

IN THE HIGH COURT OF SOUTH AFRICA
(EASTERN CAPE LOCAL DIVISION, BHISHO)

CASE NO: 307/2018
Date Heard: 27/05/2021
Date Delivered: 14/09/2021

In the matter between

PHUMZA FUNDA obo LINGOMSO FUNDA

Plaintiff

And

**THE MEMBER OF THE EXECUTIVE COUNCIL FOR THE
DEPARTMENT OF HEALTH – EASTERN CAPE**

Defendant

JUDGMENT

SMITH J:

Introduction

[1] The plaintiff claims damages in her personal and representative capacities as the mother and natural guardian of Lingomso Funda, a male infant born on 8 February 2016 at the All Saints Memorial Hospital, Stutterheim, by means of normal vaginal delivery.

[2] The plaintiff's claim is founded upon the alleged negligence of the medical or nursing staff of the hospital, which allegedly caused the child to suffer a hypoxic ischaemic brain injury during birth, resulting in cerebral palsy.

[3] The issue that falls for decision at this stage is only the liability of the defendant and consequently whether, on a balance of probabilities, negligence of the staff during the intrapartum period caused the brain injury and the eventual cerebral palsy.

[4] Apart from the plaintiff herself, Dr Kara (paediatrician); Prof Davies (paediatric neurologist), Dr Chimusoro (obstetrician and gynaecologist); Prof Andronikou (paediatric neuro radiologist) and Dr Pearce (paediatric neurologist), were called to testify on her behalf. The defendant called nursing sister Nonkolisko Elsie Dilla; Dr Ntshokota; Dr Koll (obstetrician and gynaecologist) and Prof Rothberg (paediatric neonatologist) testify on her behalf. Mr J. Wessels SC and Mr P. Uys appeared for the plaintiff and Ms G.M. Goedhart SC and Ms H Cassim appeared for the defendant.

The pleadings

[5] The plaintiff pleaded that the foetus suffered a hypoxic ischaemic brain injury during the intrapartum period, and more particularly while under medical care at the hospital. The brain injury allegedly resulted from substandard care by the doctors or staff who failed or neglected correctly to assess or monitor the maternal and foetal condition during birth, and to act appropriately on such assessment or monitoring. She furthermore averred that appropriate assessment or monitoring would probably have confirmed foetal distress and the necessity to intervene through either intrauterine resuscitation or caesarean section.

[6] The defendant in her plea denied that the hospital staff were negligent or that their negligence caused or contributed to Lingomso's cerebral palsy. She averred furthermore that the plaintiff arrived at the hospital at approximately 09h00 on 8 February 2016, when she was entered into the maternity register. She was then admitted to the labour ward and examined at approximately 09h20. She had told the nursing staff that she had gone into labour at 14h00, alternatively 16h00, on 7 February 2016. The plaintiff and the foetus were monitored at hourly intervals from 09h20. The foetal heart rate fell within normal limits at all material times. Lingomso was born at 12h50 on 8 February 2016, with Apgar scores of 9/10 and 10/10. The plaintiff and Lingomso were discharged 9 February 2016.

The extent of the injury

[7] Before I commence with a summary of the evidence, and in order to provide proper context for the scientific hypotheses and conclusions advanced by the parties' respective experts, it will perhaps be helpful if I explain the agreed nature, extent and mechanisms of Lingomso's brain injury.

[8] The experts agreed that the injury was caused by hypoxia and ischaemia, resulting in a mixed (partial prolonged and acute profound) brain injury with features in keeping with hypoglycaemia. The injury occurred over a period of hours.

[9] The MRI study depicts a mixed pattern of brain injury (partial prolonged and acute profound) with injuries to the deep grey nuclei (pulvinars), white matter, watershed and main arterial areas at the peri-sylvian region and specific features of ulegyria, typically associates with hypoxia and ischaemia.

[10] The mechanism of this injury was described as follows by the experts:

- (a) the foetal compensatory mechanisms protect the foetal brain against hypoxia and ischaemia by mopping up the acids generated in the tissue to correct the deficits. When the hypoxia and ischaemia persist, the body moves to the next phase, being redistribution of blood flow from the less vital organs (for instance the skin) to preserve blood flow to the brain. With progression of this phase, blood will be shunted away from the metabolic active parts of the brain to the deep nuclei to sustain the vital organs of life, resulting in the outer areas of the brain developing a so-called watershed infarct;
- (b) this is described as a partial prolonged hypoxic ischaemic injury, where the deeper areas of the brain remain preserved. This injury will manifest through foetal heartrate, abnormalities, coinciding with uterine contractions;
- (c) an acute profound injury involves necrosis of the deeper brain structures and may either occur through a sudden catastrophic or sentinel event (for

instance cord prolapse or uterine rupture) or more commonly through multiple partial prolonged episodes with eventual cumulative effect resulting in an acute profound injury. This can result in a mixed partial prolonged and acute profound type of brain injury. Once again, the injury manifest through foetal heartrate abnormalities, coinciding with contractions; and

- (d) while isolated heartrate measurements merely confirm foetal heartrate, foetal heartrate assessment timed to contractions, informs of foetal well-being, particularly when associated with deceleration.

The Maternity Care Records

[11] The records contain the following entries:

- a. The first stage of labour started at 16:00 the day before the birth, lasting some 20 hours 20 minutes. The second stage occupied 30 minutes and the third stage 5 minutes;
- b. The membranes were ruptured at 12h50 and the child delivered at 12h50, but the partogram records that the membranes were ruptured at 12h20;
- c. The monitoring of the labour, the birth, the postpartum management of the plaintiff and neonate was done by Sr. Dilla at 12h52;
- d. The cord was normal, the membranes and placenta complete and normal with 3 vessels in the cord and a placental weight of 520 g;
- e. No retro-placental clots were present and no histology required and no sentinel event was recorded;
- f. The neonate was delivered in a healthy and satisfactory condition with recorded Apgars of 9/10 and 10/10 and received in the ward at 12h50;

- g. The discharge summary recorded that feeding options were discussed and initiated successfully with exclusive breastfeeding to be conducted on demand;
- h. The plaintiff took the child to the clinic on 10 February 2016 due to twitching or fitting. The child was examined by a nurse and doctor, prescribed medicine, and discharged home; and
- i. Eventually the child was diagnosed with developmental delays and cerebral palsy.

Evidence on behalf of the plaintiff

[12] The plaintiff testified that when she learned of her pregnancy, she immediately commenced antenatal care and had four monthly antenatal assessments prior to birth. She was diagnosed with HIV during the pregnancy.

[13] By 7 February 2016, she developed back pain, and later she experienced abdominal pains, which subsided. The next morning the labour pains were severe and an ambulance was called. She was prepared for hospital, where she arrived after 7am. There a nurse registered her and conducted an internal examination, assessing the foetal heartbeat with a funnel like object. The nurse then told her to wait for a doctor.

[14] When the doctor eventually arrived, he conducted an internal examination, assessed the foetal heartbeat with a funnel like object, “spoke about centimetres” and advised her that she would be taken to another ward. There the nurse instructed her to lie on a bed and breathe in and out. The nurse then left her alone and told her to call her if the pain became more severe.

[15] She called the nurse once or twice when the pains were very severe. The nurse then exerted fundal pressure on the upper part of her stomach, ordering her to push. This eventually resulted in her giving birth.

[16] The baby did not cry and the nurse had to hold him upside down and pat him on the back. He, however, still did not cry. The nurse did not assess the child but wrapped him in a blanket and placed him next to her. She was thereafter sutured, stepped down onto a wheelchair, and the child placed on her lap. They were then both taken to the ward.

[17] The ward sister instructed her to breastfeed the child, but he could not latch onto the breast. The sister then brought the plaintiff milk in a cup and instructed her to cup-feed the child, which she managed to do. Thereafter the child slept for approximately one hour. He cried a bit after he had woken up,. They then both slept until the next day when they were given some cards and discharged without being examined.

[18] The child was cup-fed at home. He, however, developed seizures the next day and was taken to the Mtanyana Clinic, where she was given a letter for hospital referral.

[19] At the hospital a doctor examined the child, gave him Panado Syrup (it is common cause that it was probably Phenobarbitone) and thereafter send them home. The child again suffered seizures on the 10th, which subsided the following day. She later returned the child to the clinic on a regular basis.

[20] She first realised that something was wrong with the child at approximately 10 months of age when he could not sit, speak or hold onto anything. He is currently still unable to do anything for himself. He only sleeps and can neither sit, stand or talk.

[21] During cross-examination, Ms Goedhart put to the plaintiff that Sr Dilla first assessed her after admission at 09h20. She further put to the plaintiff that the hospital records state that sister Dilla had assisted her at 09h20, 10h20, 11h20 and 12h20. She was assessed by a doctor at 09h30. The assessments were conducted by cardiotocography (CTG). The plaintiff, however, maintained that she was assessed only once by Sr Dilla and a doctor, and that no belt was placed over her abdomen at any time.

[22] Ms Goedhardt also put to the plaintiff that Sr Dilla would testify that she made certain entries on the Maternity Care Records (MCR) after the document had been requested by the plaintiff's attorneys. She, however, made those notes contemporaneously on the day, but had failed to record it. She thus admitted that she had altered the hospital records after birth, but would maintain that she had merely recorded what she had done on the day.

[23] It was also put to the plaintiff that Sr Dilla had examined the child to tick all the boxes and that the references to 12h50 is a mistake, as she would have done so after birth. The child looked well upon discharge and was feeding.

[24] The plaintiff, however, maintained that the ward sister had instructed her to breastfeed, but the child was cup fed with formula due to failure to latch. Bottle-feeding commenced only after a month. She also maintained that the seizures commenced only on the morning of the 10th and continued thereafter.

[25] Doctor Kara, a paediatric expert, testified that upon examining Lingomso he had found the child to be micro-cephalic, with probable mixed type of cerebral palsy, GMFCS V, spasticity, dyskinesia, abnormal movements, abnormal tone, limited communicative ability and epilepsy. The child also presented with myoclonic movements (which are random sudden jerks) and no dysmorphic features or neurocutaneous lesion. He had poor swallow reflexes, low central tone and variable peripheral tone. He does not suffer from a progressive disorder.

[26] He had regard to the need for the stimulation at birth, the reported history of poor sucking reflex and the convulsions on 10 February 2020. His testimony that moderate encephalopathy was present based on the moderate seizures was not disputed. He concluded that the convulsions were probably the result of intrapartum hypoxia and that the child should have been admitted to hospital. He testified furthermore that the plaintiff's history and the antenatal record exclude any antenatal or pre-conceptual risk factors for cerebral palsy, aside from HIV.

[27] The MRI indicates that the injury did not occur prior to 36 weeks gestation and HIV could likely be excluded as the cause of his cerebral palsy. HIV positive mothers

do have approximately 50% increased risk of delivering a baby with encephalopathy after birth. HIV does not cause the injury itself, but it makes the foetus more susceptible to hypoxia.

[28] The child had normal anthropometrical features upon birth, in keeping with a baby of term gestation and there is no reason to suspect growth restriction or growth asymmetry. This conclusion and the mother's report in respect of foetal movement upon admission to hospital, make it less likely that there was any antenatal insult to the foetal brain.

[29] The normal head size at birth with microcephaly postnatally, suggests that the a perinatal insult affected normal foetal brain growth after birth. The foetal condition seemed to be good when the mother was assessed at the hospital at 09h00 on 8th February 2016 (based on history and assessment) and when the mother was assessed by the nurse and the doctor.

[30] He said the mother's description of the baby not being able to suck, the presence of the weak cry, cup feeding and early discharge, are concerning, as feeds needs to be established prior to discharge. These factors are compatible with neonatal and encephalopathy in the first day of life. The convulsions occurred within 2 days after birth and the baby had a poor history of sucking. These factors point to encephalopathy in the first days of life. It appears that the staff did not detect it, or even ignored it when brought to their attention. The convulsions necessitated immediate hospital admission and discharging the neonate was thus unjustified.

[31] He excluded other possible causes of neonatal and encephalopathy at birth, namely, congenital normalities, metabolic illness or syndrome, history of progressive neurological deterioration, or that the child initially reached milestones but subsequently fell behind in respect of such milestones.

[32] Regarding the timing of the injury, Dr Kara testified that there is little evidence to support an antenatal insult. The convulsions on day two is more likely to be the result of brain injury during birth. Hypoglycaemia was probably the result of poor feeding due to encephalopathy and possibly caused the convulsions. The most

common cause of encephalopathy is hypoxic ischaemic insults, with the highest risk of such occurring during labour. The most common cause of convulsions in the first three days of life in a term infant is hypoxic ischaemic encephalopathy (HIE).

[33] In his view, although the Apgar score was normal and there is no record of resuscitation at birth, the factors that point to the probability that the encephalopathy was due to an intrapartum result are; encephalopathy, the results of the MIR and the presence of dyskinetic cerebral palsy.

[34] According to him, the probability is 80% that the injury to the brain occurred during labour. In addition, the MIR scan confirms hypoglycaemia, which is linked to intrapartum hypoxic ischaemia. Hypoxia and ischaemia depletes stores of glucose that is used to produce energy to sustain cellular activity. In addition, the child had postnatal microcephaly with no evidence of antenatal or postnatal injury. He also had overt neurological symptoms of poor feeding and crying, convulsions in the first 36 to 48 hours of life, with no record of assessment prior to discharge.

[35] In his view the following factors favour the probability that the encephalopathy resulted from intrapartum injury; the undoubted presence of encephalopathy; the MIR scan confirming hypoxic ischaemic injury (with mixed pattern of prolonged partial and acute profound), favouring the probability that the injury occurred during the labour (noting reasonable exclusion of other causes of encephalopathy); the presence of dyskinetic cerebral palsy; and features of acute profound HIE.

[36] A history of encephalopathy after birth makes it an 80% probability that the injury occurred during labour. A further factor is the MIR features of hypoglycaemia and a link between neonatal hypoglycaemia intrapartum HIE. The depletion of glucose caused by hypoxic ischaemic injury means that there are not sufficient stores of glucose left to sustain the high metabolic activity and high demand for energy which occurs after birth. A compromised baby with poor feeding and poor perfusion may not provide adequate glucose to the brain to prevent cellular injury, resulting in a neonatal hypoglycaemic brain injury.

[37] During cross-examination Dr Kara maintained that there is no history of maternal infections, intrauterine growth restriction or HIV related encephalopathy. He thus rated the possibility of antenatal brain insult as less than 10 percent.

[38] He stated that there is an 80% chance of intrapartum hypoxic ischaemic injury evidenced by dyskenetic cerebral palsy and an MIR picture of basal ganglia and thalamus brain injury. He also said that CTG's are sensitive but nonspecific, and CTG deceleration, especially late deceleration, is highly predictive of foetal compromise.

[39] Hypoglycaemia is probably a consequence of the cerebral injury that affected feeding, raised cerebral metabolism, increased glucose consumption, depleted the baby's glucose stores and caused a hypoglycaemic brain injury. As a result, the injury as such was not the cause of the brain injury.

[40] In the reply to questions put to him by the court, he testified that seizures make it more probable had the injury occurred intrapartum. He also said that despite some inconsistency in the records, the cerebral palsy was due to an intrapartum hypoxic ischaemic injury supported by the postnatal microcephaly and lack of evidence of antenatal or postnatal injury.

[41] Professor Andronikou testified that the injury occurred over many hours and that an extensive portion and important parts of the brain are involved. These include severe damage to the motor and sensory cortex (which allows a child to function). The internal capsules, which controls movement and the deep nuclei involved in the thalamus is also severely affected. A large portion of the brain, which includes the perisylvian, posterior watershed and the occipital areas are also damaged,

[42] Professor Davis testified that the child fits criteria for neonatal encephalopathy based on hypoxia and ischaemia with a Sarnat Grade 2 moderate encephalopathy, and as a result hypoxia and ischaemia should be considered as a cause of the cerebral palsy. He said that term babies born in depressed conditions in lower and middle income countries, usually result from intrapartum hypoxia or birth asphyxia. Reperfusion injury (the tissue damage caused when blood supply returns to tissue) is part of the main hypoxic ischaemic injury. Multiple potential causal pathways can

cause cerebral palsy in term infants. A broader perspective is necessary before attributing neonatal encephalopathy to an intrapartum event. It thus calls for a comprehensive multi-dimensional assessment of neonatal status and all potential contributing factors, including maternal medical history, obstetric antecedents, intrapartum factors and placental pathology.

[43] He explained that there are a number of compensatory mechanisms available to a foetus, and the basic mechanisms of the hypoxic ischaemic injury or hypoxic ischaemic encephalopathy are:

- (a) the so-call buffer stage where, because of hypoxia ischaemia to tissues, the tissues generate acids. The body then buffers or mop up these acids, attempting to correct the deficit. This is a stage where there is not likely to be neurological damaged to the foetus. However, if the hypoxia ischaemia persists, then the next phase occurs, which is redistribution of blood-flow in the foetus, when blood is then divided from so-called less vital organs, like the skin, to the brain to try to preserve blood flow to the brain in order to preserve life in the brain stem itself;
- (b) the blood is diverted from the cerebral cortex (outer regions of the brain) to the central areas, resulting in deprivation of oxygenated blood to these areas with infarction or cell necrosis, (i.e. partial, prolonged hypoxic ischaemic damage);
- (c) watershed infarcts occur during uterine contractions and can be repeated over a lengthy period (even several days), and these partial prolonged insults manifest through foetal heartrate abnormalities, particularly during uterine contractions;
- (d) acute profound injuries involve necrosis in deeper brain structures and present either as a sudden catastrophic or sentinel event (i.e. cord prolapse or uterine rupture) or a build-up process of multiple partial prolonged episodes with the final episode resulting in a cumulative effect, which results in turn in a mixed acute profound and partial prolonged picture;

- (e) partial prolonged insults manifest through changes in foetal heartrate and foetal condition, and appropriate intervention can successfully be instituted through expedited delivery; and
- (f) generally a foetus will suffer brief hypoxic bouts during labour, which are normally well tolerated during the first buffer stage. Continuation beyond the buffer stage results in partial prolonged or watershed injury, and eventual acute profound injury.

[44] He questioned the quality of foetal monitoring during the later phase of labour when the plaintiff presented to hospital, and was of the opinion that foetal distress was either missed or overlooked. In his view, had the plaintiff been given reasonable intrapartum obstetric care and the delivery expedited, the hypoxic ischaemic asphyxia injury of a combined partial prolonged and acute profound nature, neonatal encephalopathy and consequential cerebral palsy, would probably have been prevented.

[45] During cross-examination Prof Davis maintained that the seizures at 48 hours of life indicate moderate to serious encephalopathy. He said that seizures are often delayed in HIE. In his opinion, the injury most probably occurred during the intrapartum stage and most probably resulted from substandard intrapartum obstetric care.

[46] He said that intrapartum hypoxia is the most probable causal factor of neonatal encephalopathy, and substandard intrapartum obstetric care the predictable and preventable causal factors. In his opinion reasonable intrapartum obstetric care and expedited delivery would probably have prevented the hypoxic ischemic injury, neonatal encephalopathy and subsequent cerebral palsy.

[47] Doctor Chimusoro, a paediatric specialist, testified that having had regard to the hospital records, he was of the view that the foetal heartrate assessments were only done hourly instead of every 30 minutes, as required by the guidelines. In his view the partogram was obviously not completed contemporaneously, but probably after the event.

[48] The CD 4 count (a test for a type of white blood cell that helps the body to prevent infections) and the viral load was not recorded. There is a difference in the management of mothers with or without suppressed viral load. No serious ailments were noted, and the treatment prescribed for HIV seemed adequate.

[49] In his view the normal and anthropometrical medical features reasonably exclude intrauterine growth restriction.

[50] He said that the delay in assessment from 09h20 constitute substandard care. The intensity of contractions increased as the labour progressed. The foetus should therefore have been more closely monitored during the second stage of labour when the contractions became stronger, and the staff had to ensure that those strong contractions did not result in hypoxia. In his opinion, this is why the foetus had to be monitored every half hour during active phase of labour and after second contraction in the second stage (10cm dilatation). He criticised the records for not referring to the maternal and foetal condition and the progress of labour itself, and the staff for not recording the foetal heartrate every half hour.

[51] In his view, the recorded foetal heartrate was probably recorded *ex post facto*. It is highly unlikely that the foetal heartrate would be similar before and after contractions. Proper auscultation in terms of the guidelines entails foetal heartrate assessment before and after contractions. Decelerations would have been identified through proper auscultation, and could have been attended to through intrauterine resuscitation, intravenous fluids and medication to relax the uterus and contractions. Other possible causes of distress could have been identified and excluded. In his view, the partogram entries between 10h50 and 12h50 and contractions were drawn in one movement and raise suspicion.

[52] The staff had to assess the foetal heartrate before and after contractions. This is done to determine whether the foetal heartrate recovers after the contraction. Should foetal distress occur, intrauterine resuscitation should be conducted, which includes putting the patient on the left side, administration of fluids, oxygen and medication to relax the uterus to stop contractions, and determining whether there are other reasons for foetal distress, like a cord prolapse.

[53] He testified that in his opinion the foetal heartrate should have been charted at every second contraction or every five minutes during the second stage of labour. The records show that monitoring was not done effectively. Consequently, the substandard intrapartum care missed a hypoxic event. He also criticised the staff for not recording the CTG findings, even in the absence of tracing paper.

[54] During cross-examination, he explained the distinction between HIV and HIE types of encephalopathy, and said there was no evidence of optimistic infections. He also commented on the specific substandard care, stating that the condition of the foetus should have been assessed through auscultation when plaintiff was bearing down.

[55] He criticised the record keeping , explaining that the method of recordal casts doubt on whether it was truly done.

[56] In addition, he maintained that the intrapartum monitoring was substandard and had missed a hypoxic event. In his view, the foetal condition was apparently normal upon admission and the injury therefore probably occurred during birth at the hospital. In his opinion, the Apgar scores are probably incorrect and contradicted by neonatal encephalopathy and the true condition of the child.

[57] In response to a question put to him by the court, doctor Chimusoro explained that if the hypoxic event had occurred prior to the admission it would have showed in an abnormal foetal heartrate pattern. Because the heartrate was normal on admission, the hypoxic event could only have taken place after admission. If the foetal heartrate pattern is normal at admission, then it means that there has not been any brain insult, because the brain regulates the way the heart beats and how the variation in the heart changes. If the damage had occurred before she was taken to the hospital, it would have been evident from the foetal heartrate at the time of admission.

[58] In his view a hypoxic event with recovery, without any intrauterine resuscitation, is highly unlikely. It is also obstetrically unlikely that whatever insult was causing the baby to be hypoxic would spontaneously correct. Thus, he excluded antenatal brain injury due to normal heartrate patterns upon and immediately after

admission. He said that the intrapartum monitoring was clearly substandard and had missed obvious foetal distress.

[59] Doctor Pearce testified that she had assessed the child clinically and found him to be micro-cephalic, which is indicative of lack of brain growth, with no other conditions mimicking cerebral palsy. Her clinical findings correlate exactly with the MRI features of the child. The history obtained from the plaintiff fits the criteria for a Grade 2 neonatal encephalopathy, resulting from intrapartum hypoxia and ischaemia.

[60] She excluded other possible and probable causes for NE II, like infection, congenital brain abnormalities, substance abuse and medication. She had also considered and excluded other probable causes for cerebral palsy including:

- (a) intrauterine growth restriction, as the anthropological measurements were normal for gestation; and

- (b) intracranial haemorrhage (which is also excluded on MRI), inborn errors of metabolism and genetic causes.

[61] In her opinion, the child has a mixed type of cerebral palsy, predominantly dystonic with superimposed left hemiplegia. He is classified as GMFCS V (which is the worst level of impairment for cerebral palsy; MACSV (manual ability classification scale); and CFCSV (communication function classification scale).

[62] She said that the MRI, the history obtained and the clinical records support the child's diagnoses. His condition is most likely the result of peripartum hypoxia, as founded upon assessment of antenatal, intrapartum and post-natal possible causes of the encephalopathy and outcome. HIV probably played no role in the outcome as the plaintiff was at stage 1 of HIV, with an unknown CD 4 count and no indications of maternal infection, illness or intrauterine growth restriction.

[63] She testified that hypoxic ischaemic injury is divided into primary and latent energy failure, with the primary failure being the initial insult. During the latent phase, the child seems to recover, but the reperfusion of blood can result in a reperfusion

injury. It is possible that the child may be born in the latent phase of injury, with resultant decompensation resulting in neurological damage.

[64] The neonatal seizures were a significant concern. The child had to be fully examined and admitted. The seizures had probably exacerbated the brain injury in an already fragile infant and contributed to his condition.

Evidence on behalf of the defendant

[65] Sister Dilla is a registered nurse and has been employed at the hospital since 2012. She testified that she managed the labour and birth and completed the MCR and made certain corrections thereto. An auxiliary nurse made a single time entry.

[66] She first assessed the plaintiff at about 09h20, when she did a vaginal examination and monitored the foetal heart rate by CTG. The foetal heartrate remained in the normal range between 110 – 160 beats per minute. The labour had progress well, no foetal distress was present, there was no resuscitation required after birth, and the child was generally well.

[67] In regard to the initiation of breastfeeding, she testified that she had put the child on the mother's breast and he immediately started to suck her fist. The mother also tried to make him breastfeed when she was busy suturing her. Feeding was discussed and initiated successfully, with the plaintiff reporting no problems.

[68] She disputed the plaintiff's assertions that she only assessed her once on the day of the birth; that the child did not cry upon birth (stating that in the absence of crying she would have cut the cord and called for help to resuscitate the baby); and that the child did not suck from the breast, stating that the child showed intention by sucking his fist while she was drying and weighing him.

[69] During cross-examination Sister Dilla testified that the maternity ward averages five births per day.

[70] She received general training on the guidelines for maternity care during her diploma studies, specialist training on CTG during her advanced midwife course (March 2016, which was after the birth of the child), but no training on the neurology of neonates.

[71] She had no independent recollection of the birth, and was wholly dependant on the MCR in reconstructing it. She described the birth as just a normal birth “like thousands of others”.

[72] She confirmed that proper recordkeeping assists others to ascertain the condition of the unit (mother and child) during birth; identifies assessments which were not done; CTG interpretation and result should be recorded to provide lost tracking; and that all tracings should be retained and stored. She also conceded that monitoring of the foetal condition during birth identifies and avoids foetal distress.

[73] She said that the hospital did not have CTG paper on the day. She therefore assessed the foetal condition and heartrate on the screen of the CTG machine, supplemented by abdominal palpation to determine contractions. She used fetoscope auscultation to supplement CTG assessments.

[74] She, however, later on stated that she used the fetoscope during the first assessment and then continued with CTG assessment. She claimed CTG assessments were used from 09h20 for 30 minutes at a time. She forgot to note the CTG assessment or record the findings, but recorded the heartbeat in her partograph.

[75] She said that foetal distress is associated with decelerations not continuing after contraction or either bradycardia or tachycardia. Short-term foetal heartrate variability and late decelerations are indicative of a good foetal condition. When she was asked whether a healthy foetus also shows decelerations with contractions, she responded that “it can if there is contractions”.

[76] Mr Wessels put to her that she has a limited understanding of what the CTG shows, even after her training on CTG. According to doctor Koll the interpretation of a CTG is difficult and requires training. By the time she claimed to have used the CTG,

she had not yet received the training. Dr Koll testified that without paper one cannot assess the relationship between the contractions and the foetal heartbeat.

[77] During cross-examination Sr Dilla was also confronted with the *ex post facto* amendments to the original records. She maintained that she completed the records on the day of the birth.

[78] She testified that the entry “urine NAD” was noted when she assessed the patient. However, it was clear from the record that this was written in afterwards. She also said that certain new entries were not made by her when it is clear from the record that the handwriting was similar and probably hers.

[79] She was also unable to explain another entry namely, “Sister Tswago receiving the neonate” and the discrepancy with the original document.

[80] When it was put to her that alterations were made by her to validate the contents and contemporaneous nature of the records, she responded that she had no access to the record after discharge and there should only be one record for each birth.

[81] She was also confronted with various errors and mistakes in the record, inter alia:

- (a) the time and date which were not entered in the correct blocks. Commencement of labour pains was initially recorded as 14h00, but eventually amended to 16h00, being the correct time of onset of severe labour pains;
- (b) recording the contractions as being moderate, but plotting the same as weak on the partogram. She admitted the plotting was delayed by other work and waiting for the doctor to complete his examination of the plaintiff;
- (c) the plaintiff did not have mild contractions at 10h20 and the contractions were plotted continuously when the birth book was returned to her from the

doctor. She denied continues plotting of the strong contractions plotted at 11h20;

- (d) the foetal condition was not recorded at 10h20 as observations were done hourly and not half hourly in terms of the protocol;
- (e) the recorded foetal heartrates at 11h20 (140 and 134 bpm) constituted a foetal heartrate deceleration after a contraction;
- (f) the “summary of labour” entry on full dilatation was only recorded approximately 30 minutes after the delivery and care of the plaintiff and the child and scratched over when she re-calculated the duration of the birth;
- (g) she initially gave evidence that the time of onset of labour was obtained from the plaintiff after birth, then, that this time was obtained at 09h20 but recorded after birth, and finally that the time was changed after she again asked the plaintiff regarding her commencement of labour pains;
- (h) changes in the time of birth from 12h30 to 12h20 was attributed to a mistake or misprint occurring after calculating the duration of birth until full dilatation. She denied that the entry of bearing down at 12h25 fits better with full dilatation at 12h20 then 12h30;
- (i) she admitted that the records were not recorded contemporaneously, but after she cared for the mother and child and resulted in inaccurate times being noted;
- (j) discrepancy on the time of rupture of the membranes, namely 12h50 as opposed to 12h20 in the partogram, resulted from her attending to and confusing patients and findings and relying on her memory exclusively;
- (k) she did not listen to the foetal heartrate after every second contraction during the second stage of labour, but conducted continuously CTG;

- (l) generally and on the day she did not apply dedicated Apgar scoring, but instead made a general assessment while handling the child and recorded same after the assessment of the neonate and summary of labour forms;
- (m) the “First Examination of the Neonate” form was completed after admission of the mother and child to the post-natal ward and after attending to other births and routine issues in respect of the child. She relied on her memory and confused the gender of the child as this was not recorded contemporaneously;
- (n) she could not explain the meaning of tests to be conducted to determine Moro-reflex or muscle tone, referring thereto as features of the skin. She was also unable to distinguish hypotonic from hypertonic, also referring thereto as features of the skin; and
- (o) the entry relating to Sr Tshwago at 12h15 confirms that the latter had examined the child.

[82] Although she disputed that the child was cup fed, she eventually stated that she had seen the child being breastfed. She also said that she had remained with the neonate and the plaintiff for up to 40 minutes after birth.

[83] Doctor Koll is a practicing gynaecologist and obstetrician. He testified that despite the late antenatal presentation, foetal wellbeing could be determined through assessment of the height of fundus, foetal heart assessment, observing the maternal wellbeing and the standard observations conducted during antenatal visits. The birth anthropometrical measurements indicated no evidence of prematurity or post-dated birth and gestational age is not a factor in this case.

[84] He said that it is very difficult to estimate the onset of active labour and there is no significance in the time of onset of the contractions. The most important time to assess foetal heartrate it is before and after a contraction to exclude a very late deceleration. Hospital staff are trained to assess and identify decelerations.

[85] He conceded that the foetal heartrate was not monitored half hourly and that this constitute substandard care. A CTG machine measures beat-to-beat foetal heartrate and the numbers on the LED screen would change constantly. After CTG interpretation the findings should be written down to provide for lost CTG tracings. The CTG has four critical findings, but requires paper to be running to determine those. He said that Sr Dilla was apparently using the machine more as a labour saving device.

[86] For practical purposes foetal heartrate is all that is available to determine foetal wellbeing. If a foetus becomes hypoxic, the receptors in the cardiovascular system respond by slowing the heartrate to reduce oxygen usage. Decelerations, which start and end with contractions on its own, has no clinical significance. Late decelerations start after the apex of the contraction and recovers after the recovery of the contraction, and are suggestive of hypoxia. Variable decelerations are more difficult to interpret and tend to drop sharper and recover quicker.

[87] He said that variable decelerations are divided into typical and atypical decelerations, with atypical variable decelerations having a shoulder on either side, maintaining variability through the deceleration (the beat-to-beat variability continues through the deceleration) and typically last longer than a minute. In the absence of shouldering or it lasting longer than a minute, there is a loss of variability through the deceleration, which is referred to as a typical variable deceleration.

[88] Reduction in foetal heartrate recorded on the partogram at 11h20 (140 beats per minute/134 beats per minute) is variability. Deceleration has a reduction of more than 50 beats per minute or produce a change in baseline of more than 6 to 15 beats per minute. More severe reductions exceed 25 beats per minute, which is considered excessive. The patterns of variability and decelerations are completely different.

[89] There is no practical way of recording the foetal heartrate during the second stage of the active phase of labour as the midwife attending to the birth, is gloved and sterile. The midwife, however, listens to the foetal heartrate with her hand held Doppler on mother's abdomen, but only with some difficulty.

[90] Recordings of foetal heartrate are only done subsequently in the summary of labour and identified through the presence of foetal distress. He said that he has never seen a recorded foetal heartrate during this phase of labour being made on a partogram.

[91] Assessment of cervical dilatation is subjective and dependent upon the impression of the assessor. The strength of contractions is measured in conjunction with the progress of labour. If the labour is not progressing then powers (the strength of uterine contractions), the passenger (the foetus), the presenting part (the part of the baby that leads the way through the birth canal), and the progress of labour, have to be assessed. If the labour is progressing at an acceptable rate, then the strength of the contractions are irrelevant, being deemed adequate and the progress of labour acceptable.

[92] Although he chose not to comment on the accuracy of the MCR, stating that a forensic expert would be required, he ventured that labour progressed well to normal vaginal delivery of “a live male infant, the weight and the Apgars are recorded”. Foetal distress is very unlikely, as the foetal heartrate recordings remained normal. His opinion and report were based on contemporaneous records that he was applied with; as he saw them and he had no bases to dispute or confirm those records.

[93] When hypoxia occurs in the latter part of labour, it does not simply come and go. During early labour it is theoretically possible that the baby suffers a hypoxic episode and recovers. Later in labour hypoxia is a progressive condition, with contractions increasing in strength and severity. The baby is under stress of the contractions for longer, while the head is descending, making the last part of the labour by far the most dangerous.

[94] If the baby has a hypoxic episode in the latter part of labour, the hypoxia progressively increases and is at its worst at the time that the baby is born, with the resultant and expected signs of hypoxia in the baby. A hypoxic baby is expected to be flat or atonic, lack reflexes and will have a low heartbeat with a blue coloration.

[95] During cross-examination, he testified that he has not delivered a baby for more than 2 years and last worked in public healthcare approximately 20 years ago. The institution where he works pre-dominantly does CTG assessments and auscultation is hardly ever conducted.

[96] He said that he did not assess or consider placental insufficiency at all and growth restriction played no role in this case. Chronic centile insufficiency probably caused no growth restriction. Any increase in HIV related hypoxia could make the foetus more prone to hypoxia during the labour process. The exact pathway in respect of cerebral palsy and HIV has not been determined, but it is believed to be an inflammatory process causing a placental problem with oxygenation and hypoxia in a baby. This would have been determinable through foetal heartrate assessment during birth.

[97] If the foetus suffered a hypoxic period, the hypoxia would have progressively increased with contractions, building up towards the end of the birth and would probably have resulted in a decelerating heartrate showing that the foetus is in distress. The hypoxia would increase and worsen as the birth progresses and would be identifiable through foetal heartrate abnormalities.

[98] The National Guidelines for Maternity care (the guidelines) find application and sets a basic minimum protocol. All clinical events during the labour and birth need to be recorded fully and properly. In terms of the guidelines, foetal condition should be assessed half hourly, before and immediately after contractions in the active phase of labour to assess deceleration or foetal distress. It is not sufficient only to listen after a contraction, as the onset of the contraction would remain unknown. Assessment of the baseline heartrate only is of very little of assistance.

[99] The danger of a hypoxic ischaemic injury lies primarily in late decelerations and not necessarily in the presence of tachycardia or bradycardia. A foetus with a normal heartrate between 110 and 160 beats per minutes can be in severe distress. A heartrate should be seen in context and not only as an isolated number.

[100] Tying up foetal heartrate with contractions requires palpatating the abdomen from commencement to end and recording the foetal heartrate in accordance with the contraction. The heartrate should be recorded before and after the contraction and any delay in recovering should be determined.

[101] He could not confirm whether the foetal heartrate was determined before and after a contraction in this case. When he conducted his pre-trial meeting with Dr Chimusoro, he accepted that the foetal heartrate was monitored by way of auscultation and had no information of foetal heartrate assessment by means of CTG.

[102] He testified that a CTG running with paper will show:

- a) variability, which changes from beat to beat. Reduced variability can be associated with problems on its own and if persisting, it must be acted upon due to possible pre-maturity, sleep, drugging or distress;
- b) baseline, which should range between 110 and 160 bpm, with readings below 100 and above 180 being abnormal, and readings between 100/110 and 160/180 being non-reassuring;
- c) decelerations; and
- d) acceleration, which is a reassuring feature.

[103] He said that hypoxic events do not necessarily confirm the presence of a brain injury. This results from the protective mechanisms available to the foetus. Brain injury only occurs once the protective measures are no longer effective.

[104] The acute profound type of injury usually occurs right at the end of the hypoxic event, and can present either as a single acute profound event or as a result of partial prolonged hypoxia, resulting in a mixed pattern. The partial prolonged insult can cause an acute profound injury.

[105] He furthermore testified that on CTG, the foetal heartrate is shown in relation to contractions and in the absence of paper only certain features can be determined, like a sudden or acute bradycardia. The interpretation of CTG can be particularly complex and lead to significant disagreements. For a nurse to determine abnormality in a CTG, she would require training in reading of the CTG.

[106] Assessment on the CTG without paper will only yield the same results as what is seen in hand held Doppler, being the heartrate at a specific point in time. This can only be tied up to the contraction through palpation with the hand to determine the relationship with the contraction, although certain CTG's also has a display indicating the pressure in the uterus, which can be tied up to the heartrate.

[107] The CTG has to run long enough to provide an overall picture of the foetal heartrate in order to determine whether the contraction and heartrate do not correlate. But, such assessment can only be properly conducted through a CTG machine with paper, to confirm foetal distress.

[108] He said that if the court accepts that the injury occurred intrapartum and the baby was deemed to be hypoxic at birth, he would accept the probability that foetal distress occurred intrapartum. However, he does not see the condition of the baby upon birth to be hypoxic. If the court accepts that there was hypoxia during the labour, culminated in a hypoxic baby being born, then the MCR cannot be correct.

[109] Professor Rothberg has worked in neonatology from 1980 until 1996, and in health policy development until 2005, whereafter he became the Head of the Wits School of Therapeutic Sciences. In formulating his opinion, he had considered the original and amended records and was of the view that the amendments to the record made no difference to his opinion.

[110] He testified that it was significant that most of the plaintiff's labour occurred before hospital admission, when monitoring was not possible and the injury occurred as early as 17 hours before delivery. Referring to the literature entitled "**A review of the conundrum of mild hypoxic ischemic encephalopathy**", he said that the child initially had hypoxic ischaemic encephalopathy Stage 1, and he therefore disagrees

with Professor Davis that this literature does not find application. The child's encephalopathy progressed to moderate on the third day, (stage 2). He said the features of mild encephalopathy might be difficult for a non-specialist to detect. Such a child may be hyper-alert and not lethargic, but may present with other problems, such as feeding problems. The literature was written to assist with diagnoses and treatment of hypoxic ischemic encephalopathy in the first 6 hours of life and to initiate cooling.

[111] Neurological examination is subjective in nature and abnormality may be subtle and difficult to discern. The timing of the insult affects not only the clinical presentation, but also the progression of the encephalopathy. In the early hours after a significant hypoxic ischaemic event, the majority of new-borns do not demonstrate clear signs of moderate or severe brain compromise. Yet, these abnormalities may develop in the ensuing hours to days. The time of the insult affects the clinical presentation as well as progression of neonatal encephalopathy.

[112] Although the timing of the insult is predominantly perinatal, it is not always clearly identified and could encompass antenatal as well as acute on chronic compound insults. Infants who had a severe antenatal event may recover by the time of birth at which time the stage of encephalopathy is perceived as mild in infants. In contrast, an infant with a more acute insult can have only mild abnormalities on neurological examination in the first 6 hours of age, which can then evolve to moderate or severe abnormalities after the first day of life. An insult can occur many hours before birth and can also have different manifestations both in and ex utero.

[113] He also referred to the following passages in: **"Volpe's Neurology of the new born"**

"In the clinical setting available methods are not ideal for the timing and duration of hypoxic ischaemic insults... The large majority of insults occur in the late intrauterine intrapartum period."

Intrapartum asphyxia and cerebral palsy - is there a link?

“There is a group of termed infants that is triaged initially to the regular nursery without any markers of stress; these infants develop a syndrome of neonatal and encephalopathy with seizures in the first 12 to 24 hours, exhibited systemic organ dysfunction, and have acute neuroimaging changes in the distribution that are consistent with chronic intermittent interruption of placental blood flow. It is the author’s experience that this subset of infants accounts for approximately 50% of the cases of neonatal and encephalopathy; this is consistent with observations that were made by other investigators.”

[114] Professor Rothberg referred to the subacute intrapartum hypoxic ischaemic brain injury referred to in the literature and had reference to the essentials for such injury, which include a cord PH higher than 7, with no delivery room resuscitation and normal Apgar scores and presenting with encephalopathy and renal dysfunction.

[115] He then explained that while cord PH was not present in this case, he postulated that one of the reasons was that they could not test for it in hospital. He believed that they did not feel it was indicated.

[116] He then said that the majority of these requirements are fulfilled in this case and said that “it was postulated that these infants suffer a cerebral insult before labour and have a sufficient recovery to exhibit an uncomplicated labour and then develop a postnatal encephalopathy”.

[117] In his opinion, the child suffered an intrapartum hypoxic ischemic event during the early intrapartum period and might have recovered. In this regard he referred to an article entitled: “**The Bermuda Triangle of neonatal neurology, cerebral palsy, neonatal encephalopathy and intrapartum asphyxia**” and quoted the following excerpt;

“Some children with neonatal encephalopathy will have intrapartum asphyxia. Some children with neonatal

encephalopathy and no intrapartum asphyxia will develop later CP.”

He concluded that in this case insult occurred early intrapartum.

[118] With reference to an article entitled “**Education and debate: a template for defining a causal relation between acute intrapartum events and cerebral palsy: International consensus statement**”, and in particular the following postulations:

“Sentinel hypoxic event: an intrapartum or intrapartum hypoxic event can be silent. It is only when it is apparent or detectable that it helps define the probable timing of the event and the determination of whether its sequelae might have been preventable.”

He stated that there is evidence that this happened in the present case.

[119] And regarding literature entitled: “**Timing of injury in the fetus and neonate**”, he referred to the following excerpt;

“The clinical patterns...are highly supportive of the compelling experimental evidence that injury to the brain is not a single ‘event’ occurring at or just after the insult, but rather that hypoxia-ischaemia precipitates a biochemical cascade of events and changes in cerebral blood flow and activity, which lead to cell death well after the initial insult (during recovery from the insult) ... even following relatively severe insults cerebral oxidative metabolism as measured by magnetic resonance spectroscopy can pass or even fully recover in the so-called ‘latent’ phase lasting, approximately 6-9 hours before the onset of secondary deceleration, which is marked by cell swelling, overt large stereotypic seizure... and eventual accumulation of excitotoxins.”

[120] He explained that the excitotoxins will leak out due to damage to the cells, resulting in brain seizures due to energy failure in the cell, which is unable to retain

integrity, resulting in cell death. The injury might not only involve the area where insult occurred, but may extend beyond with the energy failure starting at the most damaged parts of the brain and then spreading outwards.

[121] And referring to the article: **“Neonatal Encephalopathy and neurological outcome, 2nd Edition (Report of the American College of Obstetricians and Gynaecologist (ACOG) Task Force on Neonatal Encephalopathy”**, he referred to the following excerpt:

“A category II foetal heartrate pattern lasting 60 minutes or more that was identified on initial presentation with persistently minimal presentation or absent variability and lacking acceleration even in the absence of decelerations, is suggestive of a previously compromised injured foetus.”

[122] Professor Rothberg consequently criticised Dr Chimusoro’s statement that a child with previous hypoxic ischaemia insult would always present with abnormal heartrate patterns. He said that multiple children with previous events will present with no foetal heartrate abnormalities. The earlier during labour the insult occurred, the more variable the presentation of neonatal encephalopathy.

[123] On a question by the court as to whether his opinion that the injury most probably occurred during the unmonitored portion of labour is predicated on the assumption that the MCR correctly reflect what occurred during delivery, Prof Rothberg confirmed that that was indeed the case.

[124] He said that a maximum of a third of the labour occurred while being monitored in hospital with the balance of the labour occurring prior to admission. A hypoxic event could not have occurred in the late intrauterine period without an indication of neonatal encephalopathy. In his opinion, recovery or adaptation may result in no signs of foetal distress during active labour, normal Apgar scores at one and five minutes, no requirement for resuscitation and a normal examination in the early post-natal period, as was the case here.

[125] And he gave the following explanation of the mechanism of reperfusion injury:

- a) after the hypoxic ischaemic injury, and when the flow is restored, the foetus may not show signs of insult (the latent phase);
- b) during the latent period improvements can occur to such an extent that there would be no detection of foetal distress (this is consistent with Volpe and other authors);
- c) the latent phase can last up to 15 hours and is followed by a secondary energy failure resulting in cell death; and
- d) the reperfusion of oxygen results in the exit of neurotransmitters which can result in seizures, cell death and brain swelling; and
- e) subsequent progressive signs of encephalopathy result from the underlying reperfusion injury and the later chemical inflammatory and metrical abrasions, which cause possible long-term damage.

[126] In his opinion, the abovementioned process occurred during the largely unmonitored hours of the birth, resulting in poor postnatal sucking, possible lethargy, eventual swelling, seizures and return of the child to hospital due to manifestations of the secondary energy failure. Around 50% of seizures would occur in the secondary energy failure stage during the release of electro-transmitters, associated with brain swelling.

[127] In his view it is likely that the neonate was experiencing partial prolonged ischaemia previously, which now demonstrated through the signs of the reperfusion injury i.e. seizures relating to hypoxic ischaemic encephalopathy. The initial damage occurred, followed by a recovery period, followed by a secondary energy failure, followed by the signs of damaged areas as recovery progresses.

[128] According to him it must be assumed that the staff must discharge the plaintiff and the baby based on an apparent normal labour and delivery, the discussion of feeding options and successful initiation of breastfeeding.

[129] He accepted that the MCR reflect neonate was born without the need for resuscitation and was assessed as normal. He defended the evidence of Sr Dilla, stating that she was under the impression that the child showed the intention to breastfeed, but that the plaintiff's HIV prevented enforced breastfeeding.

[130] He said that if there was no significant resuscitation of the child and the significant late intrapartum event occurred, it would be highly unlikely that the neonate would be born without respiratory impairment or would have reacted to physical tactile stimulation.

[131] In his view ,Sr Dilla differentiated and knew the difference between a flat or healthy baby, and her impression was that the baby was well enough to be put on the mother's chest. Placing the child on the mother's chest imply that the child is healthy.

[132] And in respect of the joint minute with Prof Davis he stated that:

- a) Sr Dilla's evidence of retaining the child on CTG would have enabled her to listen to the beep sound associated with foetal heartrate and identify severe foetal heartrate adjustments;
- b)** he disagreed with the statement that the injury could have been predicted and prevented if the labour were correctly monitored and managed;
- c) according to him the Apgar scores had probably not been falsely elevated;
- d) he remained of the opinion that the child presented with mild encephalopathy at birth. He disagrees with Prof Davis that the injury occurred during hospital admission, stating that the timing of the hypoxic and ischaemic event could not be timed exactly, and could have occurred the unmonitored phase;

- e) he criticised Prof Davis for diagnosing intrapartum hypoxia and ischaemia, stating that the features referred to by Prof Davis are largely absent; and
- f) commenting on the statement of Prof Davis that 91% of injuries may be predictable and preventable, he opined that same only applies to patients who are been observed during the complete labour.

[133] He said that a child with encephalopathy is simply suffering from symphatic nervous system stimulation, which might present hyper alert and only eventually present with respiratory problems, later presenting in feeding problems and being jittery. These features might be difficult for a nurse to detect.

[134] He agreed with Prof Andronikou and Dr Pearce that the injury occurred over hours, but maintained that those many hours likely occurred during the unmonitored phase of labour.

Submissions on behalf of the plaintiff

[135] Mr Wessels submitted that the plaintiff's case was strengthened by her evidence in respect of the lack of monitoring during birth; the dismal failure of the staff to adhere to applicable protocols; the absence of evidence to suggest that she or the foetus reached the hospital in a comprised condition; or that the foetus was already suffering from foetal distress before or upon arrival at the hospital; and her uncontroverted evidence in respect of neonatal feeding and sucking difficulties. And, furthermore, the fact that the child had seizures, at least from the early morning hours of the 10th (2 days after birth) and hypoglycaemia.

[136] The reassuring foetal condition upon admission leaves the probable conclusion that the foetus was uninjured on admission, but went into distress after admission due to the staff's failure to monitor the foetus, which resulted in brain damage of the mixed type and encephalopathy.

[137] He submitted that the court should draw an inference of causal negligence in the absence of an explanation from the defendant to negate the inference.

[138] The version advanced by the defendant on the aetiology, onset and process of the hypoxic ischaemic brain injury is premised solely on the assumption that the MCR are correct and that the staff acted in accordance with all protocols. Because of the MCR entries, the defendant advanced a defence that the injury could have occurred during the unmonitored part of the labour.

[139] He furthermore submitted the factual evidence of the plaintiff is probable and credible, while the evidence of Sr Dilla is reconstructed, the product of guesswork, clearly aimed at exonerating herself and thus not trustworthy. In addition, the records are not nearly contemporaneous, reconstructed, incorrect, contradictory, inadequate, inaccurate and contain fabricated amendments. The version of the plaintiff should accordingly be preferred above the contents of the MCR and the evidence of Sr Dilla.

[140] He argued that all other reasonable causes of brain injury have been excluded. Maternal HIV was not further assessed, nor was the CD4 count, or the viral load ever recorded or further examined. There is no evidence of maternal infection or infective brain injury, and according to the experts there was no reason to believe that the pregnancy did not develop normally or that the foetus was in a compromised condition upon admission.

[141] Prof Andronikou gave evidence that the injury occurred over “many hours”; and an extensive portion and important parts of the brain are involved, inclusive of the perirolandic area which contains the motor cortex and sensory cortex (which allows a child to function) being severely damaged or destructed. The internal capsules, which controls movement and the deep nuclei involvement in the thalamus is also severely affected and a large portion of the brain, which includes the peri-sylvian, posterior watershed and the occipital areas are damaged.

[142] The expert evidence showed that there should have been proper monitoring of the foetus. However, there was no proper monitoring of the foetus to detect foetal distress.

[143] There was probable foetal distress more particularly having regard to the mechanism of damage described by Prof Davis and Dr Chimusoro being that perfusion of oxygenated blood was progressively impeded as contractions increased towards the end of labour; (Dr Koll agreed)

[144] Proper monitoring would have detected foetal distress and had such distress been detected and acted upon, as it should have been, the outcome would have probably been different for the foetus in that a healthy neonate was likely to be born.

Submissions on behalf of the defendant

[145] Ms Goedhart submitted that it is undisputed that the plaintiff laboured for approximately 17 hours outside of the hospital and that only approximately 4 hours of her labour was supervised.

[146] The experts agreed that the damage to the baby occurred in the period of 36 – 38 weeks' gestation. Given that no foetal distress was noted during the plaintiff's labour, and given that the plaintiff does not dispute the admission records at 09h20 and 09h30, the greater likelihood is that the injury to the baby occurred before the plaintiff's arrival at the hospital, and that the baby was in the latent recovery stage during the observed part of the labour.

[147] The recordings on the record are all within the normal range and are not in the least indicative of foetal distress. None of them come close to the lowest parameter (110 bmp) or the highest parameter (160 bpm). In addition, the Apgar scores of 9 at one minute and 10 at 5 minutes, are reflective of a normal crying baby.

[148] The only possible sign of a neonatal encephalopathy was the difficulty to breastfeed, which it is acknowledged most new mothers struggle to do. This would amount to a subtle sign of neonatal encephalopathy, which was only so identified on a retrospective reconstruction of the events.

[149] She argued that the evidence does not support an HIE just before delivery or in the time where the plaintiff was under the care of the defendant's personnel. A baby who suffered an HIE at that late stage would not present with good Apgar scores, especially one who allegedly did not cry for either 30 minutes or one hour. A neonate who did not cry at birth and who did not receive resuscitation is unlikely to have survived, given the level of fall-out reported.

[150] The seizures on day two are indicative of a reperfusion injury, where the main injury occurred at a time distal to the birth, and where the signs of neonatal encephalopathy, if any, at birth were subtle.

[151] She submitted that the experts agreed that the ACOG criteria for an intrapartum injury have not been met, in that an Apgar score of 5 or less must be present, there was no umbilical gas showing acidemia, there was no neonatal imaging available to confirm, nor was there multi-organ system failure, as only one organ, that is neurological dysfunction, was present.

[152] She argued that there was no evidence that foetal distress was present. It has not been demonstrated to have been present, nor that it was discernible. Nor has it been demonstrated that the hospital staff ought to have, or even could have, expedited delivery to avoid the injury. The plaintiff has not proven an absence of monitoring.

[153] That half hourly monitoring instead of hourly monitoring on the part of the hospital staff would have identified foetal distress, of which there is no evidence, is complete speculation. Inferences can only be drawn from established facts. Instead, the plaintiff relies on a reconstruction of events and a reverse-engineering to make out a case for negligence.

[154] The Apgar scores were evidently contemporaneously recorded, because the plaintiff left the clinic with the Road to Health Chart (RTHC) that mirrors what was in the MCR, and could not have been fabricated after the fact.

[155] The plaintiff could not only speculate as to when there may have been an indication of foetal distress. Whilst Sr Dilla's credibility may be questioned in relation

to her subsequent insertions, there was no discernible motive for Sr Dilla to have fabricated the records from after 09h20 until the birth of the Lingomso, including the Apgar scores. There are a number of cross-references across the records (observations, Partogram and nursing notes) relating to the plaintiff's labour which are consistent. It would have required a lot of foresight to fabricate all of the entries from 09h20 onwards. She argued that such a level of fabrication is improbable. It is equally improbable that Sr Dilla would have simply vaccinated an infant who, on the plaintiff's version, must have been manifestly ill at the time of discharge.

[156] She submitted that speculation as to when the insult may have occurred, also means that it becomes speculative as to whether any intervention may have changed the outcome.

[157] She accordingly submitted that the plaintiff has failed to discharge the onus upon her of proving causative negligence on the part of the defendant's personnel.

[158] Regarding the allegation that the defendant's personnel failed to monitor and treat the plaintiff from 07h30 until 13h00, save for vaginal examination at 10h00 in consequence of which the child suffered foetal distress, Ms. Goedhart submitted that the case presented at trial is different to what has been pleaded initially by the plaintiff and in a statement to attorneys on 23 January 2018.

[159] Plaintiff's claim of not having been assessed, save for the assessment at 10h00, is not born out by the record. Not only is it uncontested that the plaintiff was assessed by Sr Dila at 09h20 and Dr. Ntshokota at 09h30, but the plaintiff also accepts that at those times, there was nothing detectably wrong with the foetus. The case presented at trial is different to what had been pleaded initially by the plaintiff and in her statement, in the original particulars of claim, in her reports to the expert and in the application for condonation..

[160] The absence of monitoring, which is denied, does not cause foetal distress as alleged by the plaintiff. Monitoring can only serve to detect foetal distress which is detectable (and it is not always) and may, if detected, alert the medical personnel to a suspicion of foetal distress. The question is then whether, if it were detectable and

confirmed, whether at that point in time, there is anything that could be done to avoid the cerebral palsy.

[161] Regarding the contention that the defendant's personal failed to determine foetal distress, failed to apply intrauterine resuscitation measures and failed to immediately deliver the child promptly upon determining foetal distress, Ms. Goedhardt argued that there is no evidence of foetal distress on the record, nor has any been elicited in evidence. Since there is no factual evidence of foetal distress, intrauterine resuscitation was not warranted.

[162] And regarding the contention that the defendant's personnel failed to provide any or adequate treatment of the plaintiff or her foetus so as to prevent the development of foetal distress or hypoxic ischemic encephalopathy or meconium aspiration syndrome or superadded hypoglycaemia, Ms. Goedhardt argued that the only evidence that the baby suffered HIE is on the MRI, performed on 25 May 2018 when he was two years and three months old, which is evidence of HIE, partial or prolonged and acute profound pattern injury. There was no evidence of discernable foetal distress, there was no evidence of meconium aspiration syndrome and the superadded hypoglycaemia evident on the MRI is not causative of HIE in and of itself, but superadded.

[163] Regarding the assertion that the defendant's personnel failed to monitor and manage the plaintiff's labour and the condition of her unborn baby during labour as required by the maternity care guidelines, Ms. Goedhardt argued that Sr Dilla was challenged repeatedly under cross-examination with regard to the use of the CTG. She was not undermined in this regard and was consistent in her evidence that she had used the CTG.

[164] Her evidence in this regard is supported by the fact that at 12h20 and 11h20 she recorded two readings of the foetal heartrate. This is explained by the fact that, at the time she wrote her notes, the reading on the CTG had changed.

[165] Regarding the allegation that the defendant's personnel failed to appreciate that the plaintiff's baby was born with hypoxic ischaemic encephalopathy, she argued that

the assertion by the plaintiff regarding the presence of encephalopathy at birth is based on her evidence that the baby did not cry or suck at birth. However, there is evidence on the MCR and the RTHC that the baby was sucking.

[166] Under cross-examination, the plaintiff twice testified that the baby was breastfed while she was in hospital. The plaintiff's experts conceded that a first time mother might have difficulty in establishing breastfeeding at the outset. The inability to suck would not in and of itself have been a self-evident sign of encephalopathy to the nursing personnel, particularly under circumstances where the plaintiff was not a high-risk patient. The labour progressed normally and the Apgar scores were normal.

[167] She argued that while there is evidence on the MRI that the baby suffered super-added hypoglycaemia, the plaintiff's own experts attribute that to the baby's poor sucking and that this was as a consequence of the alleged encephalopathy at birth. Professor Davis, in cross-examination, conceded that mild encephalopathy can be missed in the early stages after birth and that this would be more prevalent in a rural hospital where the care of the baby falls to nursing personnel.

[168] Regarding the assertion that defendant's personnel failed properly to assess the plaintiff's baby immediately after birth and to diagnose the baby as suffering from HIE, she argued that it was established that the only evidence that may have presented itself to the defendant's personnel was the poor sucking, which could also be attributable to the plaintiff being unable to breastfeed as a first time mother.

[169] The plaintiff's evidence regarding when the baby started crying was inconsistent. In court she said it was 30 minutes. She advised Dr. Kara that it was 30 minutes. She advised Dr. Chimusoro and Prof Davis that it was after an hour. This evidence would mean that the baby was born flat. In such an event, Sr Dilla would not have recorded Apgar scores of 9 and 10. The baby would also not have been admitted to the post-natal ward, but rather to the neonatal ward. Such a baby would also not have been vaccinated on 9 February 2016.

[170] Regarding the allegation that the defendant's personnel failed to introduce measures, treat and manage the encephalopathy after birth to prevent further brain

injury, she argued that there was no obvious encephalopathy present when the baby was born, given the Apgar's of 9/10 and 10/10. Fitting only occurred after discharge. All Saints Hospital is a district hospital and does not have the capabilities to perform head cooling treatment.

[171] And regarding the allegations that the defendant's personnel failed to retain the baby in hospital to undergo treatment for the encephalopathy, she argued that no encephalopathy was noted at birth or at discharge. The plaintiff testified that she breastfed the baby at hospital, which is why no warning signs were raised about the premature discharge. The only record of poor sucking and lethargy in the new-born baby is by the plaintiff herself. Her evidence in this regard is inconsistent and has varied between the different experts she had seen.

[172] Ms Goedhart also argued that the evidence of Sr Dilla is to be preferred to that of the plaintiff. She criticised the plaintiff as an inconsistent witness and in this regard pointed to discrepancies which in her submission related to: whether she took her anti-retroviral treatments; the time of the onset of her labour; whether she was attended to in the labour; by whom she was seen; the times when she was assessed and the number she was assessed; the condition of the baby at birth and when the baby first cried; the issue of whether the baby could suck or not and whether she breastfed or sip fed the child in hospital; and when the baby suffered seizures, the number of seizures and the management of the seizures. .

[173] She submitted that despite her evidence regarding the seizures, the plaintiff nonetheless failed to return to the clinic for check-ups. The RTHC has entries which do not pertain to the child and begs into question veracity of those entries. The plaintiff was discharged with the RTHC. Sr Dilla confirmed that she wrote into the RTHC the Apgar scores and she administered the vaccinations.

[174] The RTHC also reflects exclusive breastfeeding on discharge and mix feeding (breast and cup). It has also been demonstrated that both plaintiff and her attorney of record deposed to a false affidavit in order to obtain condonation. This included making reference to an incorrect hospital.

[175] Consequently the plaintiff has been demonstrated to be mendacious in her affidavit to obtain condonation and inconsistent about facts falling peculiarly within her knowledge, and which she would have been expected to be consistent about.

[176] Under the circumstances where both the plaintiff and Sr Dilla had demonstrated to be untruthful, the objective recordals of the MCR of the time of the birth must be carry greater weight, bearing in mind that, at that time, neither party had contemplated any claim.

[177] The plaintiff's claim is based on speculation that in three hours and fifty minutes that the plaintiff was in labour at the ASH and prior to the baby's delivery, something went wrong and was missed. Sr Dilla's evidence on the other hand is that the labour was indeed monitored, albeit hourly, that during the time the FRH was monitored and recorded six times, all of which fell within normal parameters, that the baby was assessed and found to be well at birth.

[178] While the guidelines require a hand held Doppler for every thirty minutes, a CTG machine was used and it did not reflect any abnormal FHR. Dr Ntshokota had confirmed that the hospital did have a CTG machine at the time, and the nursing staff used them. He also explained what the procedure was for managing the child that needed resuscitation and confirmed that he was not called in this instance to resuscitate a minor child. The subsequently inserted entries are all irrelevant to the issue as to whether there had been an omission to note foetal distress at the time.

[179] She that if the two conflicting versions are considered in the light of the guidelines for resolving conflicting factual versions as set out in *Stellenbosch Farmer's Winery Group Ltd and Others vs Matel and Others* 2003 (1) SA 1 SCA, the version of Sr Dilla is more probable.

[180] She accordingly submitted that the plaintiff has also failed to establish that the negligence of the defendant's personnel, if any, caused the child's injury

Discussion

[181] The test for negligence is whether a reasonable person in the position of the defendant would foresee the reasonable possibility of his or her conduct injuring the person of another or his or her property and causing him or her patrimonial loss; would take reasonable steps to guard against such occurrence; and that the defendant has failed to take such steps. (*Kruger v Coetzee* 1966(2) SA 428 (A) at 430E-F)

[182] Thus the plaintiff is required to prove, on a balance of probabilities, that the defendant's employees failed to exercise reasonable skill and care, in other words, that their conduct fell below the standard of a reasonably competent practitioner in their field and that the aforesaid negligence caused the child's injury. A medical practitioner is bound to employ reasonable skill and care, and is liable for the consequences if he or she does not. (*Goliath v Members of the Executive Council for Health, Eastern Cape* 2015 (2) SA 97 (SCA))

[183] The plaintiff is not required to prove that the inference she contends for is the only reasonable inference. It is sufficient for her to convince the court that the inference advanced "is the most readily apparent and acceptable inference from a number of possible inferences". (*Goliath* (supra), at para. 19)

[184] The court should select a conclusion that it deems to be the more natural appraisable conclusion from amongst several conceivable ones, even though that conclusion may not be the only reasonable one. The inferences drawn from the facts must be based on proved facts and not matters of speculation. (*AA Onderlinge Assuransie Beperk v De Beer* 1982 (2) SA 603 (A))

[185] This process is achieved on qualitative assessment of truth and inherent probabilities of evidence of witnesses and on ascertainment of which of the two versions is more probable. The court's estimate of the credibility of the witnesses is inextricably linked to the consideration of probabilities, and the court is required to consider these factors simultaneously.

[186] Where a plaintiff is not in a position to produce evidence on a particular aspect, less evidence will suffice to establish a prima facie case where the matter is peculiarly within the knowledge of the defendant. In such a situation there is an evidentiary

burden upon the defendant to show what steps were taken to comply with the standards required.

[187] The factual disputes arising from the irreconcilable versions proffered by the plaintiff and Sr Dilla, respectively, must be resolved on the basis of the following principles enunciated by the Supreme Court of Appeal in *Stellenbosch Farmers Winery Group Ltd v Martell Et Cie* 2003 (1) SA 11 (SCA), at 14I-15D:

“To come to a conclusion on the disputed issues a court must make findings on (a) the credibility of the various factual witnesses; (b) their reliability; and (c) the probabilities. As to (a), the court’s findings on the credibility of a particular witness will depend on its impression about the veracity of the witness. That in turn will depend on a variety of subsidiary factors, not necessarily in order of importance, such as (i) the witness’ candour and demeanour in the witness-box, (ii) his bias, latent and blatant, (iii) internal contradictions in his evidence, (iv) external contradictions with what was pleaded or put on his behalf, or with established fact or with his own extracurial statements or actions, (v) the probability or improbability of particular aspects of his version, (vi) the calibre and cogency of his performance compared to that of other witnesses testifying about the same incident or event. As to (b), a witness’ reliability will depend, apart from the factors mentioned under (a)(ii),(iv) and (v) above, on (i) the opportunities he had to experience or observe the event in question and (ii) the quality, integrity and independence of his recall thereof. As to (c), this necessitates analysis and evaluation of the probability or improbability of each party’s version on each of the disputed issues. In the light of its assessment of (a), (b) and (c) the court will then, as a final step, determine whether the party burdened with the onus of proof has succeeded in discharging it. The hard case, which will doubtless be the rare one, occurs when a court’s

credibility findings compel it in one direction and its evaluation of the general probabilities in another. The more convincing the former, the less convincing will be the latter. But when all factors are equipoised probabilities prevail.”

[188] It is common cause that Sr Dilla had tampered with the MCR entries after copies thereof were provided to the plaintiff’s attorneys, including the *ex post facto* completion of the partogram. When she cross-examined the plaintiff, Ms Goedhart put to her that Sr Dilla admitted to having made the changes, but asserted that she was merely inserting what she had done contemporaneously.

[189] However, when Sr Dilla testified, she simply denied having made the changes. She was obviously lying and made a very poor impression on the court. The question then is whether those changes to the MCR are indeed immaterial and inconsequential, as Ms Goedhart has submitted.

[190] Ms Goedhart has sought to explain her submission by arguing that Sr Dilla’s dishonesty must be understood in the context of her realising that she has committed a criminal offence and fearing prosecution. She argued that the untampered portions of the record nevertheless corroborates Sr Dilla’s testimony and it should therefore be preferred to that of the plaintiff, for the reasons that I have mentioned earlier.

[191] I do not agree with this submission. Sr Dilla’s dishonest, *ex post facto* tampering with the records is only part of the problem. During cross-examination by Mr Wessels, it became apparent that she did not take the completion of the records seriously, nor did she fully understand the import of what she was doing.

[192] She clearly had no understanding of how to measure and note Apgar scores, to my mind, has lied about having used a CTG, purported to assess aspects of the child’s condition that she clearly had no clue what they mean, and appeared to have confused the child with another baby.

[193] She had known that she must record her findings and had appreciated the importance thereof. Yet, she did not record that she was using a CTG without paper.

All the experts were under the impression that she was auscultating and the plaintiff had also asserted that she had been auscultating.

[194] She also did not record the findings of the CTG nor did she record the foetal heartrate before and after the contractions, as she knew she was supposed to record. She conceded that she had no independent recollection of the events, yet insisted that she had used a CTG when there was no record thereof.

[195] She also admitted that the mild contractions were all recorded on the partogram at the same time. This spans a period of two hours. Dr Chimusoro had testified that it was clear that the strong contractions were all done simultaneously. She had recorded mild contractions on the partogram, but wrote in the clinical notes that they were moderate.

[196] The heartrates recorded on the partogram differ from those in the clinical notes. In addition, on the partogram, clinical features of the foetus at 10h20 had not been filled in.

[197] At 20h20 she writes on the partogram "liquor is clear at 20h20". This could, however, only be established if the membranes have ruptured. On the summary of labour, she writes rupture of the membranes happened at 12h50.

[198] Her recording of the Apgar scores are also clearly misleading. She admitted that the Apgars were not taken at one and five minutes respectively, as depicted on the record, but that she had made a general assessment of the child as she handled the baby in the third phase. All these entries were written up 30 minutes after birth and from her memory.

[199] She clearly does not know how to do Apgar scores, if one has regard to her evidence as compared to what the modalities of scoring indicated on the Apgar score. Her first examination of the neonate is also problematic. Everything is recorded as normal even there were male and female genitalia. She does not know how to test the moro reflex and muscle tone, but purported to record findings in respect thereof. She also admitted that the features of another child may have been substituted.

[200] Her records regarding feeding are also misleading. She never saw the child breastfeeding, although that is the impression that is created in the records. She also did not have any further contact with the neonate within an hour after birth and did not assess breastfeeding, sucking reflex or blood sugar levels.

[201] Dr Koll had testified that if there were an intrapartum hypoxic event (which is common cause) and hypoxic encephalopathy after birth (which is also common cause) there must have been foetal distress. He also described how the hypoxia increases in an ascending fashion as contractions increased towards birth. The records are therefore obviously incorrect.

[202] I accordingly agree with Mr Wessels's submission that the recordings of Sr Dilla after 09h20 are false or unreliable.

[203] Sections 13 and 17 of the National Health Act, No. 61 of 2003, place a duty on the hospital to keep and maintain medical records. It is a criminal offence to alter or tamper with medical records. In *Khoza v MEC for Health and Social Development, Gauteng* 2015 (3) SA 266 in (GJ), at para. 42, it was recognised that medical staff responsible for maintaining the patient's medical records have the most to gain by ensuring the safekeeping as the records "as they are surest way of demonstrating that they had monitored and carried out proper procedures. The same staff members also have the most to gain by the disappearance of the contents of the patient's file if they failed to carry out their duties".

[204] Mr Wessel correctly submitted that it was common cause that the staff did not monitor the labour as required in terms of the Maternity Care Guidelines (the guidelines). The plaintiff testified that the foetus's heartrate was only tested by Sr Dilla at approximately 09h20, and by the doctor afterwards. There was therefore, on her version, non-compliance with the guidelines.

[205] Furthermore, the records as they stand, also indicate that there was non-compliance with the guidelines, in that the foetal heartrate was supposed to be checked every half an hour in the active phase, but it was only done every hour. The

instruction in the guidelines state that monitoring must be done before and after a contraction to detect deceleration. On the defendant's own version that did not happen.

[206] I am accordingly satisfied that the evidence has clearly established negligence on the part of the defendant's staff.

[207] Insofar as causality is concerned, Mr Wessels urged me to assess it by taking into account; the common cause aspects; what factual version should be accepted in this instance; whether the plaintiff's version is correct or that of Sr Dilla; the nature and probable mechanism of the damage and; the features exhibited by the baby after birth.

[208] The criterion for determining factual causation, namely the well-known "but-for test" was formulated as follows by Corbett CJ in *International Shipping Co (Pty) Ltd v Bentley* 1990(1) SA 680 (A) ([1989] ZASCA 138) at 700E – H.

"What it essentially lays down is the enquiry — in the case of an omission — as to whether, but for the defendant's wrongful and negligent failure to take reasonable steps, the plaintiff's loss would not have ensued. In this regard this court has said on more than one occasion that the 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 minds of ordinary people work, against the background of everyday-life experiences. In applying this common-sense, practical test, a plaintiff therefore has to establish that it is more likely than not that, but for the defendant's wrongful and negligent conduct, his or her harm would not have ensued. The plaintiff is not required to establish this causal link with certainty."

[209] Thus a plaintiff is not required to establish a causal link with certainty, but only that the wrongful conduct was probably a cause of the loss. This calls for sensible retrospective analysis of what would have probably occurred, based upon the evidence and what can be expected to occur in the ordinary course of human affairs, rather than an exercise in metaphysics. The correct approach is not to search for

scientific certainty, but to assess where the balance of probabilities lie on a conspectus of all the evidence adduced in the case (*Minister of Safety and Security v Van Duivenboden* 2002 (6) SA 341 SCA at 25); *Minister of Finance and others v Gore N.O.* 2007 (1) SA 111 (SCA); (*Michael and another v Linksfield Park Clinic (Pty) Ltd and another* 2001 (3) SA 1188 (SCA), at 1201).

[210] In *Lee v Minister for Correctional Services* 2013 (2) SA 144 (CC), it was held that:

“In the case of an omission, the but-for test requires that a hypothetical positive act be inserted in the particular set of facts, the so-called mental removal of the defendant’s omission. This means that reasonable conduct of the defendant would be inserted into the set of facts. However, as will be shown in detail later, the rule regarding the application of the test in positive acts and omission cases is not inflexible. There are cases in which a strict application of the rule would result in injustice, hence a requirement of flexibility.”

[211] And in *Mashongwa v Praza* 2016 (3) SA 523 (CC), the Constitutional Court cautioned that:

“Lee never sought to replace pre-existing approach to factual causation. It adopted an approach to causation premised on a flexibility that has always been recognised by the traditional approach. It is particularly where the harm that has ensued is closely connected to an omission of a defendant that carries the duty to prevent the harm. Regard been had to all the facts, the question is whether the harm would nevertheless have ensued, even if the omission had not occurred. However, where the traditional but-for test is adequate to establish a causal link it may not be necessary, as in the present case, to resort to the Lee test.”

[212] In *Oppeldt v Department of Health* 2016 (1) SA 325 CC, at paras. 34 to 50, the Constitutional Court held that the but-for test requires flexibility and a common sense approach when the issue of causation has to be decided on the ground of an alleged negligent omission, as opposed to a negligent commission, and explained that:

“While it may be more difficult to prove a causal link in the context of a negligent omission than of a commission, Lee explains that the but-for test is not always the be all and end all of the causation enquiry when dealing with negligent omission. The starting point, in terms of the but-for test, is to introduce into the facts a hypothetical non-negligent conduct of the defendant and then ask the question whether the harm would have nonetheless ensued. If, but for the negligent omission, the harm would not have ensued, the requisite causal link would have been established. The rule is not inflexible. Alternately, it is a matter of common sense whether the facts establish a sufficiently close link between the harm and the reasonable omission.”

[213] To my mind it is significant that the plaintiff and the foetus appeared to have been in good health prior to labour, with no indication of damage or vulnerability before the commencement of labour. The plaintiff started to feel labour pains by approximately 16h00 on 8 February 2016. Labour did not commence at that time, but probably sometime later, as according to Dr Koll it is difficult to establish when labour commences.

[214] The plaintiff was examined in hospital at 09h20 by Sr Dilla, who found her to be in good clinical condition, with foetal movements felt and the foetus being in a reassuring condition. The doctor who examined the plaintiff and foetus 10 minutes later, also found the same. The child was born at 13h50.

[215] The child had encephalopathy of at least Grade 1, which was classified as Grade 2 after developing seizures. He suffered a hypoxic ischaemic injury of the partial prolonged type, combined with acute profound injury features compatible with hypoglycaemia. The injury would have occurred over many hours and was extensive,

in that large portions of the brain of the child had been damaged, especially in relation to those areas governing motor ability.

[216] The Apgar scores of 9/10 and 10/10 were not done in the standard way, but it was a general impression noted by Sr Dilla after handling the baby. The child had cerebral palsy and the records had been tampered with years after the event in some important respects.

[217] If Sr Dilla's recordings were to be accepted, there is no indication of any abnormality during labour or until discharge the following day. One would thus have expected a normal child. Yet, it is common cause that the child suffered serious and extensive brain injury. Her version does therefore simply not accord with the undisputed expert testimony.

[218] Ms Goedhart's submission that the unaltered portions of the MCR corroborate Sr Dilla's testimony, can only be upheld if the integrity of those entries are unassailable. As I have explained above, this is clearly not the case.

[219] As I have said earlier, my impression was that Sr Dilla was a dishonest and unreliable witness who had knowingly tampered with official hospital records and had lied about it. Her recordings simply does not accord with the preponderance of the expert testimony, which concluded that there was something seriously wrong with the neonate. I am therefore of the view that no reliance can be placed on her testimony and records on the MCR.

[220] According to the plaintiff, the baby did not cry after birth and when brought to her, could not feed from the breast and had to be fed with a cup. The implication of not crying after birth is that there was respiratory distress and depleted responses to stimulation. This version accords with the expert evidence, as well as with the probable outcome that would be expected with the birth of an hypoxic neonate with encephalopathic reaction after birth.

[221] Ms Goedhart has urged me to exclude only those entries which had been compromised by the *ex post facto* changes, and to accept those entries which,

according to her, were clearly made contemporaneously and in respect of which there is no evidence to suggest that they are unreliable. In this regard she referred me to *AM obo KM v MEC for Health, Eastern Cape* (699/17) [2018] ZASCA 141 (1 October 2018), where the Supreme Court of Appeal found that the correct approach is to accept the undisputed parts of the record and to exclude the disputed parts. The court in that matter any event found that the alteration of the records was a neutral factor.

[222] The situation here is of course entirely different. The defendant's experts, in particular Prof Rothberg, has constructed a defence based on the assumption that the records are correct. Prof Rothberg has obstinately refused to contemplate the possibility that Sr Dilla, who has been shown to be an unreliable and dishonest witness, has also lied about crucial entries, for instance the manner in which the Apgar scores were taken and whether she had used a CTG.

[223] As I have mentioned earlier, when she was cross-examined by Mr Wessels, it was clear that she had based her scoring on general observations of the child's appearance and made the entries half an hour later. She was also unable to explain why she did not record that she had used a CTG when she was, by her own admission, aware of the importance of keeping accurate records. More concerning was the fact that she was quite prepared to make entries regarding aspects of the child's condition when she clearly lacked proper understanding of what they mean and how to test for them. These entries were also simply incompatible with the condition of a baby that had by all accounts suffered severe brain damage.

[224] While Prof Rothberg has persisted with his speculative hypothesis that the child had suffered the insult before the plaintiff was admitted to hospital, had sufficiently recovered to present with reassuring heartrate upon admission, and only after birth presented with features of HIE, Dr Koll conceded that if the plaintiff's version is accepted, and the court accepts that the injury occurred intrapartum, then foetal distress would most probably have incurred intrapartum,

[225] Mr Wessels has launched a sustained and comprehensive assault on Prof Rothberg's testimony. He submitted that on the probabilities and logic, the testimonies of the plaintiff's experts must be accepted and that of Prof Rothberg rejected as being

illogical, bias and speculative. In order to put his criticisms of Prof Rothberg's testimony into proper perspective, it is perhaps necessary to explain the legal principles relating to assessment of expert testimony. They are as follows:

- (a) an expert's opinion represents his or her view based on facts which are either common cause or established. The opinion can accordingly only be accorded weight if the facts upon which that opinion is based are found to exist. (*Coopers (S.A.) (Pty) Ltd v Deutsche Schädlingsbekämpfung MBH* 1976 (3) SA 352 (A);
- (b) although experts are, by virtue of their specialized knowledge and skill, better qualified than the presiding judge to draw inferences, the probative value of an expert's testimony must be considered in the same manner as that of any other witness. The court is accordingly not bound by an expert's opinion;
- (c) an experts' testimony can only be of value to the court if he or she is found to be neutral and impartial;
- (d) an expert must not advocate his or her client's case or selectively examine only the evidence that support his or her client's case and ignore other relevant evidence. (*PriceWaterhouseCoopers Inc v National Potato Co-op Ltd* [2015] 2 All SA 403 (SCA), at paragraphs 98-99); and
- (e) an expert's bald statement of his or her opinion is of no assistance to the court unless it is uncontroverted. The expert must accordingly fully disclose the premise on which his or her reasoning is based and the process of the reasoning which led to the conclusion.

[226] Does Prof Rothberg's testimony pass muster when assessed in the light of the abovementioned principles? I think not. While I think that Mr Wessels's depiction of Professor Rothberg as a "hired gun" who tried to advocate his client's case at all costs

is a trifle harsh, I do agree with his submission that the former's testimony contravened the principles applicable to expert witnesses in various respects.

[227] Prof Davis testified that the process of multiple partial prolonged hypoxic ischaemic episodes resulted in a cumulative mixed acute profound and partial prolonged injury, that the partial prolonged insults would have manifested through changes in foetal heartrate and a foetal condition and that appropriate intervention could have been instituted successfully.

[228] Dr Kara confirmed that there is little to support an antenatal insult, the undoubted encephalopathy and particularly seizures in the first 36 hours of life is likely the result of brain injury during birth, as supported by the MIR and the type of cerebral palsy suffered by the child.

[229] Dr Koll conceded that foetal distress probably occurred during the late intrapartum period, resulting in hypoxia and ischaemia and associated hypoxic ischaemic encephalopathy.

[230] Dr Chimusoro testified that perfusion of oxygenated blood was progressively impeded as contractions increased towards the end of labour, a hypoxic event prior to admission would probably have presented through foetal heartrate abnormalities upon admission and hypoxic event with recovery without any intrauterine resuscitation was highly unlikely obstetrically.

[231] Only Prof Rothberg has proffered a different opinion. Mr Wessels submitted that he is a neonatologist who stopped clinical practice in 1994, which is 27 years ago and since then appeared to have been involved in academic pursuits and training of therapists. It is common cause that obstetrics is not within his field of expertise and he conceded that the possibility of prolonged foetal distress prior to admission, with subsequent recovery, needs further comment by the obstetrician. I agree with this submission.

[232] It appears that Prof Rothberg has constructed a defence that the damage occurred after commencement of labour until admission in hospital. This falls squarely in the field of obstetricians, of which he acknowledged not to be an expert. He

accordingly had no basis to dispute the evidence of Dr Chimusoro, who stated that if the foetus were suffering from hypoxia it would not have recovered without of the mother being resuscitated.

[233] Prof Rothberg also disputed Dr Chimusoro's evidence that according to the principles stated in ACOG, if a patient would present with a category 1 foetal heartrate pattern (the normal heartrate) that convert to a category 3, it is suggestive of an hypoxic ischaemic event. It is axiomatic that the heartrate obviously would have had to change to have the outcome that what was seen in the child. This supports Dr Chimusoro when he said that on probabilities the child's damage was done after admission to hospital, because the foetus had a normal heartrate on admission.

[234] Mr Wessels has correctly argued that Prof Rothberg conceded that he had no facts to support his opinion, but relied on literature which stated that it can happen that you can have hypoxic damage before labour that can recover significantly to exhibit an uncomplicated labour. The articles which he referred to, are mainly irrelevant and others do not support his opinion. His opinion was that the damage occurred after labour commenced. Mr Wessels submitted that the features exhibited by the control group of children mentioned in the article are not the same as the child in this case and the article is thus not supportive of Prof Rothberg's theory. Notwithstanding extensive research by him he could not find any support in the literature otherwise he would surely have submitted it.

[235] Prof Rothberg's reasoning was to my mind strained and illogical in certain respects. He said that he does not know what the cause for the hypoxic event was. He conceded that if the baby is not born approximately 40 minutes after acute profound damage sets in, the baby is usually dead. On that basis the baby should on logical reasoning have been dead by the time the plaintiff was admitted to the hospital. His opinion, however, is that the damage to the brain and heart, for unexplained reasons, stops before admission to the hospital. The foetus then must have recovered sufficiently to be in a reassuring condition on admission. However, it is common cause that the baby did not recover and has suffered extensive damage according to Prof. Andronikou.

[236] I am consequently of the view that Prof Rothberg's hypothesis was speculative and without any factual basis.

[237] The plaintiff testified that she was only examined once by the nurse and once by the doctor each time with a fetuscope. She was not examined again thereafter until birth. There was then a period of approximately three and a half hours of unmonitored labour during which time the stress on the baby increased with the increasing strength of contractions and the risk of hypoxia increasing up to birth. In my view it is more probable that the damage occurred in the time. The expert testimony has confirmed that the damage could have been prevented if there was proper monitoring, which would have detected an abnormal heartrate during the buffer stage before damage is done to the brain. This would have allowed an intervention by way of maternal resuscitation and caesarean section before the damage occurred. The damage is therefore closely connected to the failure to monitor as required by the guidelines.

[238] I am accordingly of the view that the reassuring condition of the foetus upon admission, compels the conclusion that the foetus went into distress thereafter and the hospital staff's negligent failure to monitor the foetus resulted in it suffering brain damage of the mixed type and encephalopathy.

Order

[239] In the result the following order issues:

1. The defendant is held liable for the plaintiff's agreed or proven damages arising from the cerebral palsy suffered by the minor child, Lingomso.
2. The determination of the quantification of the plaintiff's claim is postponed sine die.
3. The defendant shall pay the plaintiff's costs relating to the merits, together with all preserved costs, such costs must be paid on the party and party scale, and shall include:

(a) the costs of the preparation of the expert reports and qualifying fees, if any, of the following expert witnesses:

- (i) Prof Andronikou – Radiologist;
- (ii) Prof Davis – Neonatologist;
- (iii) Dr Kara – Paediatrician
- (iv) Dr Pearce – Paediatric neurologist; and
- (v) Dr Chimusoro – Obstetrician and Gynaecologist.

(b) Costs of two counsel.

4. The defendant shall pay interest on the aforesaid costs at the current prescribed legal rate of interest, calculated from 30 days of *allocatur* or agreement, to date of payment thereof.

J.E. SMITH
JUDGE OF THE HIGH COURT

Appearing on behalf of the Plaintiff:
Instructed by:

Adv. Wessels SC with Mr Uys
NONXUBA INC.

Plaintiff's Attorneys
345 Rivonia Boulevard
Ground Floor, Block B 2
Edenburg, Rivonia
2191

JOHANNESBURG

REF. NO: ZMMN/FM/MAT725

Service Address:

14 Stewart Drive
Berea

East London

Appearing on behalf of the Defendant:
Instructed by:

Adv. Goedhart SC with Ms Cassim
ZILWA ATTORNEYS

Defendant's Attorneys
100 High Street

P O Box 2141

GRAHAMSTOWN 6140

Our ref.: AF Basson/lab/M434