in labour. He stated that before labour begins, the cervix is about 3cm long. As labour progresses, the cervix, which is the mouth of the womb, becomes shorter and shorter as it opens (dilates). The dilation from 0 to 10cm is dependent on the phase of labour. The latent phase of labour is from 0cm to 5cm dilation. During this stage of labour, the cervix dilates by 1 cm every one to two hours. Upon reaching 5 cm, the cervix should dilate 1cm per hour until 10cm dilation is reached, which is called the active phase of labour.

***Paediatrician Prof J. Smith was the third witness for the plaintiff*.**

(47) Prof J Smith testified that, in his opinion, it was after midnight that the foetal condition changed from reassuring to non-reassuring. In the absence of the CTG tracings before 00h19, he agreed to the proposition that one could not make a definitive comment on the foetal condition based on CTG tracings, because they are not there. When informed that the mother testified that a CTG monitor was applied each time her cervical dilation was assessed, he testified that the court was missing a bulk of CTG tracings.

(48) Commenting on the presence of a meconium-stained liquor (MSL) which was recorded at 00:09, he said it was a common finding in term pregnancies, but if accompanied by a non-reassuring foetal status (NRFS), foetal distress should be considered. Thick MSL is almost a *sine qua non* for foetal distress, he stressed. In *casu,* he testified that they agreed that one and a half hours after birth, there was a significant neonatal metabolic acidosis which indicated that significant prior (intrapartum) sustained hypoxic-ischemic injury (HIE) to the foetus causing the passing of the thick meconium, during the hypoxia and the aspiration as a consequence thereof.

(49) As he continued his evidence in chief, he stated that in these matters where it was alleged that labour caused the brain injury, one must have the essential component or doorway (the encephalopathy) through which the asphyxia steps on a causal pathway to cause cerebral palsy. In this matter, he testified that they agreed that there was a prior hypoxia that caused acidosis, as a consequence, there was

the development of a neonatal encephalopathy, which is an altered neurological status of the baby shortly after birth. That was the doorway through which the hypoxia stepped to cause the eventual cerebral palsy, he testified.

(50) He stated that the experts agreed that the outcome (mixed cerebral palsy) was directly related to intrapartum (labour and delivery) sustained hypoxic-ischaemic brain injury.

(51) Regarding the agreement that baby F was small for the gestational age at 39 weeks, which was probably related to some degree of uteroplacental insufficiency, related to maternal smoking, he testified that the gestational age of 38 weeks would be appropriate for baby F, and suggested that he was constitutionally small (meaning genetically small). Moreover, the placenta was not measured, and all that was recorded was that it was flat and round, which he said did not help.

(52) He opined that the hypoxia was of a duration shorter than six hours because it did not cause a significant rise in the nucleated red blood cells. Given the conclusion that there is more evidence of a shorter-lived hypoxia than a chronic, prolonged hypoxia, he testified that this somewhat argued against a chronic placental insufficiency before labour.

(53) He pointed out that they agreed that there was no identifiable or recorded perinatal sentinel or catastrophic event in baby F's case. In the absence of a sentinel event, he postulated that suboptimal/ substandard intrapartum obstetric management emerged as the probable cause underlying the sequence of events and the recorded outcome of the baby. He testified that this manifested itself in the form of the failure to recognise the non-reassuring foetal status that required intensive attention and probably expedited delivery.

(54) Repeating that it was after midnight that there was a change in the CTG, he opined that delivery should have been expedited over and above intrapartum resuscitation. He deferred to the obstetrician on the readings of CTGs.

(55) In addressing the radiologist's description of the structural brain injury as acute profound, he clarified that the term acute profound as used by the radiologists was incorrectly applied since it described the structural brain lesion (basal ganglia-thalamic [BGT]). He further testified that it was recognised that the terminology created a problem as it misleadingly suggested a pathogenesis involving an abrupt, short-lived hypoxic event, and this was incorrect.

(56) The radiologists looked at a static image on an MRI, and without the knowledge of the clinical context, they could not comment on the process, he stated. The use of this term did not suggest a brief or abrupt sentinel event; in this case, the process of hypoxia was more prolonged, considering the abnormal CTG tracing after midnight, he testified.

(57) They also agreed that maternal smoking was a prevalent cause of intrauterine growth restriction. He said smoking affected the foetal placental function, and if it was insufficient, it showed itself during labour, especially as labour progressed. "What happens", he continued, "was that the uterine contractions become stronger and longer in duration during the active phase. The hypoxic stress of normal labour becomes more; thus, an insufficient placenta will not cope with oxygenating the foetus, and the foetus will show a change in the foetal heart rate. Hence, monitoring is important."

(58) He maintained that there was an association between smoking and low birth weight, and the more cigarettes one smoked, the more likely one would have a foetus of lower birth weight than a woman who did not smoke. If one stopped smoking before the last trimester, that reduced the risk; therefore, it all boiled down to placental function, he concluded.

(59) He expressed doubt about the accuracy of the recorded APGAR score of 2 out of 10 because the baby exhibited bradycardia, with a pulse rate below 100 beats per minute, and required manual respiratory support. Based on this, he suggested that an APGAR score of 1 out of 10 would have been more correct.

(60) Addressing the issue of fundal pressure, he stated that it is described as a form of maternal abuse. This maneuver exerts external pressure on the uterus when the contractions are at their peak and the mother is bearing down, subjecting the foetus to three simultaneous forces. He said that these forces accumulatively distort the foetal skull bones, and this mechanical distortion may lead to intracranial pressure that drops the blood flow to the brain by even 95%.

Cross-examination

(61) He was challenged on his understanding that the mother stopped smoking. He responded that, save for reducing the foetal weight, whether she smoked 5, 6, 7, 8, or 9 cigarettes per day, that, in and of itself, had zero chance of causing brain injury. When asked if he knew when the injury occurred and whether it occurred at 00h19 or 02h00, he answered that it was seldom possible to pinpoint the time of the injury. He explained that this type of injury typically followed a preceding hypoxic event, and because of the depletion of foetal reserves, the final insult knocked the brain, commonly during the last 20 or 30 minutes of labour. When pressed to state whether it occurred at 00h19 or 02h00, he elucidated that it was the final insult that occurred during that time, as opposed to the injury. In his opinion, the threshold for injury was reached when the significant threshold of acidosis was crossed, which occurred sometimes after midnight.

(62) Having agreed that 60 minutes was the permitted limit within which to initiate a C-section, he deferred to the obstetricians for a reasonable response time for a doctor, since the doctor took a little more than an hour to respond to calls at 00h45 and 01h00 He said that the Neonatologists weigh babies after birth. Asked by the court about CTG and FHR, he said that CTG was not the only instrument used to measure the foetal heartbeat. He mentioned other instruments such as a foetal scope or a sensor resembling a sonar probe. As the court continued to interact with him about the appropriateness of the C-section, he mentioned that other ways could have been considered such as vacuum or a forceps, what is termed operative vaginal delivery, which includes episiotomy. He explained that operative vaginal delivery or assisted vaginal delivery could only be done once the cervix was fully dilated. When questioned about the fact that an episiotomy was done, he said that it

should have been done 20 minutes 40 minutes after midnight. The court pointed out that she was 3 cm dilated and, therefore, they could not make that call. At this point, he mentioned that the C-section should have been opted for.

***Prof Du Plessis, a Midwife, was the final plaintiff's witness**.*

(63) In their joint minutes, the midwives agreed that, in this case, the midwife did not act according to Maternity Guidelines. The Guidelines clearly stated that when dealing with a patient in labour, a medical opinion must be obtained if that patient's cervix has not dilated to 4cm (active phase of labour) after 8 hours following her admission into the facility, she testified. Upon realising that the labour was not progressing well and 8 hours had passed since admission, she stressed that midwives should have called a doctor to assess the reasons for the slow progress.

(64) Discussing the plaintiff's first stint at TDH at 06h00, she said that it was not uncommon, and as a matter of fact, it was a standard practice to send the patient home for the contractions to get stronger. She noted that the plaintiff was already in the latent phase of labour with her cervix 1cm dilated.

(65) She testified that there was a prolonged latent phase of labour as she was admitted at 11h50 am with a cervical dilation of between 2 to 3 centimetres. With the cervix remaining at 3cm dilation at 00h00, she testified that the normal protocol would have been to call a doctor. This was only done at 00h45, after observing the non-reassuring CTG tracing.

(66) The prolonged latent phase of labour could be caused by various issues, such as the malalignment between the baby and the pelvis (the baby might be too large), or insufficient uterine contractions, she said. Therefore, the midwife had to refer the patient to the doctor to determine the cause of the delay and issue appropriate instructions for further management, she continued.

(67) Even though they agreed with the cause of action of internal resuscitation, she testified that in a situation such as this one of non-reassuring CTG tracing, with a poor beat-to-beat and decelerations, a proper cause for a midwife is to prepare the

patient for surgery over and above internal resuscitation. The fact that she phoned the doctor twice was indicative of her concern, she added.

(68) They agreed that the administration of pethidine was incorrect because there was already meconium and a non-reassuring CTG tracing. She said pethidine has a depressive effect, and the child becomes floppy after birth, but this can be reversed.

(69) She explained that the progress of labour was poorly plotted on the partograph, which has a predictive nature and was supposed to provide the midwives with an overview of what was happening to the mother and foetus. Consequently, she added that they could not interpret that the latent phase was prolonged, and medical opinion had to be obtained.

Cross-examination

(70) When asked about a doctor's reasonable response time to a call from a nurse, she answered that she would not know, but the midwives were concerned; hence, they called the doctor twice at 00h45 and 01h00, and he arrived at 02h00.

(71) She stated that delays in such circumstances were unacceptable. Upon re-examination, she said that on seeing the non-reassuring and possibly pathological CTG tracing, her response would have included initiating a drip, administering oxygen, and correcting the positioning of the patient. Following that, she would have given the tracing 15 minutes to determine if the condition improved. She added that in most cases, a doctor would be contacted simultaneously. In this case, she continued, the doctor's opinion should have been sought at 00h30 The reason for that was that the decelerations observed on the CTG tracing were continuous; it was not just one deceleration or an isolated event, she explained.

(72) The fact that the doctor came an hour and a half late was of great worry. In response to the court's queries regarding the prolonged latent phase, she stated that the applicable Guideline provided that where there is no progress in labour 8 hours after the admission of a patient, a doctor must be consulted. The court enquired what could have been the proper course of action if at 20h00 the CTG tracing was still

reassuring, she mentioned that under such circumstances where the foetal heart rate (FHR) remained normal, the Guidelines dictate that the membranes should be ruptured, or labour be augmented. To augment labour, she explained, typically involved the administration of medication such as oxytocin, via an intravenous drip, to strengthen the uterine contractions, if that was identified as the cause of the delay.

(73) She testified that 20h00 would have been the correct time to call the doctor, who could have changed the plan of action and taken the decision for C-section before the foetal distress, or the foetal problems occurred. She referred the court to Regulation 2488 of the Nursing Act, which regulates the practice of midwives and prescribes that a midwife must obtain a medical opinion when labour is prolonged at any stage. The Maternity Guidelines 2007 regulate the 8-hour stage of the latent phase, she concluded.

Defendants' case

The defendants' first witness was Dr. Mbokota, an Obstetrician-Gynecologist

Dr. Mbokota was cross-examined extensively and at length. The following constitutes his testimony.

(74) Firing an opening salvo, he testified that there was nothing the hospital staff could have done to alter the outcome of this matter. The hypoxic-ischemic encephalopathy (HIE) was not preventable. He testified that when the first plaintiff arrived at the hospital, the fetal condition was normal. She disclosed a history of cigarette smoking and social alcohol consumption. He conceded that her latent phase of labour was slightly prolonged, lasting for approximately 13 hours instead of 8 hours, but that did not have an impact on the outcome of the pregnancy, because, throughout that prolonged period, the fetal condition remained normal. He related that she had a quick progression of labour from 3 centimeters to full dilation in a space of two hours, from 00h30 to 02h00, when she became fully dilated. He testified that this suggested that she had what they call precipitous labour.

(75) Cigarette smoking during pregnancy was a major risk factor for what is called, in utero placenta insufficiency, IUGI, he asserted. The nicotine in the cigarette affects the blood vessels in the body, including the blood vessels in the placenta, by reducing the efficiency of the placenta in transferring oxygen and nutrients from the mother to the baby, he attested. Consequently, she entered labor with the placenta with reduced reserves, and that put her at risk of rapid decompensation during the stress of labour, he continued.

(76) He opined that she suffered an acute fetal compromise during the second stage of labour. He illuminated the stages of labour as follows: The first stage of labour has two phases, namely the latent and active phases. The latent phase is from 0 to 4 cm cervical dilation, and the active phase is from 4cm to 10 cm cervical dilation. The second stage of labor is from the 10-centimeter cervical dilation up to the time the baby is delivered. The final stage is the delivery of the placenta.

(77) During the active phase of labour at approximately 02h05, when she was fully dilated, she suffered a foetal compromise, which he said necessitated that the delivery be expedited. The method chosen to be utilized was dependent on the available time. He explained that the quickest way of delivery was for the mother to bear down and deliver the baby through spontaneous pushing. If that was not feasible, the next option included assisted delivery using either a vacuum device, which would typically be applied to the baby’s head to facilitate delivery, or forceps, which resemble jaws that would be placed around the baby's head to aid delivery. Finally, a Cesarean section, which required more time for preparation. Vacuum and the forceps usually took about 15 to 20 minutes to prepare, and the Cesarean section took about 60 minutes, he concluded. Therefore, at that time, they had to determine the quickest and safest way of delivery.

(78) In this case, the baby decompensated, which meant the heart failed to provide oxygen to the brain during the active phase of labor; he maintained. He said the most likely causes of this were: first, there was in utero placenta insufficiency, IUGI as a result of alcohol, and second, the baby may have aspirated meconium, which had to be confirmed by the neonatologists and pediatricians. When the baby aspirates the MSL, its effects are seen after birth. MSL is a stool that babies normally

pass still in the womb, and generally, it is passed before going into labour or during labour when the baby experiences episodes of distress, he narrated. They are normally grouped into three grades. Grade one is usually very thin and gets passed before the baby goes into labour, grade two is greenish, and grade three is a thick meconium, which indicates that it was passed recently.

(79) The type of injury that the baby suffered, as contained in the radiologist's report, suggested that the baby had an acute profound hypoxic injury, he asserted. By definition, this type of injury occurs when there is a complete blockage of blood and oxygen supply to the baby for a period of not less than 10 minutes; furthermore, he said that if that blockage lasted for longer than 25 to 30 minutes, the baby would die in the womb. Therefore, when the baby is born alive, doctors deduce that the injury would have occurred at least 30 minutes before birth. Regarding how she was managed during labour, he reiterated that she had a slightly prolonged latent phase of labour, which, in his opinion, did not affect the outcome. If it did, the type of injury observed would have been what is called a partially prolonged injury, he concluded.

(80) Examining the issue of CTG tracing, he explained that a CTG has two probes: one probe records the fetal heart rate, and the other probe records the contractions of the mother. The readings produced a graph that would be interpreted. In interpreting the graph, some rules had to be followed, namely: examining the heart rate, rhythm, variability, and whether there were accelerations or decelerations, he averred. He explained that the CTG tracing could either be normal (reassuring), abnormal (pathological), or in between (non-reassuring).

(81) At approximately 00h30, the staff interpreted the CTG tracing as non-reassuring, and he agreed with their interpretation. He testified that midwives followed the Guidelines by initiating intrapartum fetal resuscitation, which entailed altering the position in which the mother was lying, administering oxygen, and putting her on a drip. They continued monitoring to observe if the fetal heart improved. If it improved, they would allow labour to progress, but if it did not improve, they were expected to intervene, either through the C-section or using the two other methods mentioned above, depending on how far she was in labor, he testified.

(82) His view was that the staff intervened timely which resulted in the improvement of the CTG, hence the labour was allowed to progress. Accordingly, the doctor agreed with the staff and instructed them to continue monitoring her with specific attention to any further decelerations, as she was about 4cm dilated. Thereafter, she progressed rapidly to almost full dilation by 2h05 and went on to deliver the child vaginally. He testified that it was during delivery that she suffered acute distress, which resulted in this acute profound hypoxia injury. When such distress occurs, all that one does is simply to expedite the delivery and hope that one will deliver the baby quickly enough before the onset of an injury. He mentioned that the chronology of events is the distress, then an insult, and finally an injury. By the time one sees the FHR changes, it means the baby's heart is struggling to pump enough blood to the baby's brain. So, if one can deliver the baby quickly, one would save the baby. Thus, he concluded that this outcome was not preventable. Had the staff opted for the C-section, it would have taken one hour, which would have been well after 2h30 , he stated. Likewise, the other options would not have been of any assistance because they would have taken 15 to 20 minutes, he reasoned. Therefore, the intervention would not have helped; hence, his conclusion.

(83) When questioned about what constitutes a reasonable response time for doctors to attend to an emergency call, he said 30 minutes was, on average, a reasonable time. He noted that the mother was restless, and to calm her, she was sedated with opiates in the form of pethidine and ataraxia, which prevents the side effects of opiates. He suggested that since the clinical notes were written at 02h00, the doctor must have arrived before 02h00, because he would have first assessed the patient before writing the notes. The doctor allowed the labour to progress and mentioned regular with FHR at 128bpm. He testified that he saw up to seven (7) CTG tracings, namely:

> "CTG TRACINGS
> 5.1. CTG done on 26/02/2012 at 06h10: this trace is reactive with baseline FHR of 150 bpm, good variability and accelerations.
> 5.2. CTG done on 26/02/2012 at 11h18: this trace is reactive with baseline FHR of 140 bpm, good variability and accelerations.

5.3. CTG done on 26/02/2012 at 17h30: this trace is reactive with baseline FHR of 145 bpm, good variability and accelerations.

5.4. CTG done on 26/02/2012 at 23h56: this part of the trace shows poor contact with lots of interference, impossible to interpret.

5.5. CTG done on :27/02/2012 starting at 00h20 to 00h40: this part of the trace is faint but shows mild to moderate contractions, with FHR showing good variability and some accelerations and no decelerations. The baseline FHR is around 120 bpm.

5.6. CTG done on :27/02/2012 starting at 00h50 to 01h10: this part of the trace shows a baseline FHR of about 140- 125 bpm, variability of at least 5-10 bpm, no decelerations noted. Contractions were good. Between 01h00 and 01h10, there is poor contact with a suggestion of a deceleration at the time when the patient was restless.

5.7. CTG done on :27/02/2012 starting at 01h20 to 01h40: this part of the trace shows a baseline FHR of about 120- 125 bpm, variability of at least 5-20 bpm, no decelerations noted. Contractions were good."[7]

(84) During the process of pointing out the disagreements with his counterpart, Prof Coetzee, counsel for the plaintiff objected to the mentioning of IUGI and asserted that Prof Coetzee had excluded IUGI as a cause. He said alcohol was a well-known teratogen with its most severe effects manifesting in foetal alcohol syndrome. The effect of the insult as a result of alcohol would be seen after birth. On the other hand, smoking causes the narrowing of blood vessels in various organs, the placenta being the most important organ affected. That results in IUGR, meaning the baby does not grow to its full potential. At this stage, counsel for the plaintiff objected and submitted that it was common cause that there was no IUGI in this case. It is excluded by Paediatricians, Neurologists, Geneticists, Neonatologists, and Prof Coetzee, he added. Counsel for the defendant did not dispute that assertion and stated that it was not an issue. The objection was upheld.

(85) The focus shifted to the issue of precipitous labour. Responding to the court's question on rapid dilation, he said that he found it hard to believe that she

[7] Medico-legal report of Dr Mbokota para 5

progressed from 3cm to full dilation in less than an hour, especially for a person with a prolonged latent phase of labour and whose membranes were ruptured past midnight. However, he said, to quote him: “Patients do not read textbooks.” He concluded that it was possible, hence he called it precipitous labour.

(86) Reverting to the issue of CTG tracings, he explained that there were three standards for interpreting CTGs, namely: the International Federation of Obstetricians and Gynecologists, the American College of Obstetricians and Gynecologists, and the National Institute of Clinical Excellence in the UK. At this stage, the court’s interest was piqued, and it enquired whether it was possible for one practitioner, applying one standard, to arrive at non-reassuring CTG tracing, while another, examining the same CTG tracing, to conclude otherwise. He responded in the affirmative and added that it was even more likely if one already knew the outcome. He elaborated that two individuals could interpret CTG tracing using the same standard and yet arrive at different conclusions.

Cross-Examination

(87) Referring to Dr. House, a TV Programme, counsel enquired whether Dr., Mbokota accepted the TV doctor's statement that if a doctor gets more than one call from a nurse about the same patient, the doctor should interpret that as an emergency and should stop whatever she was doing and rush to the patient because that would be a matter of life and death. He answered that it depended on where the doctor was. If she were at home, yes, but if she were in the casualty attending to other patients who were also facing life-and-death challenges, no. He explained that when in the casualty, a doctor would be busy seeing patients, otherwise she would be in her room. Answering a question about the doctor’s notes, he called the notes retrospective notes because a doctor first does the examination and then writes.

(88) When asked about the literature he relied upon, specifically the 2014 publication by the American College of Obstetricians and Gynecologists (ACOG) titled *Neonatal Encephalopathy and Neurologic Outcome*, he responded that this Guideline or article was relevant because it outlined that one must fulfill the criteria that exclude proximal risks which are: genetic disorders, congenital infections,

smoking before concluding that the Hypoxic Ischemic Encephalopathy suffered by the child occurred intrapartum, during labour, Secondly, to prove the relevancy of this article, if the AFGAR score at 5 minutes is 5 or less, it was most likely that the insult occurred intrapartum. Additionally, he stated that the presence of multi-system organ failure in the baby was indicative of an intrapartum insult.

(89) Under cross-examination, he was asked why he referred to an article that had no bearing on the issues. He mentioned that the court needed to know whether HIE occurred before labour (antepartum), during labour (intrapartum), or after delivery (postpartum). Concerning the seven CTG tracings he had reviewed, it was put to him that, save for the first batch of CTG tracings which were normal, there was no clear picture of what was happening when focusing on the three CTG tracings from 00:20 onwards, he disagreed with this proposition and asserted that in his view the staff had a reasonable picture of what was happening on the day in question, especially, if regard was had to the Latent Phase observations, namely:

Latent Phase observations

Date	Time	BP	Contractions	FHR
26/02/12	14H00	137/77	Mild	139
26/02/12	16H00	129/68	Mild	144
26/02/12	18H00	120/57	Mild	150
26/02/12	20H00	128/81	Mild	150
26/02/12	22H00	141/65	Mild	150
27/02/12	00H00	131/68	moderate	134

(90) Pressed to answer whether the table on Latent Phase shows beat-to-beat variability or anything else that one picks up on a CTG tracing, he said the record showed a normal FHR condition. At this point, he cautioned against creating an impression that CTG tracing was the only instrument for fetal monitoring. He protested that it was, in fact, the last thing, as the first and most important monitoring tool, and the Guidelines confirmed that much, was manual auscultation with the fetal stethoscope, which was where the recordings came from. Further, he said, if there was an electronic one, which also does not produce a graph, that too was sufficient.

Only in high-risk patients do they use CTGs, and the guidelines record if available, the CTG was not the only thing that monitored a baby, he attested.

(91) Consequently, he concluded that they had a reasonable picture of what was happening to the baby. He conceded that there was a failure to comply with HPCSA 2016 Ethical Guidelines on record keeping, because the patient's records, which include test results and imaging investigation results, were not kept as direct evidence in litigation.

(92) Questioned about the statement that alcohol tended to have adverse effects in the first two months after conception and the most extreme negative outcome of alcohol being fetal alcohol syndrome, he said the effects of alcohol were only seen after the birth of a baby. When pressed on this point, he stated that this was a statement from a textbook that was based on research, and the aim was to show mothers and women that they should cease consuming alcohol when planning to fall pregnant. The damage occurs within the first two months to the neural tube, which forms a baby's brain, he said. But that could only be seen after the birth of a baby, and it was the Pediatricians who could tell whether a baby had that spectrum, and the most extreme form of that spectrum was fetal alcohol syndrome, he reiterated.

(93) Since the Pediatricians who came before the court did not find any fetal alcohol syndrome, he acknowledged that he could not dispute their findings. Given that the plaintiff was a social drinker, he was asked whether he had asked the plaintiff if she had attended any social functions before realizing that she was pregnant, he conceded that he did not and added that because she stopped alcohol consumption early, it may be the reason there was no fetal alcohol syndrome. Ultimately, he conceded and agreed with counsel that alcohol had to be ruled out as a contributing factor to the CP.

(94) When questioned about the agreement between the parties that there was no IUGI and therefore that ruled out placental insufficiency, he disagreed and said the definition of IUGI was relative, if a child has a birth weight of 10th percentile instead of a 15th percentile that meant that baby had not grown to its full potential and in obstetric terms that would be considered as growth restrictions but in pediatric terms

they did not consider it to be growth restriction at 10th percentile. For that baby not to have grown to its full potential, there may have been many causes, and one of them was placental insufficiency, he said. He stated that he did not disagree with the pediatricians that there was no IUGI.

(95) He was cross-examined on why the placenta was not investigated, but simply said that it was flat and round. He answered that it was not routine practice to send the placenta for epistemology unless there was a reason. He confirmed that all placentas are around and conceded that it would have been better if the placenta had been sent for epistemology. He said the effect of smoking was at the level of the blood vessels and microscopic.

(96) Explaining the statement that maternal hemoglobin was inactivated by carbon monoxide from smoking, he stated that hemoglobin was not reduced, but it was the capacity to carry oxygen that was reduced, and he made an example of Izimbawula (braziers).

(97) He explained that cerebral palsy was the end result of brain cell injury caused by a lack of oxygen supply, due to failure of the organ responsible for the delivery of oxygen. Therefore, it could not be said that cigarette smoking was not a proximal risk factor for cerebral palsy, he answered.

(98) Confronted with the possibility that the child's low birthweight could be attributable to genetics because his mother was a tiny lady with a BMI of 20.7, he accepted that possibility but added that at the end of the day the baby was small in relation to what the average weight should be at term, which should be 3.2 kg and hers was 2.7kg. At this point, counsel queried that she was at term and said she was 38 weeks. He clarified that at term was from 37 weeks to 41 weeks, and at 38 weeks, 3.2 kg would be expected.

(99) Faced with the Neurologists' agreement that they have excluded as possible aetiologies: congenital brain abnormalities, intrauterine growth restriction, intracranial hemorrhage, neonatal infection, genetic disorders, inborn errors of metabolism and acquired metabolic causes, he said they excluded IUGI as a direct cause of cerebral

palsy. Given these exclusions, he was asked what was left as an aetiology. He reiterated that what caused the intrapartum hypoxia was the result of something because the baby was not fully grown, the placenta did not allow the baby to endure the stress of labour, especially at the second stage of labour. His evidence was that the birthweight was below average at term.

(100) In his comments regarding the presence of meconium, he explained that thick meconium, classified as Grade 2 or 3, was of greater clinical concern. In contrast, Grade 1 meconium, which is thin, typically indicates that the fetus passed meconium prior to the onset of labour.

(101) When presented with his incomplete recording of the hospital records, he conceded that he left out a word that was illegible when recording: “CTG 128bpm…… regular and history of non-reassuring CTG on a primigravida at 2h00.”

(102) Again, when it was pointed out that he omitted repeat deceleration, he accepted that the handwriting was difficult to read. He was challenged on his statement that she remained in the latent phase until 01h00 the next day; he referred to notes which indicated that PV done CX 9cm at 02h00 27/2/2012. Therefore, the active phase started at 02h00.

(103) The court enquired if he knew when exactly the dilation from 3cm to 9cm occurred. He said we could never know when she moved from 3cm to 9cm. The last assessment was in terms of the Guidelines, which in the latent phase required the assessment every 4 hours and in the active phase every two hours. He explained that the progress of labour meant the dilation of the cervix and the descent of the head. So, the last time she was found to be 3cm dilated in the latent phase was at 01h00, and the next time they were prompted to assess her because she was bearing down was at 02h00 when she was 9cm dilated.

(104) He testified that delays in the latent phase could be caused by various factors, such as when it was a woman’s first labour, or when there was poor uterine activity, meaning the uterus was not contracting sufficiently to facilitate cervical dilation. If the latent phase of labour had gone beyond 8 hours from the time of admission, he said

the Guidelines dictated that a reassessment should be done and the membranes should be ruptured (ROM) to speed up the contractions. Due to the prevalence of HIV, he said it was difficult these days. When asked whether a doctor should be called under such circumstances, he answered that the intervention was determined by the cause of the delay, and if appropriate action was ROM, it would be proceeded with. If there was no progress after 2 hours, then labour could be augmented, meaning giving medicine to add to the contractions, and that required a doctor, he explained.

(105) He referred the court to the Guidelines, specifically under the heading Poor Progress in the latent phase of labour, which stated that the latent phase was prolonged when it exceeded 8 hours. From the reading of the Guidelines, it soon became apparent that it did not mention the calling of a doctor. He added that each institution could develop protocols that were specific to the management of labour, but should be in line with the Guidelines.

(106) He conceded that the midwives did not intervene when the latent phase was prolonged, and that was substandard care. He explained the differences between rupturing the membranes and augmentation of labour. The former is just allowing the mother's own internal processes to speed up labour so that the head of the fetus can descend to the mouth of the womb, and the latter means a doctor is giving medicine, called oxytocin, through a drip to exogenously improve the contractions. Augmentation carries significant risks, such as uterine hyperstimulation, which is when the uterus over-contracts without intervals of relaxation, potentially resulting in foetal hypoxia.

(107) It was put to him that the risks associated with prolonged labour include infections. He responded that if the membrane was not ruptured, there was no such risk. He acknowledged that it could cause fetal distress if there were contractions. In response to the question that labour was induced to prevent these risks, he clarified that induction of labour refers to initiating labour in someone with no labour pain or not yet experiencing contractions by exogenously starting labour, such as by giving them a gel or medicine to drink, to stimulate the onset of labour.

(108) On the issue of meconium stain, he explained again that meconium is green in colour, but over time it changes to brown. When challenged with his reliance on Cronje *et al* writings which stated MSL does not necessarily mean fetal distress, it just places the patient in a higher risk category during labor and only the presence of fetal heart abnormalities in the presence of MSL increases the risk of fetal acidosis and birth asphyxia and thus agent delivery is needed. He mentioned that thin meconium staining required no special management. To explain the process that resulted in the passing of MSL, he referred the court to the days of capital punishment. He said that in the case of a condemned person as the noose tightened around a neck, it precluded the blood flow and oxygen supply resulting in the anus sphincters loosening leading to the passage of stool. This was analogous to what happens to a fetus experiencing hypoxia, its anus sphincters loosen and meconium is passed. A thin meconium was not a cause for concern and may be ignored, he concluded.

(109) Under cross-examination, he was informed that Prof Coetzee said he did not want to be unkind to the colleague, he said that Prof Coetzee had every reason to disagree with him, but he had not been active whilst he was still in active service. He stood his ground when he was told that he was the only one who did not note the decelerations noted by several experts who agreed that there were deep decelerations on the CTG tracing at 00h20. He testified that when the baby was still in the womb, it was the Obstetrician's field, and when it came to CTG interpretation, it was subject to misinterpretation, especially when one already knew the outcome.

(110) He insisted that the management of non-reassuring CTG tracing was not delivery but intrapartum resuscitation, and observing if there was improvement. If it did, labour would be allowed to progress, but if it did not, then delivery would be. A pathological CTG is the one that calls for an expedited delivery. He was asked to interpret the CTG tracing, which prompted the nurses to record it as non-reassuring at 00:19. He answered that what he saw were two variable decelerations that rendered the CTG non-reassuring, but not pathological.

(111) His cross-examination continued on 17 October 2024. He was asked that in the latent phase, the Guidelines require 4 hours vaginal examination and this was

not done. He conceded that the care was substandard, noting that following the 19h30 assessment, the subsequent assessment should have taken place at 23h30. However, it was conducted 40 minutes late, at 00h10, and, in his view, this was not a significant deviation. He readily conceded that there was no compliance with protocol when he was referred to the first gap in monitoring between 12h00 and 19h30.

(112) It was put to him that the Guidelines required that all the risk factors be clearly noted, given the evidence of smoking and drinking, this was not done. He said it was documented on the antenatal card. When counsel pointed out that the Guidelines stated that women with problems or risk factors should be referred to an experienced midwife or doctor who may transfer the mother to a hospital, he answered that the patient was already in the hospital and confirmed that he did not know the experience of the midwife who assessed her. They both agreed that the Tshwane District Hospital is a Level 1 hospital; the Steve Biko Hospital is a Level 3 Hospital. Counsel enquired why the patient was not transferred to SBH. He answered that it was not as simple as counsel thought. For good and sound reasons, Level 1 Hospitals transfer to Level 2 Hospitals.

(113) He was asked about the plaintiff's unchallenged evidence, especially the failure to record in the hospital notes the following: (1) she had a CTG machine on her at 20h00; (2) she complained of strong pains as if the baby wanted to come out; (3) a drip was put on her and it accidentally came out wetting her bed; (4) she lost consciousness ;(5) at about midnight she was woken up and (6) she described a maneuver called fundal pressure which was also not recorded. When assessing patients, he stated that clinicians record both the important positives and important negatives. An important positive refers to something that is discovered, whilst an important negative refers to something clinicians specifically look for but do not find. He stated that clinicians were left to exercise their judgment on what would be documented, and did not include everything the patient mentioned.

(114) On the question of fundal pressure, he stated that the practice was discouraged, and his views were derived from the Multi-Centre Review of Interventions. Explaining further, he said that: first, it did not shorten the second stage of labour; second, it did not cause hypoxia in the baby; and third, it could harm

the mother by causing tears to the uterus. However, a soft abdominal belt was still being investigated. He agreed that the nurses did not follow the doctor's instructions to continue monitoring the CTG; hence, the tracings were not available. In agreement with the midwives, he said the CTG tracing was incorrectly discontinued.

(115) When addressing the importance of the partograph during labour, he unpacked the partograph by showing that it was plotted at 02h with a circle on 2, which speaks to the level of the head, and an x on 9, indicating the cervical dilation. He mentioned that he was taught by the person who designed the partograph. He emphasised that the partograph was not plotted during the latent phase but during the active phase of labour. On the issue of rupturing of the membranes, he referred to the Maternal Guidelines, which required that other causes, such as abdominal pains like abruption placentae, false labour, fetal distress, and cephalopelvic disproportion (CPD), be excluded before the membrane could be ruptured. If there was no progress in labour two hours after rupturing the membranes, he said, then and only then would augmentation of labour be undertaken, and this meant starting oxytocin infusion.

(116) When cross-examined about the reasons for ROM, he answered that:

> (1) ROM is done to allow the fetal head to come onto the cervix, the mouth of the womb, thereby facilitating cervical dilation as the uterine contractions push the head down.
> (2) When the membrane is ruptured, prostaglandins, which are produced by the body, assist in the uterine contraction to assist with the process of labour; hence, the two-hour delay in calling a doctor. This allows prostaglandins time to work.
> (3) The membrane gets ruptured to observe if the amniotic fluid is clear or contains meconium, and if so, to assess the type of meconium. When the membranes are still intact, the amniotic fluid remains.

(117) Still, under cross-examination, he was asked if speeding up the delivery was not the primary reason for ROM and the administration of oxytocin; he emphasised that it was not speeding up the delivery but assisting in the progress of the delivery.

When pressed on this, he expanded and explained the concept of the four Ps: Power, Passenger, Passage and Psyche. Finally, he said, when the labour was prolonged, the first person to get tired was the mother, and next was the fetus.

(118) Answering the asphyxia complication, he said that it only happened where there had been consistently strong uterine contractions, meaning a minimum of three contractions every ten minutes lasting for more than forty seconds, and the strength of contractions was determined by the duration they lasted over forty seconds without labour progress. This would lead to fetal distress. With each uterine contraction, the blood flow to the placenta was reduced, and when the contraction relaxed, the blood flow would be restored, he explained. A healthy foetus was able to tolerate these intermittent reductions in the placental blood flow for a period of 18 to 20 hours.

(119) He conceded that ROM was supposed to be done at 8 o'clock (20h00) and it was not done.

(120) He was referred to the pediatric growth chart for calculation of newborn babies' weights; it was suggested to him that they are more competent to comment on the issue of weight. He testified that Pediatricians dealt with the babies after birth. As obstetricians, he said, they assessed fetal growth during pregnancy, based on, *inter alia*, the last normal menstrual period, the clinical examination of a patient, the cervical measurement, fundal height measured in centimeters from the pubic area to the top of the uterus, and ultrasound estimations. If the estimated foetal weight was less than the expected range for the gestational age, the fetus would be classified as small for the gestational age. Furthermore, if the weight estimate fell below the 10th percentile, by definition, it was called intrauterine growth restriction, he said.

(121) Confronted with Prof Smith's statement that "it, therefore, appears that the baby was either a small gestational age baby (SGA) (pathological state) all constitutionally small (genetic traits). The length and weight of the baby suggest that he was of very small stature. His body proportions, IE, ponderal index (weight length ratio), were 97th percentile for gestational ages between 38 and 42 weeks. These

again point to a constitutionally small baby rather than a pathological small size."[8] He deferred to the pediatricians.

(122) Reverting to the issue of CTG tracing recorded at 00h19, about which Prof EJ Coetzee said it showed marked decelerations and about which the staff recorded at 00h30 that it was non-reassuring with decelerations present, whilst Dr. Mbokota said it was faint but showed no decelerations, counsel asked for an explanation. He maintained that he agreed with the staff, and his CTG tracing was faint as recorded in his notes. Having seen the clearer CTG tracing, he confirmed that there were two or three variable decelerations, but they were not pathological. It was put to him that he was not certain and required clarity, hence he wrote: "that the birth weight of 2.7 kg places the baby just above the 10th centile which is the cut off for low birth weight, we, unfortunately, do not have the weight of the placenta which would clarify us if there was indeed placental pathology which could be due to cigarette smoking and would have contributed to the baby not growing to its full potential average had the weight of 3kg."[9] He answered that: "it is as clear as it is". When it was put to him that he was not sure, he said what we were sure of was that the birthweight of the child was 2,7 kg, which meant the baby was small for its gestational age. He continued that small babies tended to decompensate quickly in the second stage and said: "As to whether there was placental pathology due to cigarette we do not know because: (1) we do not have the weight of the placenta and (2) we do not have the histology."

(123) He accepted that all CTG tracings should have been made available, that the nurses did not follow the protocol after 8 hours of the latent stage, and that the partogram was not completed. However, he said the plotting started at 2 hours. He said that his opinion remained unchanged, but agreed that the progress of labour should have been plotted on the partogram, and failure to do so was substandard. With all of that, or even if the partogram was well plotted, he believed that it would not have changed the outcome.

My impression of Dr. Mbokota and his counterpart, Professor Coetzee.

---

[8] medical legal report of Prof Smith page 10.
[9] join minutes of obstetricians at page 6

(124) I found Dr. Mbokota's knowledge of the subject matter to be exceptional and second to none. Over the course of six hours on the witness stand, including four hours of gruelling and rigorous cross-examination, he not once lost his cool or became animated. He conducted himself with dignity throughout. I am, indeed, indebted to him for the illuminating lecture, which shed light on the intricacies of this topic. The same is true of Prof. Coetzee. However, like all the plaintiff's witnesses, he was not subjected to any robust cross-examination. I found both these experts unwilling to concede in the face of patent contradictions. They were both wedded to their positions. Bias is not the word that comes to mind. If anything, they viewed questions that demanded them to concede as some form of personal attack on their knowledge of the subject. Overall, they added value and shed light, *albeit* under duress at times. Certainly, there were moments in their testimony where their opinions were properly supported by the facts and accorded with logic.

The Paediatrician, Dr. W. Kganane, was the second witness for the defendants.

(125) Her testimony was succinctly summarised in her statement that her expertise was limited to the post-natal care of a baby. When it came to reading CTG tracings and intrapartum care, she deferred to the obstetricians. She testified that exposure to teratogens, such as alcohol, smoking, and traditional medicines, had adverse effects on foetal development. Babies who were exposed to these teratogens tended to be born smaller for gestational age, with a reduced reserve, which made the normal stress of labour and delivery significantly more strenuous, she testified. Moreover, these babies were comparatively compromised at birth and may present with developmental abnormalities relative to their unexposed counterparts. She testified that the plaintiff told her that she smoked 8 to 9 cigarettes per day during pregnancy.

(126) In response to the question about the terms partially prolonged hypoxic-ischemic encephalopathy (HIE) and acute profound, she deferred to the Radiologist and mentioned that she was a Pediatrician.

Cross-examination

(127) When counsel for the plaintiff pointed out that in her report, she noted that the child was born on 27 February 2017, she said it was a typo. She was asked if it was not another typo that the plaintiff said she smoked 8 to 9 cigarettes a day during pregnancy. She answered in the negative and said that there was no mistake in that regard. Having referred to her notes, she confirmed that the statement about 8 to 9 cigarettes was correct. It was pointed out to her that the pregnancy was classified as a low-risk pregnancy despite the issue of smoking.

(128) Her recording of the plaintiff's first visit to the hospital was incorrectly captured as 20 October 2011 instead of 1 August 2011. When she was asked if that was not another typo, she conceded that her record was incorrect.

(129) Confronted with her failure to record that the patient was 2 to 3 centimeters dilated, that the next PV was due at 15hrs as contained in the hospital records, and that atarax was given intramuscularly when recording what was mentioned in the hospital notes, she did not have an answer. She once more reiterated that her role started after the birth of a baby. On the dilation questions, she deferred to the Obstetricians. On the artificial rupture of the membranes, which showed a thin meconium stain, she deferred to the Obstetricians. She conceded that there was another typo in that she recorded at 00h30 "CTG non-reassuring with poor beat-to-beat decelerations" instead of "AND decelerations'(my emphasis).

(130) As already stated, she reiterated that she did not have any comments on CTG tracings. Asked why she was selective in her recording of the hospital records, she said she did not record everything. She testified that any cigarette exposure in the first trimester was significant. When informed of the fact that Dr Mbokoda and Miss Smit were told that she smoked 2 to 3 cigarettes per day, she stood by her record at the time of the interview. She did not have enough evidence to decide on the presence of IUGR (head circumference and length). She explained that LGA, SGA, AGA and IUGR (Large, small, appropriate for gestational age and under or intrauterine growth restriction) were not ticked or circled, just disregarded.

(131) She conceded that between midnight and two o'clock, the fetal monitoring was not done. When reminded that she said that she could not read CTG tracing and

yet agreed with the interpretation of CTG tracing as non-reassuring, she said she was reading an interpretation, not expressing her opinion.

(132) She disavowed her position in paragraph 7.4 in the joint minutes in which she said: “Adequate fetal monitoring was done” rather than agreeing with her counterpart, Prof Smith, who wrote: “The compromise during labour is readily detectable by appropriate fetal monitoring which frequently shows late decelerations and/or decelerations (cord compression).” She stated that she should have deferred to the Obstetricians.

(133) When questioned about the fungal pressure, she deferred to the Obstetricians. Answering the court’s queries, she said the first trimester (first three months) was important as that was when the baby was developing and organs were forming. She said that the weight of the baby, which was 2,7 kg, was relatively small for the gestational age.

My impression of Dr. Kganane and her counterpart Professor Smith

(134) Dr. Kganane was not confident at all and had to be told to speak up on several occasions. She conceded readily; it is not an exaggeration to say that she deferred to the Obstetricians on almost all the activities before birth. She disavowed her earlier position in the joint minutes. She was overwhelmed by the occasion. Despite the court’s reassurance, she never settled down. On the other hand, Prof Smith made a good impression and was not afraid to challenge the Radiologists’ conclusions. Her opinion was sound and supported by facts and reason, even though she was not cross-examined extensively. She illuminated why it cannot be said that F.B.W. sustained acute profound hypoxic brain injury.

The last witness for the defendant was Ms.Smit, a Midwife.

(135) She testified that in this case, by looking at the Latent Phase monitoring, nothing was alarming. The Latent Phase monitoring read:

TIME	Cervix	Contractions	Rom	FHR	BP	PULSE

26.2.12						
14h00	CM	Mild	Intact	139	137/77	80
16H00		Mild	Intact	144	124/68	95
18H00	3-4 cm	Mild	Intact	150	120/57	80
20H00	3cm	Mild	Intact	150	128/81	99
22H00		Mild	Intact	150	141/65	79
26.2.12						
00H00	3cm	Moderate	Raptured	134	131/68	61

At 00h30, the nurse's notes read that CTG was non-reassuring, poor beat to beat with decelerations noted.

(136) She confirmed that the mother was progressing slowly, but the latent phase was difficult to assess as there was no time limit. She referred the court to the differences between The Maternal Guidelines, which record that the latent phase is 8 hours in hospital from the time of admission, and the World Health Organisation (WHO), which records that the latent phase is from 12 to 24 hours. The Maternity Guidelines of South Africa changed in 2016 to align with WHO's 24 hours for a mother to dilate from 1cm to 4 cm. In line with WHO, 4 cm had been altered to 5cm, as the beginning of the active phase of labour, she stated.

(137) Reading from the nurse's notes, she mentioned that Ms. W was restless and moving around, which negatively impacted the quality of the CTG tracings. There were two bands around her abdomen, one for the contractions and the other for the foetal heart rate, since she was moving around, the reading would not be accurate. On the time it took for the casualty doctor to respond, she stated that it depended on what the doctor was doing at the time.

(138) She confirmed that the plaintiff was given pain medication, pethidine and atarax, intramuscular, meaning as an injection. Following the midwife's phone call to the doctor at 01h15, she testified that the doctor must have arrived earlier, perhaps at 01h45, because he wrote his notes at 02h00, which was after the examination. The CTG was found by the doctor to be regular with FHR at 128 bpm.

(139) Asked if the doctor had arrived at 01h30 and decided on a C-section, she said it would have taken about 90 minutes because the procedure involved sourcing a porter, an anaesthetist, and obtaining her consent. However, the books stated that C-section should occur within an hour, she said.

Cross-examination

(140) Confronted with her incorrect narration that the plaintiff was admitted to Steve Biko Hospital, she could not explain the obvious error because the plaintiff was admitted to Tshwane District Hospital. She answered that it was her mistake.

(141) When told that the plaintiff was admitted at 07h15. and no problems were identified, she conceded that she was in the latent phase in the morning. Explaining the differences between 3-4cm cervical dilation at 18h00 and 3cm cervical dilation at 19h00, she said that different staff took over, and the new staff could have arrived at 3cm dilation. When asked about the dilation from 3cm to 9cm within an hour, she said that when pethidine was given the mother relaxed, and she had seen patients promptly dilating from 3 to 4 cm to full dilation in under 5 minutes but conceded that the dilation was too quick, as the norm was 1cm dilation every hour.

(142) Asked if it would not have been appropriate to immediately contact the doctor whilst performing the resuscitation, she responded that they did not have to, but she did not see the CTG, which was horrendous.

(143) She stated that there were two doctors on duty, but still they did not attend to the call immediately; she could not confirm whether either of them was a Gynaecologist. She explained that a partogram was based on the active phase of labour and only started in the active phase. She conceded that the documentation in this case was substandard and confirmed that a partogram was essential as it has a predictive nature. That failure to document the progress of labour on the partogram was substandard care, she agreed.

(144) She stated that she did not receive any CTG tracings. She saw the reading written by the doctor as regular at 128 bpm.

(145) Under cross-examination, she was questioned about her conclusion that Ms. W. was managed according to the Maternal Guidelines; she confirmed that her opinion did not alter. It was put to her that that conclusion contradicted her joint minute in subparagraph 2.1, which records an agreement that the midwife did not act according to protocol when the latent phase of labour did not progress to the active phase of labour after 8 hours of her admission into the hospital facility. She stood by her joint minutes, but later conceded. Her discussion with Prof Du Plessis changed her opinion, she said. She conceded that the Guidelines were not followed.

(146) She conceded that a doctor could have done a whole host of things. She conceded that in a case like this, where labour was prolonged, the Guidelines directed that more frequent monitoring should take place, which was not done.

(147) She was asked a series of questions focused on whether the obstetric management provided was optimal or suboptimal:

> Q: that a doctor took approximately one hour and 15 minutes to respond to a nurse's call?
> A: She said it was not optimal, but one had to think about the period of the day. Q: that a doctor was not called to investigate that her cervical dilation was not progressing after 8 hours in hospital?
> A: Suboptimal, she answered
> Q: that pethidine and aterax were administered, given the CTG tracing and evidence of MLS?
> A: she deferred to the Gynaecologist.

(148) Counsel challenged her answer by referring to her joint minutes at 4.2, which records: "While awaiting assessment by the doctor, the midwife administered Pethidine for pain management.

> 4.2.1 Pethidine, however, is known for its respiratory depressive action.

4.2.2 Administering Pethidine on observation of a non-reassuring CTG tracing was incorrect, and potentially contributed to the adverse neonatal outcomes."

(149) She did not agree but finally conceded that it was suboptimal.

Q: that the CTG was stopped at 01h50 ?
A: She agreed that the CTG was stopped at 01h50 and it was suboptimal.
Q: that the partogram was not plotted in the latent phase of labour?
A: she answered that the partogram was plotted in the active phase.

Q: What comment would she advance to the fact that the hospital notes have no doctors' notes or midwife up until 00h09 of the next day?
A: She said that the Latent Phase observations indicated that they knew what they were doing.

(150) Whilst she agreed that the placenta should have been investigated, she suggested that it might not have been done because of religious reasons.

My impression of Ms. Smit and Professor Du Plessis

(151) Ms Smit made several concessions which assisted the court. For example, she conceded that the Guidelines were not followed, and if a doctor had been called sooner, a doctor could have made meaningful interventions. Together with Dr Mbokota, Prof. Du Plessis and Ms Smit assisted the court to adopt a broader perspective of this matter. They opened the court's eyes to that a narrow approach based on what happened at 00h30 was tantamount to a red herring. The root of this case is deeply embedded in the failure to follow the Maternal Guidelines. They laid a proper factual basis and explained their reasoning to the court.

My Total Impression of the Experts

(152) I found the experts to be well-drilled in slanting their case in the direction they wanted it to go. They tended to over-elaborate and give a detailed explanation if it favoured their version, and hold back if it harmed their version. Unless one has a

bench made up of Gynaecologists or Obstetricians, it is very easy to over-emphasize a particular point to advance one's version and understate a particular point to avoid harming one's version. If funds were not an issue, one or two assessors would be of great assistance to achieve justice in these matters. For example, the fact that Dr. Mbokota said a CTG tracing can be interpreted differently, especially when one already knows the outcome, is most worrying. His assertion was confirmed by Prof Coetzee that variabilities are open to interpretation. It is trite that an expert witness is required to assist the court and not to usurp the function of the court. Upon a proper analysis of the evidence of the experts and their joint minutes, it was imperative to separate the wheat from the chaff and search for an imprint of truth. Accordingly, I think the truth lies in the testimonies of both the plaintiff's and defendants' experts, when viewed in totality.

The joint minutes recording the agreements of the six experts who took the stand:

(153) The Paediatricians // Neonatologists' agreement as reflected in their joint minutes:

> "1.1 The experts agree that the antenatal period appears to have been unremarkable except for the acknowledgement that the first plaintiff smoked 2-3 cigarettes a day (Dr. M. Mbokota [for the defendant; Dr. D Pearce [for the Plaintiff]), after realizing she was pregnant. From Dr W Kganane, she did not consume alcohol during pregnancy but has been smoking throughout pregnancy. She smoked 8-9 cig/day.
>
> 1.2 The plaintiff probably entered labour with a reassuring foetal condition/status.
>
> 1.3 During labour (intrapartum) the foetal condition changed from reassuring to non-reassuring. When this occurred is disputed.
>
> 1.4 Thin meconium-stained liquor (MLS) was recorded at 00:09.
>
> 1.5 A CTG was attached to the patient. This CTG tracing between 00:19 and 01:40 was in keeping with a non-reassuring foetal status (NRFS). This NRFS was recognized as such at 00:30, since there was poor beat-to-beat-variability and accompanying FHR decelerations. Intrapartum 'resuscitation' was

initiated, and it was planned to review the CTG in 15 minutes. A doctor was called.

1.6 F was born in a compromised, depressed condition at 02:30.

1.7 F required resuscitation in the delivery room.

1.8 The paediatric doctor noted that there had been thick MSL.

1.9 F developed meconium aspiration and was meconium stained.

1.10 Within 1½ hours after birth a significant uncompensated metabolic acidosis was recorded (pH 7.06; BE – 22.3 mmol/L; HCO3:-6.1 mmol/L: PaCO2:27 mmHg)

1.11 F developed an early onset neonatal encephalopathy with accompanying multi-organ failure/dysfunction.

1.12 The cause of the brain damage (encephalopathy) is considered to be hypoxia (reduced oxygen delivery to any tissues, such as the brain) and ischemia (depressed blood flow to the body tissue), hence the encephalopathy is referred to as “HIE” (hypoxic-ischemic encephalopathy).

1.13 F was correctly so, subjected to therapeutic hypothermia

1.14 F now suffers from non-progressive mixed cerebral palsy and associated co-morbidities (Dr's W Kganane [for the Defendant]; Dr. VR Mogashoa [for the Defendant]; Dr. D Pearce [for the Plaintiff])

1.15 The MRI of F's brain revealed what is described by radiologists (Dr. T Kamolane [for Defendant]; Prof S Andronikou [for Plaintiff]; Dr. W Burger [for the Plaintiff]) as 'acute profound hypoxic ischaemic injury, i.e. injury to the basal ganglia-thalamus and peri rolandic cortex, also known as cerebral-deep nuclear neuronal injury (Volpe JJ. Neonatal Encephalopathy: An Inadequate Term for Hypoxic — Ischemic Encephalopathy. Ann Neurol 2012; 2012;72:156— 166)

1.16 The respective radiologists found no features of congenital infection or malformation

1.17 The experts are in agreement that the outcome (mixed cerebral palsy) is directly related to intrapartum (labour and delivery) sustained hypoxic-ischaemic brain injury, as also acknowledged by Dr. VR Mogashoa (for the Defendant); Dr. M Mbokota (for the Defendant); Dr. D Pearce (for the Plaintiff)]; Dr. G Gericke (for the Plaintiff); Prof EJ Coetzee (for the Plaintiff) and Prof D du Plessis (for the Plaintiff)

1.18 There is evidence to state that F was small for gestational age at 39 weeks, which was probably related to some degree of utero-placental insufficiency, related to maternal smoking.

1.19 Blood tests were negative for infection. The FBC was normal in all respects; the blood culture was negative and the CRP (C-reactive protein) was slightly raised on day 4 of life. A raised CRP is found in the presence of inflammation due to hypoxic ischemia and/or meconium exposure, in the absence of infection. The nucleated red blood cell count (NRBC) was normal (8/100 WBCs). This normal NRBC count is strong evidence against a prolonged (> 6 hours) exposure to intrauterine hypoxia. The normal NRBC < 10/100 WBCs

2. Smith: The CTG recording of between 00:19 and 01:40 revealed a non-reassuring foetal status (NRFS) throughout. The features it contained, namely poor beat-to-beat variability and accompanying FHR decelerations were in keeping with probable foetal acidemia (pathologic foetal status)

Kganane: I would ask for comment from the Obstetricians on this.

3…

4. Smith: There was no recorded perinatal sentinel or catastrophic event (uterine rupture, uterine tear, placenta praevia, abruption placenta, umbilical cord prolapse, facto-maternal haemorrhage, maternal collapse, shoulder dystocia) which occurred during labour which could theoretically explain the outcome of the birth of a depressed baby who subsequently developed an encephalopathy and then developed neurological disability

Kganane: Agreed

5…

6…

7…

7.1 Maternal smoking is a prevalent cause of intrauterine growth restriction, especially noting drop off in birth weight.

Kganane: Agreed

7.2 The infant with IUGR is much more likely to experience difficulties during labour and delivery, primarily related to utero-placental insufficiency

Kganane: Agreed

7.3 These foetuses are partly compromised, but become completely compromised as labour progresses
Kganane: Agreed."

(154) **The agreement of the Midwives, as recorded in their joint minutes:**

"1. Ms.W started antenatal attendance early in pregnancy and attended the clinic regularly without default.
1.1 But for maternal smoking, the pregnancy progressed without complications. 1.2 The management and care provided by the midwives at the antenatal clinic were appropriate and according to protocol.
2. Ms.W was admitted on 26.2.2012 at 11H50 to TSHWANE DISTRICT HOSPITAL in the latent phase of labour at 2–3 cm dilation of the cervix.
2.1 The midwife did not act according to protocol when the latent phase of labour did not progress to the active phase of labour, 8 hours after admission to a hospital facility [DOH:2010]
2.2 According to R.2488 which regulates the Scope of the midwives Practice, the medical practitioner must be informed of all abnormal findings during labour such as a prolonged latent phase.
2.2.1 Ms.Smit: the doctor was informed at 00h45 on 27.2.2012 after internal resuscitation was done at 00h30
3. Assessment of fetal wellbeing was not done according to the Guidelines for Maternity care [2010]
4. The CTG tracings at midnight was markedly non-reassuring with deep decelerations.
4.1 The midwife correctly communicated the abnormal findings to the medical practitioner and instituted intrauterine resuscitation according to protocol.
4.2 While awaiting assessment by the doctor, the midwife administered Pethidine for pain management.
4.2.1 Pethidine, however, is known for its respiratory depressive action.
4.2.2 Administering Pethidine on observation of a non-reassuring CGGtracing, was incorrect, and potentially contributed to the adverse neonatal outcomes.
5. The delay in assessment by the medical practitioner, considering the observation of fetal distress, falls outside the midwife's control.

6. The decision by the doctor to continue with labour progress, despite the observed non-reassuring trace, falls within the scope of the obstetrician.

7. Continuous fetal monitoring is indicated especially when FHR abnormalities were observed.

7.1 The tracing was incorrectly discontinued at 01:50, despite the instruction from the doctor to carefully monitor the fetus for further decelerations.

7.1.1 Sr R Smit: Doctor Notes at 02h00 indicated CTG was regular with FHR at 128bpm.

7.1.2 Ms.W was fully dilated at bearing down at 02h05.

7.2 The FHR was not assessed during the second stage of labour as per protocol, which resulted in an unanticipated asphyxiated newborn baby.

8. Sr R Smit: Baby was born at 02h30, birth was within 25 minutes of full dilatation (second stage)- intervention e.g., Caesarean section would have taken longer than 25 minutes.

8.1 Prof du Plessis: Agree.

9. Documentation of the progress of labour was poor as it was not plotted on the Partograph. The DOH regards omission to document the progress of labour on the Partograph as sub-standard care."

(155) **Finally, the agreement of Obstetricians-Gynaecologists, as reflected in their joint minute:**

"LW was a 19-year-old G2PO, not a primigravida (EJC). Her last normal menstrual period (LNMP) started on 03/06/2011 and her expected due date (EDD) was therefore 08/03/2012. Her gestational age (GA) and EDD was supported by an Ultrasound Imaging examination (U/S) done on 01/11/2011 when the fetal measurements showed a fetus of 21 weeks GA (using the 50th centile. Her height was 1.6m and weight was 53kg. She smoked and did drink alcohol socially, but stopped drinking alcohol when she discovered that she was pregnant and according to her history was only smoking 2 cigarettes/day towards the end of the pregnancy,

(1) (Neither the alcohol nor the smoking can be linked to the subsequent condition of F.B.W. i.e. neonatal encephalopathy & mixed type cerebral palsy [EJC])

(2) MM opinion: Disagree:

(3) i. Alcohol is a known teratogen meaning that it is an agent that act adversely on the fetus causing permanent anatomical (Cillier 2015:129). Alcohol's teratogenic effects occur in the first 2 months after conception and the most extreme outcome of alcohol syndrome (Brand 2011:442). There is no safe maternal blood alcohol level and thus any amount during

(4) We agree that the routine antenatal tests were all normal and she attended antenatal clinic regularly. No problems were encountered.

(5) Labour

(6) First note concerning labour is at 07:15, but there is no date. It is not clear when labour pains commenced, but as she was only 1cm dilated she apparently returned home and was instructed TCB (to come back) when membranes ruptured (ROM), pains get worse, or any bleeding occurs. She was readmitted at 11:50. Still no ROM, but now 3cm dilated. She is reviewed regularly, but there was no further dilation of the cervix. (MM&EJC agree).

(7) There is a CTG (cardiotocograph tracing) with the patient's name on it and the machine records the date and time i.e. 17:30 on 26/02/2012. This CTG is normal (re-assuring) [MM & EJC agree]

(8) At 00.00 membranes are ruptured artificially and thin meconium-stained liquid is seen. A CTG tracing at 00.19 shows marked decelerations.

(9) At 02:05 LW was fully dilated, and F.B.W. is delivered after an episiotomy was done at 02:30 with APGAR score of 2 and 4 at 1 & 5-minutes i.e. birth asphyxia. There were no CTG tracings or observations during the 2nd stage

(10) Both MM & EJC find it difficult to believe that the LW progressed from 3cms. dilated to full dilation in under an hour.The 3rd stage was normal. There is no weight given for the placenta. All the requirements of MM are therefore met to indicate an intrapartum event.

The law

(156) It is trite that to be successful in a claim for delict, a plaintiff must prove positive conduct or an omission, causation, wrongfulness, fault, and harm, on a balance of probabilities. In *casu,* the parties are *ad idem* that the issue for

adjudication by this court is whether the Tshwane District Hospital staff were negligent, and, if so, whether their negligence caused FBW's hypoxic-ischemic injury, which resulted in cerebral palsy. It bears mentioning that there must be a causal nexus between the negligent act alleged and the damages suffered. The onus is on the plaintiff to prove negligence on the part of the defendant's servants, be it by act or omission.[10]

Causation

(157) As Nkabinde J stated: "The point of departure is to have clarity on what causation is. This element of liability gives rise to two distinct enquiries. The first is a factual enquiry into whether the negligent act or omission caused the harm giving rise to the claim. If it did not, then that is the end of the matter. If it did, the second enquiry, a juridical problem, arises. The question is then whether the negligent act or omission is linked to the harm sufficiently closely or directly for legal liability to ensue or whether the harm is too remote. This is termed legal causation"[11]

(158) When all is said and done, the court in *Lee* did not replace the "but for" test as set out in *International Shipping Co (Pty) Ltd v Bentley,*[12]especially in delictual liabilities involving commission. This test is "designed to determine whether a postulated cause can be identified as a *causa sine qua non* of the loss in question. In order to apply this test, one must make a hypothetical enquiry as to what probably would have happened but for the wrongful conduct of the defendant"[13]. Dealing with this test, the constitutional court in *Lee* said:

> "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. Or, as was pointed out in similar vein by Nugent JA in *Minister of Safety and Security v Van Duivenboden – and -Gore. In Gore*

[10] Telematrix (Pty) Ltd v Advertising Standards Authority SA 2006(1) SA 461 (SCA) para 12
[11] Lee v Minister of Correctional Services (CCT 20/12) [2012] ZACC 30; 2013 (2) BCLR 129 (CC); 2013 (2) SA 144 (CC); 2013 (1) SACR 213 (CC) (11 December 2012) para 38.
[12] 1990 (1) SA 680 (A)
[13] Supra para 65.

the approach adopted in discharging the onus in relation to factual causation was described thus:

'With reference to the onus resting on plaintiff, it is sometimes said that the prospect of avoiding the damages through the hypothetical elimination of the wrongful conduct must be more than 50%. This is often followed by the criticism that the resulting all-or-nothing effect of the approach is unsatisfactory and unfair. A plaintiff who can establish a 51% chance, so it is said, gets everything, while a 49% prospect results in total failure. This, however, is not how the process of legal reasoning works. The legal mind enquires: What is more likely?"[14]

(159) The constitutional court concluded that: "Our existing law does not require, as an inflexible rule, the use of the substitution of notional, hypothetical lawful conduct for unlawful conduct in the application of the but-for test for factual causation."[15]

Negligence

(160) The *locus classicus* on negligence remains *Kruger v Coetzee*,[16] *viz*:

"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.

This has been constantly stated by this court for some 50 years. Requirement (a) (ii) is sometimes overlooked. 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. Hence

[14] Supra para 47.
[15] Supra para 50.
[16] 1966(2) SA 428 (A).

the futility, in general, of seeking guidance from the facts and results of other cases."[17]

The role of experts

(161) Unpacking the role of expert witnesses, the court in *Bee v RAF*[18] stated:

"It is trite that an expert witness is required to assist the court and not to usurp the function of the court. Expert witnesses are required to lay a factual basis for their conclusions and explain their reasoning to the court. The court must satisfy itself as to the correctness of the expert's reasoning. In *Masstores (Pty) Ltd v Pick 'n Pay Retailers (Pty) Ltd* **[2015] ZASCA 164**; **2016 (2) SA 586** (SCA) para 15, this court said '[l]astly, the expert evidence lacked any reasoning. An expert's opinion must be underpinned by proper reasoning in order for a court to assess the cogency of that opinion. Absent any reasoning the opinion is inadmissible'. In *Road Accident Appeal Tribunal & others v Gouws & another* **[2017] ZASCA 188**; **[2018] 1 ALL SA 701** (SCA) para 33, this court said '[c]ourts are not bound by the view of any expert. They make the ultimate decision on issues on which experts provide an opinion'. (See also *Michael & another v Linksfield Park Clinic (Pty) Ltd & another* **[2002] 1 All SA 384** (A) para 34.)"[19]

(162) The court continued and held:

"In *The State v Thomas* (CC 19/2015) [2016] NAHCMD 320 (19 October 2016), the mental condition of the accused, which was in question, was enquired into by two psychiatrists and they produced reports. In respect of the experts' reports, the court at para 29 said:
'When dealing with expert evidence the court is guided by the expert witness when deciding issues falling outside the knowledge of the court but within the expert's field of expertise; information the court otherwise does not have

[17] Paras E, F and G.
[18] (093/2017) [2018] ZASCA 52; 2018 (4) SA 366 (SCA) (29 March 2018)
[19] Supra para 22.

access to. It is however of great importance that the value of the expert opinion should be capable of being tested. This would only be possible when the grounds on which the opinion is based are stated.7 It remains ultimately the decision of the court and, although it would pay high regard to the views and opinion of the expert, the court must, by considering all the evidence and circumstances in the particular case, still decide whether the expert opinion is correct and reliable.'"[20]

(163) On agreements between the experts, the court said:

"This raises the question as to the effect of an agreement recorded by experts in a joint minute. The appellant's counsel referred us to the judgment of Sutherland J in Thomas v BD Sarens (Pty) Ltd [2012] ZAGPJHC 161. The learned judge said that where certain facts are agreed between the parties in civil litigation, the court is bound by such agreement, even if it is sceptical about those facts (para 9). Where the parties engage experts who investigate the facts, and where those experts meet and agree upon those facts, a litigant may not repudiate the agreement 'unless it does so clearly and, at the very latest, at the outset of the trial' (para 11). In the absence of a timeous repudiation, the facts agreed by the experts enjoy the same status as facts which are common cause on the pleadings or facts agreed in a pre-trial conference (para 12). Where the experts reach agreement on a matter of opinion, the litigants are likewise not at liberty to repudiate the agreement. The trial court is not bound to adopt the opinion but the circumstances in which it would not do so are likely to be rare (para 13). Sutherland J's exposition has been approved in several subsequent cases including in a decision of the full court of the Gauteng Division, Pretoria, in Malema v The Road Accident Fund [2017] ZAGPHC 275 para 92.

In my view, we should in general endorse Sutherland J's approach, subject to the qualifications which follow. A fundamental feature of case management,

[20] Supra para 29.

here and abroad, is that litigants are required to reach agreement on as many matters as possible so as to limit the issues to be tried."[21]

(164) From the reading of this judgment, it is patent that where experts in the same field have reached an agreement "a litigant cannot be expected to adduce evidence on the agreed matters. Unless the trial court itself were for any reason dissatisfied with the agreement and alerted the parties to the need to adduce evidence on the agreed material, the trial court would, I think, be bound, and certainly entitled, to accept the matters agreed by the expert."[22]

Submissions:

Plaintiff's counsel

(165) Counsel for the plaintiff submitted that the defendant's witnesses fared very poorly. He reminded the court that the Pediatricians excluded IUGI as an etiology in this case; both the Obstetricians found it hard to believe that the plaintiff promptly dilated from 3cm to full dilation in under an hour; the midwives agreed that R2488 was not observed and the FHR was not assessed during the second stage of labour which resulted in the asphyxiation of the newborn baby. When summarising the evidence of the plaintiff's experts, he bemoaned the defendants' failure to produce CTG tracings between 20h00 and 00h00.

(166) He asked the court to draw a negative inference for the defendant's failure to call the nurses and doctors to testify. Dealing with the defendant's experts, he submitted that they recorded in their reports that the management of the plaintiff was according to Maternity Guidelines and everything was done according to the book, yet all of them, without exception, conceded under cross-examination that that was not the case. He submitted that Dr Kganane was of no assistance to the court and questioned her presence because she could not comment on the obstetric management. In essence, it was his submission that all the witnesses of the defendants were biased.

---

[21] Supra paras 64 to 65.
[22] Supra para 70.

(167) He questioned how the defendants conducted their case, noting that Sister Smith was not furnished with CTG tracings despite having asked for them nor was Dr. Mbokota furnished with the reports prepared by other experts. He asserted that Sister Smith was horrified upon being shown the 00h19 CTG tracing and had no hesitation in concurring with Prof Coetzee. She testified that the failure to plot the partogram constituted substandard care. Consequently, she said the staff did not know what was happening to the patient, he reminded the court.

(168) Referring to Dr. Mbokota's statement that the CTG tracing on caseLines was clearer than the one given to him, he questioned his failure to ask for a clearer copy. Questioning Dr. Mbokota's classification of CTG tracing into reassuring, non-reassuring, and pathological, he said these were not contained in his report or his joint minutes. He had no option but to acknowledge that Dr. Mbokota was an educated man, but concluded that he was biased. He questioned Dr. Mbokota's reliability in the face of agreements that IUGI was ruled out by pediatricians, neurologists, and geneticists, and added that he was not able to counter the opinion of Prof Smith that the baby was a constitutionally small baby rather than a pathologically small baby. He asserted that the geneticists effectively removed IUGI. He pointed to a contradiction in Dr. Mbokota's assertion that smoking was the cause of IUGI, yet he concluded in the joint minutes that " we unfortunately do not have the weight of the placenta, which will clarify if there was indeed placental pathology which could be due to cigarette smoking." He said one can not express something with certainty and also seek clarity.

(169) He referred to some negative comments made by another court about Dr. Mbokota. He submitted that the plaintiff was diagnosed at 06h00 and something should have been done at about 14h00, but was only seen by a doctor at 02h00, 12 hours later than required. Given Dr. Mbokota's evidence, he continued, the negligence and causation are evident. In addition, there were insults such as the administration of pethidine which was contraindicated, the use of fundal pressure, the non-recording of FHR during the second stage of labour, which according to the midwives resulted in this outcome, and the failure to plot the progress of the labour on the partogram, he added.

(170) He referred to *Lee's* case and stated that causation had been established. He then dealt with costs and asked for costs on a punitive scale because of the defendants' failure to produce CTG tracings and call the doctors and nurses to testify. He argued that the more than one-hour delay in responding to the nurses' call required an explanation. He submitted that counsel for the defendants failed to cross-examine the plaintiff's witnesses. He found the attempt to restrict the court's attention to only what occurred between 00h30 and 02h30 alarming and misleading. The evidence of smoking and alcohol, which was not pleaded, he submitted, was inadmissible. He said Dr. Kganani's testimony was a waste of the court's time.

Defendants' counsel

(171) Counsel for the defendant submitted that the plaintiff had failed dismally to demonstrate the causal connection. He submitted that this matter could be disposed of easily, even if it was brought on a default judgment. Referring to the matter of *Bee v Raf*, he said that the joint minutes are binding and cannot be deviated from without an application. He submitted that both Coetzee and Smith attempted to deviate from their joint minutes. Responding to the issue of cross-examination, he argued that cross-examination was a choice, and to paraphrase, he said if a witness was not hurting one's case, one was not bound to cross-examine; it is not a must. He argued that experts are different from factual witnesses; they analyse the data and express their opinions, and the court decides which one is backed up by the facts.

(172) He referred to the Radiologists' joint minutes and asserted that this was an acute profound injury. When the court enquired about the failure to amend the plea, he reiterated that this case could be dismissed even if the defendant was not there. On further questioning by the court, he confirmed that smoking and alcohol were not pleaded. Eventually, on the amendment issue, he said: "Why bother on something that is conceded? It is a fact." He argued that the court could not exclude that evidence simply because it was not pleaded and stated that the witnesses were cross-examined on it.

(173) Questioning the credibility of Prof Coetzee, he submitted that he disavowed his medico-legal report's position, in which he wrote: "Gestational age was therefore 38+ weeks at birth…
Of note is the fact that this newborn only weighed 2.7 kg at almost 39 weeks which makes him small for gestational age (see Prof Smith's notes). This could have been due to placental insufficiency (mother was a smoker), which was not detected and this could explain why the fetal condition deteriorated so rapidly."

(174) He submitted that he recanted this position in the joint minutes and said "Neither the alcohol nor the smoking can be linked to the subsequent condition of F.B.W. i.e. neonatal encephalopathy & mixed type cerebral palsy [EJC]"

(175) He submitted that Neurosurgeons agreed in their joint minutes that: "Possible risk factors include maternal nicotine, prolonged labour, meconium-stained liquor and fetal distress. We defer the management of these and their possible contribution to the expert obstetrician." Furthermore, he referred to the joint minutes of Geneticists, specifically where they wrote: "There is a Maternal history of cigarette smoking during pregnancy but denied alcohol ingestion."

(176) Continuing in this line of submission, he pointed to the joint minutes of Pediatricians, particularly where they said:

> "1.18 there is evidence to state that F was small for gestational age at 39 weeks, which was probably related to some degree of utero-placental insufficiency, related to maternal smoking."

(177) Contrary to Prof Coetzee's statement that the CTG was subtle following the intrapartum fetal resuscitation, Dr. Mbokota said it was normal, he said. He said Prof Coetzee has a propensity to be partisan because he tried to be polite to Dr. Mbokota on the interpretation of CTG and put his credibility into question because he has the capacity to please people. Second, he submitted that Prof Coetzee cannot be relied upon as he wrote different things in his report and the joint minutes.

(178) On the issue of the C-section, he submitted that there was just not enough time for it because it had to be done within an hour from the action plan, which includes organizing nurses, anesthetists and securing consent from the patient. He embarked on a mathematical computation of possible time for a C-section and asserted that: Dr. Mbokota testified that 30 minutes is a reasonable response time for a doctor to attend an emergency call; the doctor was called at 00h45. He suggested that the doctor would have been expected at 01h15 and upon arrival, he would have examined for 10 to 15 minutes, according to Dr. Mbokota, which would lead to 01h30. According to him, the C-section would have been done by 02h30. Therefore, that window of opportunity to perform a C-section was not available. He argued that the experts agreed that the injury occurred in the last 20 to 30 minutes.

(179) Referring to the matter of *MEC of Health and Social Development of the Gauteng Provincial Government v M,*[23] he submitted that this case was similar to this matter and mentioned paragraph 26 in which the court said:

> "…The courts have cautioned against commencing with an unfavourable outcome and working backwards in search of a cause. Hornbuckle J warned that with the benefit of the knowledge that there has been a neurologically unfavourable birth outcome, a plaintiff's attorney 'can take any foetal monitor strip and make a malpractice case out of it."

(180) Examining *Lee's* case, he submitted that *Lee* was not applicable in this case. He submitted that *Lee's* case is not applicable where the cause and timing of the injury are known and referred to *AN v MEC for Health, Eastern Cape* (585/2018) [2019] ZASCA 102 (15 August 2019).

(181) Since the nurses did not call the doctor for the third time, having already called him at 00h45 and 01h15, he submitted that the situation was no longer so adverse and that there was no longer any emergency. Hence, he added, the doctor simply instructed the staff to monitor for further deceleration.

---

[23] 272/2022) [2024] ZASCA 21 (05 March 2024)

(182) He revisited the issue of pleadings and referred to the case of *Minister of Safety and Security v Slabbert,* stating that the issue was fully canvassed and the court cannot ignore it simply because it was not pleaded. He submitted that the plaintiff did not raise any prejudice, but cross-examined witnesses extensively on it.

(183) In conclusion, he submitted that the matter should be dismissed for lack of a causal nexus or connection, but conceded that there was evidence of non-compliance with the Guidelines.

Reply

(184) In reply, counsel submitted that the plaintiff was prejudiced by the defendant's failure to amend pleadings to include the issues of smoking and alcohol. He insisted that this was an issue between the experts and dealt with it as such, in anticipation of a possible amendment from the defendants. He stood by his original submissions and argued that the defendant had a duty to discover every document, including all the CTG tracings. He enquired where the CTG tracings from 20h00 were. Finally, he asked why the witnesses were not called to decipher the illegible notes and testify about the missing CTG tracings.

ANALYSIS

(185) It is common cause that the plaintiff presented herself at Tshwane District Hospital on 26 February 2012 at approximately 06h00. She was diagnosed as being in the latent phase of labour, with a cervical dilation of 1cm. She elected to return home. When her contractions intensified, she went back to the TDH and was admitted at about 11h50 as a low-risk patient, with her cervix assessed to be 3cm dilated, still in the latent phase of labour. It is also common cause that the next cervical dilation was at 02h05 of the following morning when her cervical dilation was 9 cm.

(186) Furthermore, it is common cause that the midwives did not follow the Maternal Guidelines' procedures in dealing with the plaintiff. In terms of the Maternal Guidelines, the latent phase is prolonged when it exceeds 8 hours. The plaintiff first

attended the Hospital at 06h00, and her 8-hour cut-off would have been 14h00. However, in terms of the same Guidelines, the computation of the 8 hours commences on admission, which was 11h50. Consequently, the correct timeline is 19h50 or 20h00.

(187) It is noteworthy that at 20h00, she experienced contractions and told the nurses that she felt like the baby wanted to come out. This time coincided with the 8-hour deadline in terms of the Maternal Guidelines. Surely, the moment had come to act and rupture the membranes after excluding other causes mentioned above, such as abdominal pain and false labour in terms of the Maternal Guidelines. Whether one accepts the version of Dr. Mbokota, whom I hold in high regard due to his expertise in the subject, or the midwives' version, this was not done. The membranes were ruptured at 00:10, almost four hours late.

(188) Again, regardless of whether one accepts the version of the midwives or Dr. Mbokota, at 22h00, two hours after the 8-hour deadline, a doctor should have been called to initiate an oxytocin infusion if the delay in the progress of labour was attributable to the weak contractions. This was not done. The doctor was called at 00h45 for the first time and at 01h15 for the second time (2hrs 45 minutes and 3hrs 15 minutes late). The precise time of the doctor's arrival is disputed, but what is not in contention is that he recorded his clinical notes at 02h00, some four (4) hours late according to Dr.Mbokota or six (6) hours late according to the midwives.

(189) Following the Maternal Guidelines, she was supposed to be vaginally examined at two-hour intervals. This protocol was not observed. When it was eventually followed at 00:10 , she was still 3cm dilated. Having regard to the plaintiff's uncontested testimony that a drip was inserted into her, which accidentally got dislodged, leading to the change of beds, this court is not told, nor is it noted in the hospital records, what necessitated the insertion of a drip. Moreover, the absence of CTG tracings between 20h00 and 00h19 when a non-reassuring tracing was recorded is both suspicious and devastating. It is suspicious when viewed alongside the plaintiff's uncontested testimony that at 20h00 a heart machine was put on her. Dr. Mbokota saw a CTG tracing done on 26/02/2012 at 23h56 and stated: "This part of the trace shows poor contact with lots of interference, impossible

to interpret". This is devastating because if the tracing was non-reassuring at that time, what was done at 00h19 would have been done earlier. It is more than probable that a C-section at that time would have resulted in a different outcome.

(190) Whilst I understand that CTG is not the only tool to measure the foetal heartbeat, in this case, a CTG was utilized. Accordingly, the tracings were supposed to be made available. The defendant's failure to call witnesses such as midwives and doctors on duty on that faithful day to fill in the gap does not put the defendant in a good light, see Galante *v Dickison.* [24]

(191) When the membranes were ruptured, some four hours later, contrary to the Guidelines, a thin meconium-stained liquor was observed. Nine minutes thereafter, at 00h19, the CTG tracing was non-reassuring. The comedy of errors, which would be funny if it were not a matter of life and death, continued as they administered pethidine and ataraxy. To their credit, they did intrapartum resuscitation. This did not help; hence, they called the doctor at 00h45 am, 55 minutes after the non-reassuring CTG tracing report. The fact that they called the doctor for the second time, 30 minutes later at 01h15, is indicative of the seriousness of the situation. In his testimony, Dr Mbokota made too much of the doctor's notes at 02h00, which recorded 128 bpm and regular. Besides the fact that he did not capture the notes correctly, as he left out some words, his testimony is at variance with the defendant's amended pleas at 6.16 which noted: "At 02h00 plaintiff's cervix was still 3cm dilated and CTG tracing was non-reassuring and it was reading 128bpm and was regular." This CTG tracing was not made available to the plaintiff or the court. Be that as it may, the truth is that CTG tracing was non-reassuring. This reinforces the conclusion that a C-section should have been prepared as early as 00h20, if not significantly earlier. During the court's conversation with Prof Coetzee, it became evident that a C-section was both indicated and long overdue, particularly in light of the fact that she was still 3cm dilated.

(192) Dr. Mbokoda had sight of seven CTG traces. The ones that are of interest are the following:

---

[24] 1950 (2) SA460 (A).

“CTG TRACINGS

5.4. CTG done on 26/02/2012 at 23h56: this part of the trace shows poor contact with lots of interference, impossible to interpret.

5.5. CTG done on :27/02/2012 starting at 00h20 to 00h40: this part of the trace is faint but shows mild to moderate contractions, with FHR showing good variability and some accelerations and no decelerations. The baseline FHR is around 120 bpm.

(193) It is on the reading of this tracing that the midwife was prompted to not only embark on intra-foetal resuscitation but also call the doctor. Dr. Mbokota’s explanation for not seeing decelerations on this CTG tracing is that the one given to him was illegible. The rhetorical question is, why make a definitive reading on a tracing you cannot see properly, especially in a matter of life and death? I reject his explanation and accept Prof Coetzee's version that the CTG tracing was horrendous and required expedited delivery.

(194) To his credit, though, Dr. Mbokota revised his initial position of not observing decelerations at 00h19 or 00h20 on the CTG tracing to seeing at least two to three decelerations. His counterpart all along regarded this CTG tracing as horrendous and necessitated an expedited delivery. Had this been done, the child would have been delivered within 60 minutes in terms of the Guidelines, which could have been any time before 01h20 or 01h30 at most. Seeing that she was still 3cm, the other modes of expedited delivery would have been contraindicated.

(195) Dr. Mbokota testified that the CTG tracing was not the only tool to monitor the progress of labour and referred to Latent Phase Observations.

(196) LATENT PHASE OBSERVATIONS

DATE	TIME	TEMP.	PULSE/	RESP.	B/P	URINE	CONTRACTIONS	FHR
26/2/12	14h00	36.c	80-18	15/77	Toilet	mild		139

26/2/12	16h00	36.5c	93-20	129/68	---	mild 144
26/2/12	18h00	36.2c	80 -18	120/57	Toilet	mild 150
26/2/12	20h00	86.6c	99 – co	128/81	Toilet	mild 150
26/2/12	22h00	36.	79 – 20	141/65		mild 150
27/2/12	00h00	36. 2c	61- 18	131/ 68	Toilet	moderate 134

(197) He stated that this showed the monitoring was reliable. I do not agree; if anything, these notes reinforce the testimony of the plaintiff. Indeed, she went to the toilet at 20h00 as recorded in the notes. She said she lost consciousness just after 20h00; this dovetails with the recording as there is no toilet visitation between 20h00 and 00h00 She testified that she was awoken at 00h00 when she was told it was time. This corresponds with the toilet visitation at 00h00. It is probable that the plaintiff was not attended to between 20h00 and 00h00 when ROM was supposed to have been done. Given what is stated in the Guidelines, this was a critical period, regardless of whether the CTG tracing was reassuring or non-reassuring.

(198) The FHR, as shown, is of little help if there is no record of baseline variability, accelerations, and/or decelerations. Documentation of the progress of labour was poor as it was not plotted on the partograph. It is patent that from 19h30 to 00h30 there was no proper monitoring of the plaintiff. All the experts are in agreement that this was substandard.

(199) Whilst on the issue of negligence, counsel for the defendant in his closing remarks categorically stated that there is evidence of non-compliance with the Guidelines, to quote him: “There is evidence about non-compliance with the Guidelines that is there, we accept that we don’t have to… it is there. You check what they did, you check the guidelines, it is there … but it does not establish causation, you still have to establish a causal connection.” From this concession and given Dr. Mbokota’s concessions, I am of the view that negligence has been established. Accordingly, the issue of negligence is no longer in dispute.

What am I to make of the fact that the plaintiff was a social drinker and smoker?

(200) It is common cause that she (a) attended clinic regularly and all routine antenatal tests were normal, (b) the plaintiff was a social drinker and a smoker and (c) she testified that she stopped drinking alcohol when she discovered that she was pregnant and reduced her intake of cigarettes to two per day.

(201) It is impermissible for a court to have recourse to issues falling outside the pleadings. It is undeniable that there is no reference to smoking and alcohol in the defendant's plea. When the court asked why an amendment was not effected to their plea, counsel for the defendant said: "Why bother on something that is conceded."

(202) The court stated, in the matter of *Imprefed (Pty) Ltd v National Transport Commission,*[25] that:

> "At the outset it need hardly be stressed that: - The whole purpose of pleadings is to bring clearly to the notice of the Court and the parties to an action the issues upon which reliance is to be placed…'The object of pleadings is to ascertain definitely what is the question at issue between the parties; And this object can only be attained when each party states his case with precision.'"[26]

(203) *In Robinson v Randfontein Estates GM Co Ltd,* [27] the court put it as follows:

> "The object of pleading is to define the issues; and parties will be kept strictly to their pleas where any departure would cause prejudice or would prevent full enquiry. But within those limits the Court has a wide discretion. For pleadings are made for the Court, not the Court for the pleadings. And where a party has had every facility to place all the facts before the trial Court and the

[25] 1993(3) SA 94
[26] Supra page 107 C to E
[27] 1925 AD 173

investigation into all the circumstances has been as thorough and as patient as in this instance."[28]

(204) Examining the purpose of the pleadings, the court in the matter of *Minister of Safety and Security v Slabbert* [29]said:

> "[11] The purpose of the pleadings is to define the issues for the other party and the court. A party has a duty to allege in the pleadings the material facts upon which it relies. It is impermissible for a plaintiff to plead a particular case and seek to establish a different case at the trial. It is equally not permissible for the trial court to have recourse to issues falling outside the pleadings when deciding a case."

(205) Besides the fact that the defendant did not amend its pleadings to include this issue, I agree that the issue was canvassed at great length and, therefore, this court must consider it. In examining this issue, it is noteworthy that, as already stated, the experts excluded, *inter alia,* intrauterine growth restriction as an aetiology.

(206) Dr. Mbokata, who seemed to be a lone crusader on these issues of alcohol and smoking, made serious concessions under cross-examination. On the issue of alcohol, he conceded that he did not ask nor know how many social events the plaintiff had attended before she knew she was pregnant and stopped alcohol consumption since she was a social drinker. Having accepted the conclusion of the Paediatricians, to whom he had deferred, that there was no foetal alcohol syndrome in this case, he was constrained to concede that alcohol played no role in the HIE. On the issue of smoking, it is common cause that nicotine affects the placenta, which in turn has an effect on the growth of the foetus. In this case, Dr. Mbokota pointed out that the birthweight was 2700 g. This was small for the gestational age of 38 weeks.

(207) It is common cause that smoking, per se, does not cause hypoxia. It is one of the proximal causes of hypoxia. To arrive at the decision that nicotine had a hand in

---

[28] Supra para 198
[29] (668/2009) [2009] ZASCA 163; [2010] 2 All SA 474 (SCA) (30 November 2009

the hypoxia, the placenta needs to be investigated. In *casu*, this was not done. It was simply said that the placenta was flat and round. Under cross-examination, Dr. Mbokota stated that: “as to whether there was placental pathology due to cigarette we do not know because (1) we do not have the weight of the placenta, (2) we do not have the histology”. Moreover, Prof Smit was not challenged in his conclusion that this was a constitutionally small baby, having regard to the head circumference and the body length of the baby. In this respect, he was supported by the joint minutes of the Geneticists and Neurologists, who excluded (intrauterine growth restriction) IUGR as an etiology.

(208) Prof Smith made a revealing statement that is based on the science of red blood cells. To repeat, He opined that the hypoxia was of short duration, of less than six hours, because it did not cause a significant rise in the nucleated red blood cells. There is more evidence of a shorter-lived hypoxia than a chronic, prolonged hypoxia. This, he testified, somewhat argues against chronic placental insufficiency before labour.

(209) The Paediatricians agreed that there was a significant neonatal metabolic acidosis, which indicated that significant prior (intrapartum) sustained hypoxic-ischemic injury (HIE) to the foetus caused the passing of the thick meconium.

(210) By contrast, Dr. Mbhokota talks of precipitous labour. Indeed, if there were a sentinel event, there would have been very little that the doctors could do. However, this was not such a case. Given Prof Smith's elucidation on what the Radiologists called acute profound and the Pediatricians' joint minutes that this was not a sentinel event, it is most probable that the hypoxia was of a short duration of fewer than six hours due to prolonged labour, which, in my view, is the aetiology of F.B.W.’s neonatal encephalopathy.

(211) Both Dr.Mbokota and Professor Coetzee questioned the rapid dilation of the plaintiff from 3cm to 9cm in less than an hour. Secondly, the records of the progression of labour were inadequate and unreliable as it was not plotted on the partograph; the CTG tracing was incorrectly discontinued at 01h50. The paediatric doctor noted that there had been thick meconium-stained liquor. The midwives did

not comply with R2488 in that they did not call a doctor when there was no progress in labour after 8 hours following her admission into TDH.

(212) The courts are not imbued with magical retrocognition powers, nor can they foretell the future like zangomas or soothsayers. In civil matters, South African courts rely on their Roman-Dutch colonial inheritance of the balance of probabilities[30]. A very nebulous and ill-defined term and method for a party to discharge the onus it bears to prove that its version is more likely to be true than the other party's version. This rule "that a judge may, in his discretion, act on the probabilities of the case, in other words that a reasonable presumption or a strong probability may shift the *onus probandi*, and, if not rebutted, may form the basis of judicial decision, is traceable to the civil law, and is fully recognized by the Roman-Dutch jurists."[31] To me, a sound test, which is located in the constitutional edifice, is what is just and equitable, given the proven probabilities. I dare say that is more in line with the constitutional values than to doggedly follow the archaic Eurocentric method of thinking of jurists such as Westenberg, Wigmore, and Voet, to name but a few.

(213) The *fonds et origo* of this case can be found in the varying versions which outline the probable aetiology for HIE suffered by F.B.W. The first version is that there was no sentinel event, as noted in the joint minutes of Pediatricians and Geneticists. In terms of this version, the catastrophe (HIE) would not have occurred had a C-section been prepared at 00h30, upon observing the horrendous CTG tracing.

(214) The second version is that there was an acute profound brain injury, as observed by the Radiologists in their joint minutes; consequently, there was very little the staff could do to change the outcome. Furthermore, a C-section would not have been of assistance since she experienced a rapid dilation from 4cm to 9cm at 02h05. By this period, it was too late to organise a C-section as an acute profound brain injury had occurred.

---

[30] Ley v Ley's Executors and Others 1951 (3) SA 186 (A) at 192-3.
[31] West Rand Estate Ltd v New Zealand Insurance Co Ltd 1925 A.D. 245 at 263

(215) At the centre of these first two versions are the terms acute profound, and partial profound brain injuries. Elucidating the differences between the two, the court in *NSS obo AS v MEC for Health, Eastern Cape Province*[32] said:

> "An acute profound HII must be distinguished from a partial prolonged HII. According to the reports by both experts, an acute profound HII is essentially a severe asphyxial event (deficient supply of oxygen) that occurs suddenly and progresses rapidly in term neonates, resulting in a primarily central pattern of injury involving the deep grey matter of the brain. The cause of an acute profound HII is generally referred to as 'a sentinel event'. Partial prolonged partial HII develops over a period of time, allowing compensatory redistribution of blood flow to occur, which results in a different pattern of injury to the white matter or peripheral structures of the brain."

(216) As already mentioned, Prof Smith testified this was not an acute profound brain injury, and he was not cross-examined on this issue. Dealing with cross-examination, the court in *President of the Republic of South Africa and Others v South African Rugby Football Union and Others*[33] held:

> "The institution of cross-examination not only constitutes a right, it also imposes certain obligations. As a general rule it is essential when it is intended to suggest that a witness is not speaking the truth on a particular point, to direct the witness's attention to the fact by questions put in cross-examination showing that the imputation is intended to be made and to afford the witness an opportunity, while still in the witness box, of giving any explanation open to the witness and of defending his or her character. If a point in dispute is left unchallenged in cross-examination, the party calling the witness is entitled to assume that the unchallenged witness's testimony is accepted as correct. This rule was enunciated by the House of Lords in *Browne v Dunn* and has been adopted and consistently followed by our courts."[34]

---

[32] (017/22) [2023] ZASCA 41; 2023 (6) SA 408 (SCA) (31 March 2023
[33] (CCT16/98) [1999] ZACC 11; 2000 (1) SA 1; 1999 (10) BCLR 1059 (10 September 1999
[34] Supra para 61.

(217) In the joint minutes, the Paediatrician agreed that there was no recorded perinatal sentinel or catastrophic event.

(218) The third version, which was highlighted by the midwives and Dr. Mbokota, is that several conceivable postulations could have played themselves out if, at 20h00 the Maternal Guidelines were followed, and membranes were ruptured.

(219) On a balance of probabilities, the test of negligence as outlined in *Kruger v Coetzee* has been met. The next question is whether the negligent act or omission caused the harm giving rise to the claim. Had the midwives followed the protocol at 20h00 would the injury have occurred? *In Ocean Accident and Guarantee Corporation Ltd v Koch,*[35] the court said:

> "The fact that, scientifically speaking, the aetiology of the disease is uncertain, does not hamper the Court in deciding on the facts and on the expert evidence adduced in a given case, whether a likely cause was proved in such case. Judicial decisions reflect the particular facts and testimony of each case and are not intended and cannot be regarded as scientific treatises. Accordingly, the possibility of future scientific disproof of the opinion of one or the other expert medical witnesses is, judicially, a matter of no moment- the court must do the best it can on the material presently before it in each case."[36]

(220) For a court to draw a proper inference in civil matters, the inference sought to be drawn must be consistent with all the proven facts; secondly, on a balance of probabilities, the court must "select a conclusion that seems to be more natural or plausible from amongst several conceivable ones, even though that conclusion be not the only reasonable one."[37] Had the Maternal Guidelines been followed, it is plausible that she would have remained at 3cm and even after the administration of oxytocin. A C-section would have been prepared within an hour; per the midwives'

---

[35] 1963 (4) 147 [A.D.]
[36] Supra page 159 F
[37] Supra page 159 C

version, it would have been ready at 10h30, even after factoring in an extra hour for the doctor's arrival and examinations of the patient, and per Dr. Mbokota's testimony, it should have been ready at 00h30 On a balance of probabilities, this is the most plausible conclusion, given that at 00h19 ROM did not lead to cervical dilation beyond 3cm.

(221) The constitutional court in *Lee* said that:

> "56 Even if one accepts that the substitution approach is better suited to factual causation, the preceding discussion shows that there is no requirement that a plaintiff must adduce further evidence to prove, on a balance of probabilities, what the lawful, non-negligent conduct of the defendant should have been. All that is required is "the substitution of a hypothetical course of lawful conduct and the posing of the question as to whether upon such a hypothesis the plaintiff's loss would have ensued or not".116 What is required is postulating hypothetical lawful, non-negligent conduct,117 not actual proof of that conduct. The law recognises science in requiring proof of factual causation of harm before liability for that harm is legally imposed on a defendant, but the method of proof in a court room is not the method of scientific proof. The law does not require proof equivalent to a control sample in scientific investigation."

(222) The defendants' counsel submitted that this matter resembled *MEC of Health and Social Development of the Gauteng Provincial Government v M.* I do not agree. First, in M, the partogram was plotted correctly, and the mother was properly monitored using Doppler. Second, in M, there was progress in labour; hence, it is recorded that she was in the active phase of labour at 07h30. When the "Foetal distress was diagnosed at 14h30 and Ms M was prepared for a caesarean section. However, she became fully dilated at 14h45, after being wheeled into theatre, and she delivered L naturally at 15h10." In *casu,* a cesarean section was not prepared, notwithstanding the following:

1. The presence of MSL.
2. Non-reassuring CTG tracing (a horrendous tracing).

3. Lack of progress in labour despite ROM (3cm cervical dilation).
4. Failure to comply with Guidelines (8-hour deadline missed).
5. Delay by the doctor.
6. Failure to plot the partogram.
7. Failure to follow the doctor's instructions.

(223) I am alive to the presence of other conceivable postulations, such as the cervix would have dilated and she could have given birth naturally, or that a precipitous delivery could have occurred. Even a sentinel event could have occurred.

(224) On a balance of probabilities, and having regard to the but-for test, the plaintiff has proven the causal nexus between the HIE and the negligence of the defendants. On a balance of probabilities, I am convinced that had the staff followed the Guidelines and called the doctor before 00h00, the CP would not have occurred. But for their failure to prepare for a C-section after 20h00 at the earliest or after 00h20 at the latest, the catastrophic result would not have occurred. By the time ROM was performed, the die had already been cast; expedited delivery was imperative. Indeed, the plaintiff correctly heard the doctor say she should have long delivered, and certainly, smoking was not the smoking gun in this case. Consequently, the plaintiff has proven the causal nexus.

Costs

(225) It is trite that costs follow the results. I do not think a case for costs on a punitive scale has been made. In the result, I make the following order:

Order

1. The Defendants are jointly and severally ordered to pay 100% of the Plaintiff's agreed or proven damages consequent on the hypoxic ischaemic cerebral damage sustained by F.B.W. (ID NO…) born on 27 February 2012 that has resulted in cerebral palsy.
2. The Defendants shall pay the Plaintiff's taxed or agreed costs of suit to date on party and party scale C.

3. The costs shall include the following:-

3.1 The costs of the liability trial;

3.2 The costs attendant upon obtaining the medico-legal reports including addendum reports, preparation, consultations and reservation fees, if any, of the plaintiffs' experts, namely: -

3.2.1 Dr.R.F.Scott

3.2.2 Prof. S. Andronikou

3.2.3 Dr. G.S. Gericke

3.2.4 Dr.W.Burger

3.2.5 Dr.D.Pearce

3.2.6 Prof.D.DuPlessis

3.2.7 Dr.G.S.Gericke

3.2.8 Prof.J.Smith

3.2.9 Prof.E.J.Coetzee

4. The appearance costs of Prof. D. du Plessis, Prof. E.J.Coetzee and Prof. J. Smith.

5. The costs consequent upon the employment of counsel including the costs of consultations, preparation, attendances at pre-trial conferences, drafting, preparation of heads of argument, appearances on trial and argument.

6. The costs consequent upon the employment of counsel on Scale C.

7. The costs of the plaintiffs' attorneys of record subject to the discretion of the Taxing Master in preparation for trial, travelling costs, and attendance at court.

8. The reasonable costs of the Plaintiffs to attending the medico-legal examinations of both parties.

9. Costs consequent to the preparation of trial and witness bundles.

10. Costs of holding pre-trial conferences, the parties having specifically agreed that the costs of Counsel were necessarily incurred.

11. The notice of taxation shall be served on the Defendants' attorneys of record.

12. The Defendants shall pay interest at the prescribed rate on the Plaintiffs taxed or agreed costs of suit calculated within thirty-one days

after agreement or from the date after affixing of the Taxing Master's allocatur to the date of final payment.

13. Any payment due in terms of this order shall be paid into the Trust account of the plaintiffs' attorneys of record:

    Ivan Maitin Attorneys Inc,
    First National Bank,
    Account no: 6[…],
    Branch code:250 655.

14. The determination of quantum and the issue of prescription of the Plaintiffs' personal claims are postponed sine die.

_____

M.P. MOTHA

JUDGE OF THE HIGH COURT, PRETORIA

Date of hearing: 26 August 2024 – 06 September 2024; 15 & 17 October 2024 and 14 February 2025

Date of judgment: 26 June 2025

APPEARANCES:

For the Plaintiffs: Adv M. Patel instructed by Ivin Maitin Attorneys Inc.

For the defendants: Adv S. Malatji, Adv. L. Rakgwale and Adv. M. Mpama instructed by The State Attorney