

Editorial note: Certain information has been redacted from this judgment in compliance with the law.



**IN THE HIGH COURT OF SOUTH AFRICA
(EASTERN CAPE DIVISION, MTHATHA)**

Case No: 35/2019

In the matter between

S S

Plaintiff

And

MEC FOR HEALTH

Defendant

JUDGMENT

PAKATI J

INTRODUCTION

[1] The plaintiff is Ms SS, a woman who claims damages against the defendant, the MEC for Health, in her name and representative capacity as mother and natural guardian of the child, hereinafter referred to as ES, which arose out of the alleged negligence committed by the defendant's employees that resulted in brain damage suffered by ES while she was admitted at Zithulele Hospital, on 11 March 2014. The defendant defended the action.

[2] When the trial started on 26 January 2021, the parties agreed to separate the special plea of prescription as far as the plaintiff's claim in her personal capacity is concerned, as

well as *quantum* from the plaintiff's claim in her representative capacity. I made the joint application as an order of court.

THE ISSUES

[3] The issues for determination are whether the defendant's employees were negligent in the management and monitoring of the plaintiff's labour and the subsequent delivery of ES on any of the grounds pleaded by the plaintiff in her amended particulars of claim, dated 07 September 2020. The issue further is whether or not such negligence caused ES to develop cerebral palsy as a consequence of a hypoxic-ischaemic event at birth.

THE PLEADINGS

[4] Regarding negligence, the following was pleaded in paragraph 13 of the amended particulars of claim:

"13. The defendant was negligent in one, or more of the following respects:

13.1 At Mqanduli Community Health Care Centre, she failed to:

13.1.1 permanently, alternatively temporarily employ the services of suitably qualified and experienced nursing practitioners who understood the adverse consequences of use of phenytoin 300mg, Degranol 200mg BD by pregnant women; and

13.1.2 train the employees on the adverse consequences of phenytoin 300mg, Degranol 200mg BD by pregnant women.

13.2 At the hospital, she failed to:

13.2.1 permanently, alternatively temporarily employ the services of a suitably qualified and experienced medical practitioners who would be available and able to examine, manage and/or give appropriate advice in respect of the plaintiff's labour and to perform a caesarean section if and when required to;

13.2.2 ensure that at least one medical practitioner was in attendance at the hospital at all the material times relevant hereto;

13.2.3 permanently, alternatively temporarily employ the services of a suitably qualified and experienced nursing staff who would be able to properly assess, monitor and/or manage the plaintiff's labour;

13.2.4 ensure that the hospital was suitably, adequately and/or properly equipped to enable the timeous and proper performance of a caesarean section if and when required by the plaintiff;

13.2.5 take any or all reasonable steps to ensure proper, timeous and professional assessment of patients, their monitoring and management of labour and transfer of patients to other suitable hospitals and/or medical facility indicated, required and/or requested; and

13.2.6 prevent [E] from suffering cerebral damage at birth and the consequences thereof when, by the exercise of reasonable care, skill and diligence, he could and should have done so.”

[5] In paragraph 4 of the plea to the plaintiff’s amended particulars of claim, the defendant pleaded thus:

“4. 4.1 The defendant denies all allegations of negligence and/or breach of legal duty on the part of the defendant.

4.2 In amplification the defendant pleads as more fully set out below.

4.2.1 The plaintiff presented at Mqanduli Community Health Centre at approximately 14h05 where she was assessed at 14h30 and found to be in labour.

4.2.2 The plaintiff was a known epileptic.

4.2.3 The plaintiff was referred to the Hospital for management.

4.2.4 The plaintiff and foetus were monitored on an ongoing basis and in accordance with applicable prescripts, which monitoring included continuous cardiotocography (“CTG”). The tracings of the CTG, while not re-assuring at some time, showed good recovery and variability in between.

4.2.5 [E] was born through vaginal delivery at approximately 20h55.

4.2.6 [E]’s Agpar score at birth were recorded as 5/10 at 1 minute and 6/10 at 5 minutes.

4.2.7 [E] suffered intra-uterine growth restriction (“IUGR”) which was not (and could not be) detected during the plaintiff’s pregnancy as she booked only on 11 February 2014 – only four weeks before she went into labour and delivered [E] on 11 March 2014 – and therefore too late.

4.2.8 [E] was born with a tight nuchal cord around the neck after the plaintiff’s first stage of her labour had progressed rapidly to full dilatation in just 40 minutes.

4.2.9 [E]’s IUGR primed her for birth hypoxia and the rapid progression of the plaintiff’s labour from 3 cm to fully dilated contributed to the tightening of [E]’s nuchal cord at a time when it was too late to detect, intervene and prevent the resultant progression of hypoxia immediately before her delivery.

4.3 Alternatively to paragraph 4.1 above, and in the event of the above Honourable Court finding that the medical staff and staff providing related services at Zithulele Hospital negligently and wrongfully breached their duties in one or more of the respects alleged in the amended particulars of claim, or at all, the defendant denies that such negligence and/or breach contributed to, or was a cause of, the outcomes alleged and/or any damages which the plaintiff may prove she or [E] has suffered and accordingly denies the contents of paragraphs 7 to 15 of the amended particulars of claim.”

[6] For the plaintiff to succeed and hold the defendant liable for damages, she must prove on a balance of probabilities, causal connection between the defendant’s negligent acts or

omission relied upon and the harm suffered.¹ In *Minister of Safety and Security v Van Duivenboden 2002 (6) SA 431 (SCA)*² Nugent JA remarked:

“[12] Negligence, as it is understood in our law, is not inherently unlawful - it is unlawful, and thus actionable, only if it occurs in circumstances that the law recognises as making it unlawful. Where the negligence manifests itself in a positive act that causes physical harm it is presumed to be unlawful, but that is not so in the case of a negligent omission. A negligent omission is unlawful only if it occurs in circumstances that the law regards as sufficient to give rise to a legal duty to avoid negligently causing harm. It is important to keep that concept quite separate from the concept of fault. Where the law recognises the existence of a legal duty it does not follow that an omission will necessarily attract liability - it will attract liability only if the omission was also culpable as determined by the application of the separate test that has consistently been applied by this court in *Kruger v Coetzee*, namely whether a reasonable person in the position of the defendant would not only have foreseen the harm but would also have acted to avert it. While the enquiry as to the existence or otherwise of a legal duty might be conceptually anterior to the question of fault (for the very enquiry is whether fault is capable of being legally recognised), nevertheless, in order to avoid conflating these two separate elements of liability, it might often be helpful to assume that the omission was negligent when asking whether, as a matter of legal policy, the omission ought to be actionable.”

[7] The plaintiff testified in her case. Four expert witnesses also testified. Dr Constant Ndjapa, a gynaecologist and obstetrician and Dr Yatish Kara, an expert paediatrician, testified on behalf of the plaintiff. The defendant called Dr Thandi Nothanduxolo Mtsi, a gynaecologist and obstetrician, and Professor Cooper, an expert paediatrician and neonatologist, to testify. Drs Ndjapa and Mtsi compiled joint minutes, dated 11 March 2020 and 25 January 2021. Dr Kara and Prof Cooper also compiled a joint minute, dated 15 August 2019. The radiologists, Professor J Lotz, for the plaintiff, and Dr J Swartzberg, on behalf of the defendant, prepared a joint minute dated, 10 December 2019, in an attempt to present to court the imaging features of the Magnetic Resonance Image (“MRI”) brain scan and advance a diagnosis for the described pattern.

[8] Various medico-legal reports of the above experts formed part of the evidence. The maternity case records, inclusive of fluid and feeding charts, Zithulele Maternity Ward

¹ See *Minister of Police v Skosana 1977 (1) SA 31 (A)* and *Blyth v Van Den Heever 1980 (1) SA 191 (A)*.

² At para [12].

progress notes, Zithulele Hospital prescription charts, post-delivery discharge from Zithulele maternity, Road to Health Card, Zithulele laboratory reports and cardiotocography (“CTG”) scans were also part of the evidence. The applicable maternity guidelines were the Guidelines for Maternity Care in South Africa, 2007 3rd edition.

[9] It is undisputed that according to the antenatal records only two antenatal visits appear, the first being on 11 February 2014 and the second, on 11 March 2014. The experts agreed that the plaintiff was *primigravida* and was on treatment for epilepsy. It is also common cause that on 11 March 2014 she was given antibiotics for vaginal discharge. The gestation period was estimated to be 36 weeks, presentation was cephalic and the head was 3/5 above the symphysis pubis. The foetal heart rate (“FHR”) was recorded as 121, with mild contractions and membrane not felt. She was referred to Zithulele Hospital as she was a known epileptic.

THE EVIDENCE OF SS (THE PLAINTIFF’S CASE)

[10] The plaintiff was born on [...] December [...] in Macosa, in the district of Mqanduli. She was 28 years old when she testified. In November 2013, she went to Mqanduli Clinic to collect her treatment for epilepsy and because she missed her periods she also reported same to the nurse. She did not know the date of her last menstrual periods. She also did not recall the date of her visit to the clinic. At the clinic, urine sample was taken from her and tested. However, the results were inconclusive, whereupon blood was drawn and she was told to collect results in a month’s time. In December 2013, she again visited the Mqanduli Clinic and was told that “*I was at the beginning of the six months, sixth month [of] my pregnancy.*”

The nurse checked the status and progress of her pregnancy and she had a review date in January 2014, for antenatal care. She was further informed that there was nothing wrong with her and the baby.

[11] According to the plaintiff, her medical and antenatal cards were kept at the clinic. Only numbers were issued to her. She was expected to give the said numbers to the nurses in order to locate her cards when visiting the clinic. In January 2014, she returned to Mqanduli Clinic for an antenatal review. During that visit, she was told that her medical and antenatal cards were missing. However, she was subjected to antenatal procedures and given a review date in February 2014. In February 2014, she went to Mpunzana Clinic because she was unhappy with the fact that her cards could not be found at Mqanduli Clinic. She thought that the nurses in Mqanduli Clinic were negligent in handling same. At Mpunzana Clinic she was asked if she had previously attended another clinic. In response, she told them that she had been to Mqanduli Clinic. She explained to the nurses that at Mqanduli Clinic they lost her cards. A new antenatal card was opened for her with instructions that she should keep it and produce it at a clinic or hospital when in labour. Thereafter, she was subjected to antenatal procedures and given a review date in March 2014. Again, during this visit, she was told that there was nothing wrong with her or the baby. She could not remember the date that she went to Mpunzana Clinic for the first time. She only remembered that it was in February 2014.

[12] On 11 March 2014, the plaintiff returned to Mpunzana Clinic at approximately 07h00 in the morning for an antenatal review. She also reported that she had been experiencing lower back pains since 06h00 that morning. However, she was treated for vaginal discharge which was noted and given treatment. She returned home. While at home, the lower back

pains persisted. She informed her mother who suggested that they go to Mqanduli Clinic, which they did. The plaintiff estimated that she and her mother left home at about 10h00 in the morning and arrived at the clinic at approximately 11h00. After examination, the nurses established that she was in labour. Considering that she was epileptic, she was referred to Zithulele Hospital for a doctor to manage her labour. She waited for an ambulance that was going to transport her to Zithulele Hospital, which ultimately arrived and they left.

[13] As they travelled to Zithulele Hospital, the progress of her labour was not monitored. She was told to lie on her back throughout the journey. She was uncertain as to what time the ambulance arrived at Zithulele Hospital but estimated that it was approximately at 14h00. She thought that they travelled for about an hour. The plaintiff was in the company of her mother and ambulance staff when they proceeded to Zithulele Hospital. Upon arrival at the hospital, she was wheeled by a wheelchair to the maternity ward, registered and admitted. A nurse who first interacted with her commented that she did not appear to be in labour and asked her to urinate. After that, the nurse told her to climb on a bed, which she did. She inserted a finger in her vagina and made no comment. She then left at around 15h00. She again interacted with another nurse at approximately 17h00 who placed a belt like a computer (CTG) on her stomach and said nothing to her. He/she thereafter left. The same nurse interacted with her again at approximately 18h00. He/she looked at the computer and wrote something down and thereafter left. At around 19h00 the same nurse returned, looked at the computer, recorded something down and left. At about 20h00, she felt pains and shouted for help and a nurse approached her. When the nurse was at the door, she commented that “*the baby’s head was coming out*”. One nurse pressed on her abdomen and the other cut her, and the doctor arrived. The baby was then delivered.

[14] The baby did not cry at birth. The doctor took her to a separate room and placed her in ‘something like a box’ made out of something like a glass’ (incubator), as the plaintiff put it. Some oxygen was placed in her nose with some tubes. The plaintiff did not breastfeed her. She was given a mug and a syringe. She expressed breast milk into the mug, sucked it with the syringe from the mug and placed it in the tubes in order to feed the baby. The baby was given to her after five days and she was asked to breastfeed her.

[15] After some time, the baby could not crawl, walk or speak. She also could not attend school. She can do nothing on her own.

THE MATERNITY CASE RECORDS

[16] According to the maternity records, the initial assessment was at 14h30, and the plaintiff was 3cm dilated, pelvis adequate, head 3/5, contractions < 20 seconds, FHR normal and 36 weeks on palpation. At 17h20 bradycardia was noted, meaning that the FHR had dropped up to 100 beats per minute. There was no progress in labour. The plaintiff was put in left lateral position, and oxygen was given via mask and ringers lactate running well, PV was done, and she was still 3cm dilated. Contractions were moderate to strong. The doctor was informed about the problem at 18h45. The doctor indicated that there was no problem, the nursing staff should continue with the then-current management. He/she did not come to assess the plaintiff.

[17] The next assessment was at 19h00 and three decelerations were noted despite the then-current management. The doctor was informed of the plaintiff's condition. At the time, the plaintiff was still on continuous CTG, oxygen and ringers lactate and lying in left lateral position. The FHR dropped again to 100 beats per minute. The doctor was again called but did not come.

[18] At 19h05 Dr Glaze recorded that the plaintiff was "*primigravida, 39 weeks by dates, 3 cm dilated, and good strong contractions. Concern re late decels on CTG but CTG with toco looks to show just early decels with good recovery and good variability in between. Several also witnessed - decels to 100bpm but good recovery to 140. Plan – left lateral position + oxygen- keep on CTG and monitor closely. If not progressing or late decels, call Dr.*"

[19] At 20h00 the progress of labour was good. Regarding the foetal condition, the "*CTG was observed to be still up and down*". The overall assessment was that the progress went very quickly from 3cm to full dilatation in 40 minutes. There was no urge to bear down. At 20h50 the plaintiff was fully dilated and delivered a flat and floppy baby with a cord tight around the neck twice at 20h55, with Agpar score of 5/10 and 6/10. Resuscitation and head cooling were done. The baby had a seizure witnessed by the plaintiff. However, her condition improved. She was kept in high care in IV fluids as feeding per mouth was not initiated. The placenta was normal. It appeared that the baby was premature, delivered at 36 weeks in keeping with the birth weight of 2.5kg.

[20] On 17 August 2018, ES was subjected to a MRI scan which proved that the MRI study showed a mixed pattern of prolonged partial (peripheral) and acute profound (central) hypoxic-ischemic injury of the brain in a chronic stage of evolution. The acute profound injury was dominant and global white matter loss was severe. There were no findings of congenital abnormalities or genetic disorders or inflammatory brain disease. Prof Lotz and Dr Swartzberg compiled a joint minute dated 18 and 25 November 2019, in which they agreed as follows:

“20.1 The study shows a mixed pattern of prolonged partial (peripheral) and acute profound (central) hypoxic ischemic injury;

20.2 The findings of the MRI suggest that genetic or metabolic disorders are unlikely causes of brain injury;

20.3 Inflammatory or infective causes are unlikely as causes of the child’s brain damage; and

20.4 A review of the clinical and obstetrical records by appropriate specialists in the field of neonatology and obstetrics to be essential in determining the cause and probable timing of the hypoxic ischemic injury.”

[21] The expert witnesses who testified on behalf of the parties agreed with the findings of the radiologists. However, what the radiologists did not say is the timing of the brain injury. In their view, a review of clinical and obstetrical records by neonatologists and obstetricians was essential to determine the cause and probable timing of the hypoxic-ischemic injury, as stated above. Commenting on the radiological report, Dr Ndjapa stated that perinatal asphyxia, more appropriately known as hypoxic (lack of oxygen) ischemic (lack of blood flow) encephalopathy (brain damage) (HIE), is characterised by clinical and laboratory abnormalities with evidence of acute or sub-acute brain injury due to asphyxia. He stated further that the cause of this condition was systemic hypoxemia and/or reduced cerebral blood flow. He contended that birth asphyxia causes 840,000 or 23% of all neonatal deaths worldwide. Dr Kara confirmed that the probable cause of cerebral palsy was the prolonged partial and acute profound hypoxic-ischaemic insult. The experts agreed with the radiological

report, hence there arose no need for the radiologists to testify. The evidence by the experts shows that the insult probably occurred during labour and not ante or post-natally.

[22] Dr Ndjapa testified that in compiling his medico-legal report he had regard to information from the history of events narrated to him by the plaintiff, available information in the maternity case records, a perusal of reports made available to him by the plaintiff's attorney of record, Mr Dayimani, a trial bundle containing NICE guideline on epilepsies diagnosis and management (2012) as well as ACOG guidelines for perinatal care, 7th edition. The purpose of the report was to establish whether the hospital staff was negligent in managing and monitoring the plaintiff's labour and if so, whether such negligence caused ES' injury and resultant cerebral palsy. His commentary would be confined to his area of expertise namely, pregnancy, labour and delivery with special reference to the guidelines as well as other literature and reviews in order to assist in establishing whether birth asphyxia and cerebral palsy could be attributable to antiepileptic drugs used during pregnancy and if there was any association between labour monitoring, FHR abnormalities, delay in delivery time, hypoxia, birth asphyxia and cerebral palsy.

[23] Dr Ndjapa opined that at 14h30 the partogram of the clinical record showed that the plaintiff, who was at the time 3cm dilated, was transferred from the latent phase of labour to the active phase, on to the alert line. At 17h20, when there was no progress of labour, the partogram was not plotted. He asserted that, had the nursing staff plotted the partogram, they would have noted that the plaintiff had crossed the action line with the FHR rate abnormalities present and immediate action needed to be taken by delivering the baby by the fastest route possible. This would have been by caesarean section. The next entry on the

parthogram was at 20h50 while the clinic card showed that she was fully dilated at 20h00, showing incoherence in the records. This suggested that five minutes later she delivered ES with Aggars of 5/10 and 6/10, if the parthogram entry is accepted. What is clear is that from 3cm dilatation at 14h30 up to delivery at 20h55 it took at least six hours of no monitoring as the parthogram has no recordings between 14h30 and delivery at 20h55. This, according to Dr Ndjapa, suggested that the baby was exposed to hypoxia over a long period of time and further foetal abnormalities were missed or unattended resulting into negligence and substandard care of over at least four hours. He was also critical of the CTGs, especially at 18h20 which showed that there was a late deceleration, which position changed at 18h50 when the FHR abnormality became worse and yet no action was taken. At 20h10 the CTG continued to be non-reassuring. He asserted that there were neither records to show that the plaintiff was given hexoprenaline 10 mg to delay labour, nor was she prepared for immediate caesarean section. He asserted further that the plaintiff's condition of having prolapse, was a high risk considering that she was pregnant.

[24] According to the Guidelines, in the active phase the FHR must be checked every 30 minutes and the patient assessed every two hours and that information be recorded on the parthogram. In this case, from 14h30 the plaintiff was only assessed at 17h20, approximately three hours later. Dr Ndjapa said: *“The staff that was examining the patient noticed that the patient was not progressing. It is evident that because the patient crossed the action line it is a true reflection that the patient was not progressing, and we know that there was foetal bradycardia, meaning that the heartbeat was abnormal...There was no plotting at 17h20 because you plotted, you will notice that there is a labour problem.”* According to him, failure to plot on the graph was against the Guidelines. That is because *‘with a graphic representation, it is very easy to pick up that something is wrong.’*

[25] After the doctor was notified at 18h45 that the foetal heartbeat was dropping, he/she came at 19h05 to assess the plaintiff. Again, the graph does not show plotting at 19h05 either by the doctor or nurse, Dr Ndjapa stated. Instead, what appears from the maternity case record at 18h45 is the following: “*Dr informed about the problem – No problem, should continue with the current management.*” According to Dr Ndjapa, at that stage, there was a problem with foetal bradycardia and no progress in labour. He contended that at this stage, it was imperative that the doctor on call come and examine the plaintiff personally as it appeared that the nurses examined the plaintiff and noticed that there was a problem. He contended further that although there was a problem at 17h20, the labour was allowed to progress until 18:45, approximately more than an hour later.

[26] Dr Ndjapa interpreted the CTGs which consisted of numbers 127, 128 and 129 as the first CTGs, the second, 130, 131, and 132, and the third, 139, 140 and 141. He remarked that there were CTG’s around 18h12, 47 seconds until about 20h00, and none between 14h30 to 17h20. There was nothing before 127 and a part starting before 128 was cut and he did not know what actually happened there, which made it difficult for him to comment. Some pages had no continuity especially from 133 to 138. The defendant accepted that the CTG’s were incomplete. As custodians of the documents, the defendant led no evidence explaining whether a diligent search for the missing portions was done, when it was done and by whom or that the whereabouts of the missing ones were determined.

[27] Dr Ndjapa explained different types of decelerations namely, early, late and variable. He defined early deceleration as a drop of the FHR below the normal range. A CTG is used

to monitor labour and indicates what kind of deceleration takes place at a particular time. Management of labour depends on it. Dr Ndjapa said that foetal monitoring includes monitoring the maternal condition as the mother is examined regarding contractions. Concerning foetal monitoring, he referred to the Guidelines where the following is recorded:

“FETAL MONITORING

For low risk labour, listen to the fetal heart with a stethoscope or hand-held Doppler instrument every 30 minutes, before, during and after contractions. Cardiotocography (CTG) is used for high risk labour only, and should be available in hospitals. CTG machines are however in short supply. All CTG tracings must be kept safely in the mother’s file. After CTG interpretation, write a note in the file with a comment on the CTG, so that a record is available even if the CTG tracing is lost.” Emphasis added

[28] Dr Ndjapa explained further that a normal CTG does not have decelerations but good beat-to-beat variability. The normal FHR is between 110 and 160 beats per minute: anything above 160 beats per minute is tachycardia, and anything below 110 beats per minute is bradycardia. Dr Ndjapa stated that a deceleration is timed with a contraction to decide whether it is early or late. To do this, the baseline of the CTG must first be identified. Then it must be determined by looking at the peak of a contraction as well as that of a deceleration to see which one comes first. Therefore, every drop of the FHR below the baseline is a deceleration and should be monitored by the medical staff concerned even when it is not bradycardic. That is so because under normal circumstances there should be no deceleration. Dr Ndjapa explained further that if the peak of a deceleration comes after the peak of a contraction, then that deceleration is late. The opposite is early deceleration which is usually not problematic.

[29] Concerning CTG 127, 128 and 129, Dr Ndjapa testified that it had a baseline of 120 beats per minute. In this CTG, he opined that several decelerations dropped below 120 beats

per minute which meant that the FHR was dropping. Some of the decelerations took time to recover, although some had a quick recovery. He opined further that the decelerations started much earlier and then moved to the baseline. Amongst others, he identified another deceleration, which he referred to as U-shaped, whose recovery took time. The decelerations were not only late but also pathological. He also identified contractions and late decelerations in CTG 128.

[30] In CTG numbers, 130 – 132, Dr Ndjapa identified about four late decelerations with poor variability and testified that the baseline was 130 beats per minute. What complicated these decelerations is that they were problems related to FHR variability. It was not just late decelerations, but the variability appeared to be reduced, which meant that the baby was no longer recovering well like before, as late decelerations continued. That is because once there were many components in the abnormal heart rate condition, the CTG became ‘*somewhat pathological.*’ It then became difficult for the baby to keep up with the recovering process because foetal oxygenation kept becoming impaired. That was hypoxic and the baby developed acidosis. Dr Ndjapa contended that the condition of the baby continued to deteriorate. The clinical records showed that this CTG was classified as “*up and down*”. He contended further that this classification meant nothing to an obstetrician ‘*as there is no such thing as up and down*’ in a CTG. He said: “*It either shows that you are managing labour ward but you do not know what you are actually talking about. ...[U]p and down is not in a language of the obstetrician.*” He asserted that in the circumstances, a wrong decision could be taken because the person making that kind of entry, was not sure of what was going on. He asserted further that the baby was fine only when the CTG was normal otherwise when abnormal, a diagnosis should have been made and appropriate treatment given. This CTG was abnormal, and the foetal heart condition remained non-reassuring. According to Dr

Ndjapa, this CTG, unlike the first one, showed that the foetus had a reduced capacity to recover, and it was like a person who had been subjected to choking for a prolonged period of time. Only when a CTG is normal, can it be said that the foetus is fine. If it is not, an investigation to determine the cause of the abnormality, should be done and appropriate treatment must follow.

[31] Regarding the third CTG, Dr Ndjapa testified that the recording showed that the baby was trying to compensate but there was decreased variability and signs of increased hypoxia. There was almost no zigzag like the previous one.

[32] When Dr Ndjapa was asked as to the appropriate treatment that had to follow after bradycardia and no progress in labour was noted, he said that, that condition was quite serious. The doctor was expected to act immediately by coming and assessing the plaintiff's and the foetus' condition and make his/her findings. In that manner the doctor would have had a clear understanding of what was happening in relation to the baby and mother. The plaintiff had strong contractions. The intervention that was done when bradycardia was first noted was proper but was meant as a temporary measure to buy time to prepare to deliver the mother by the fastest route possible. Considering that the plaintiff was not fully dilated and still far from delivery, it would have been impossible to have a vaginal delivery. Therefore, the only option would have been a caesarean section. That is because there were warning signs of foetal distress and the FHR did not return to normal after intrauterine resuscitation.

[33] When it was put to him that ES was primed for foetal distress due to the tight nuchal cord twice around the neck and the rapid progress of labour, as well as the outcome, he said

that when labour is monitored and tachysystole develops, tocolysis should be given to the patient to reduce that tachysystole as it is an abnormal condition, otherwise it is negligent not to.

[34] Regarding a condition called Intrauterine Growth Restriction (“IUGR”), Dr Ndjapa asserted that such a condition might afflict a foetus. He admitted that during labour a growth-restricted foetus also preserves oxygen supplies for the vital parts of the brain. However, he disputed that it would be impossible to detect growth abnormalities between the period 11 February 2014 and 11 March 2014 when the plaintiff visited Mpunzana Clinic for antenatal care. According to him, if the antenatal graph was properly plotted on the first visit based on the information given by the plaintiff, the abnormality would have been picked up and acted upon. Dr Ndjapa also admitted that the entry by Dr Glaze at 19h05 showed that there was a sign of late decelerations. Dr Ndjapa was concerned about the interpretation of the CTG by Dr Glaze saying it was not only wrong but he/she ignored the late decelerations that were brought into his/her attention. There were also further decelerations after he/she had seen the plaintiff. Dr Ndjapa remarked that a patient cannot be put on a CTG and be given oxygen. According to him, good recovery of the heart rate should be maintained. When there are still decelerations, there can be no good recovery.

[35] In paragraph 9.5 of his report filed on 20 February 2020, Dr Ndjapa stated:

“9.5 Fetal condition was inappropriately monitored suggesting that the fetus may have been exposed to hypoxia over a period of time, long enough to cause cerebral damage and resulting in cerebral palsy. The outcome at delivery (poor apgars score, the need for resuscitation and admission to ICU, seizures in the first 24 hours) suggest that the damaging event may have occurred during intrapartum period. There is no evidence that the damaging event may have occurred in the antenatal or the postpartum period, even though the patient was a known epileptic there is no evidence to suggest that her epileptic treatment was attributable to baby

[ES's] birth asphyxia and cerebral palsy, the damaging event may have occurred during labour and delivery.”

[36] Dr Ndjapa testified that for his conclusions, he relied on the Guidelines for Maternity Care, in South Africa, 2007 edition the Guidelines which stipulate that when there is foetal distress and delivery is imminent, meaning that the cervix is fully dilated, the baby must be delivered immediately by vacuum extraction, if necessary. Forceps can also be used. If vaginal

delivery is not imminent, meaning the cervix is not fully dilated, as in the present case, the mother must be given hexoprenaline 10 micrograms IV to delay the labour and prepare for immediate caesarean section. The mother should urgently be transferred from a community health centre to hospital. *In casu*, the plaintiff was already in the hospital. In this regard, Dr Ndjapa testified that according to the hospital records, the plaintiff was far from full dilatation

(3 cm dilated) and there was no record that she was given hexoprenaline 10 micrograms IV or prepared for an immediate caesarean section. He asserted that monitoring and management of the plaintiff's labour was not according to the Guidelines.

THE EVIDENCE OF DR KARA

[37] Dr Kara's mandate was to conduct a medico-legal assessment of ES and advise on the causal connection between the delivery and subsequent neurological outcome, that is, the probability of intrapartum asphyxia being the causal factor. As sources of information, he

used the history from the plaintiff, medical records, road to health card, obstetric and neonatal records and an MRI scan by Prof Lotz and Dr Swartzberg, in order to compile his report dated, 13 November 2018. After examining ES, he concluded that she has cerebral palsy called Dyskinetic Cerebral Palsy, a type of injury that is '*highly specific for injury occurring during labour*'. She has no functional use of the hands, severe expressive and receptive impairment and microcephaly (small head). She appeared to be able to see and hear and has no history of epilepsy.

[38] Dr Kara gathered from the antenatal records that the plaintiff had no risk factors except a history of epilepsy. The antenatal records showed that her first antenatal visit was on 11 February 2014, although she told him that she visited the clinic before that date. There was also a comment that she booked late. In February 2014, it was estimated that she was 32 weeks' gestation. A month later, she was estimated to be 36 weeks' gestation. It appeared that there was no concern about the foetal condition because she was epileptic and therefore was a high-risk pregnancy and that her delivery should be managed in a hospital, which was done.

[39] Regarding decelerations, Dr Kara explained that a variable deceleration is a little more suspicious of foetal compromise. He contended that late decelerations are an ominous and highly suspicious sign that there are concerns about the foetal condition, and that foetal compromise is highly suspicious. However, the attending doctor looked at the CTG and said the decelerations were not late but early, with good recovery. According to Dr Kara, this should have been a concern to the doctor as the FHR was dropping to about 100 beats per minute, hence he was called. He confirmed Dr Ndjapa's evidence that referring to the CTG as '*up and down*', is not a medical comment. Dr Kara remarked that the plaintiff's progress

from 3cm dilated to full dilatation in 40 minutes was very rapid and concerning. He stated that considering that late decelerations were picked up by the CTG from 17h20, as well as the comment that the CTG was up and down, clearly showed that the doctor observed the condition of the plaintiff and the foetus. However, the records of the summary of labour made no comment about foetal distress. The parthogram which commenced at approximately 19h00 recorded a normal FHR at 19h00, 19h30 and 20h00, different from what the CTGs showed. Concerning was the observation that the baby was flat, floppy and had a tight cord around the neck twice, as well as that the placenta looked healthy, but there was no comment about foetal distress or that the placenta looked healthy. Regarding the neonatal record, Dr Kara found it strange that the initial examination noted that the baby had caput, no respiratory, distress, normal tone, weak and absent primitive reflexes, and normal cry. According to Dr Kara, this is unusual to have weak reflexes, and normal cry, a normal tone and no respiratory signs in a baby that had been resuscitated. He mentioned that the discordance between what the records said, and interventions done to the baby, was a means of minimising injury to foetuses.

[40] Dr Kara explained that when a foetus is in the mother's womb it gets its oxygen from the mother *via* the umbilical cord and passes it through the placenta. When the uterus contracts it cuts the blood supply to the foetus and that gives relative oxygen deficit to the foetus. When the baby has compensatory mechanisms to cope with the short period of lack of oxygen and when the contraction stops, the oxygen flows back into the baby. The baby will be fine because that period of reperfusion restores the oxygen supply to the baby and protects it for the next contraction when the oxygen is going to be reduced. This is a normal consequence of labour.

[41] In abnormal labour, there is an accumulative deficit in the oxygen supply to the baby with each contraction. For instance, if the foetus has 100% oxygen before the contraction and the oxygen is stopped during a contraction, the oxygen saturation drops to 50%. The oxygen gets restored to the foetus. Before the next contraction, the foetus would have saved 95%, which would not be 100% anymore. When next is a prolonged contraction and the period for oxygen to be restored after the contraction is shorter for example, 90%, this means that over many contractions there is an accumulative deficit of oxygen supply to the foetus. The foetus will compensate by shutting down blood and oxygen supply to non-critical organs like, the lungs, liver, kidneys etc. Eventually, as the oxygen deficit becomes more and more it affects the oxygen supply to the heart and the heart will then stop pumping. That is what manifests by foetal heart tracings because the foetal heart tracings record that the heartbeat during a contraction starts dropping and that is a sign that there is foetal compromise. That is the reason why the foetal heart should be monitored during labour.

[42] Dr Kara continued that as the foetal heart compromise increases, it affects the blood supply to the brain. If this is a gradual process, the brain compensates for this by shutting down the oxygen and blood supply to the non-critical areas of the brain and preserving oxygen and diverting that blood and oxygen to areas of the brain necessary to maintain life, like areas of thought, movement, function etc. As that compensation fails further because of continued hypoxic ischemia, only then will there be damage to those high-functioning areas of the brain. He explained further that the first area of the brain that gets damaged in ongoing persistent hypoxia, is the cerebral cortex and the term of the injury is called Watershed Hypoxic-Ischaemic Injury. When that compensatory mechanism fails, that injury extends to

the deep nucleus of the brain called the Basal Ganglia and Thalamus and then there will be what is called a pattern of injury, which ES had due to a long-standing process of hypoxia.

[43] Dr Kara observed that the maternity and neonatal notes were disorganised. He explained that Hypoxic-Ischaemic Injury is what happens to the brain and also means the effect of the injury. He added: *“If you have to take HI Encephalopathy and brain injury in term or near term babies the most common period for that injury to occur is immediately before, during or immediately or soon after labour. ...If they assessed the foetal condition to be normal on admission to the hospital or clinic to the best of their ability, of course, they assessed that they were of the opinion that the foetal condition was normal. That means that it is highly unlikely that there was brain injury immediately prior to her presentation at the hospital or clinic. She presented at the hospital at 14h00 and so it is highly unlikely that an injury occurred at 09h00 or 10h00 that morning.”*

[44] Regarding the definition of long-standing injury, Dr Kara opined that it varies from one and a half to two hours, maybe three. In others, it may be five or six hours. He asserted that one cannot say that in each baby a prolonged injury occurs over a short or long period of time, it is unknown but it can be said that it does occur over hours.

[45] Concerning the MRI scan, Dr Kara stated that ES was born with a cerebral palsy and there is an 80% probability that the injury was due to hypoxic ischemia because she had a specific type called Dyskinetic Cerebral Palsy which is very commonly associated with

hypoxia. That is what the attending doctor who managed the plaintiff at birth, thought, which was confirmed by the MRI scan. He testified that taking that together with the condition of the child at birth, it makes it highly probable that this injury occurred during labour. That is so because when the plaintiff was admitted there were no concerns over the foetal condition until 17h20 when the CTG was abnormal. That is the time when the foetus was not in optimal condition.

[46] During cross-examination, Dr Kara disagreed with the opinion that being growth restricted on its own causes hypoxic injury during a normal labour. According to him, it is a factor that makes it more likely. He agreed that the labour process is hypoxic and that is why monitoring is important in order to work out what is normal and abnormal and in an increased risk, an intervention would be made. Even though he was uncertain about the gestational age of the baby, he opined that in the last four weeks of the plaintiff's pregnancy, foetal growth seemed to have progressed normally which does not accord with a growth-restricted foetus.

[47] Dr Kara compiled a joint minute with Prof Cooper, on 15 August 2020. Dr Kara concluded that both components of the hypoxic-ischaemic injury occurred in the labour, as indicated earlier. Prof Cooper agreed that the acute profound component of the injury probably occurred in labour during the last 45 minutes prior to delivery. Regarding the partial prolonged component of the hypoxic ischaemic brain injury, Prof Cooper's opinion was that in view of the significant asymmetrical intrauterine growth restriction and poor antenatal attendance with lack of information regarding foetal well-being during the last four weeks of pregnancy, the chance of the partial prolonged component occurring prior to labour, is substantial. There was no basis for this opinion as no factual evidence was led to support it.

About Prof Cooper's opinion that the partial prolonged injury could have occurred prior to labour, Dr Kara stated that this is possible. However, he stated that there is little reason to support this possibility. He mentioned that the initial assessment in labour indicated no concern over the foetal condition. He contended that asymmetrical growth restriction increases the risk of intrapartum asphyxia but is not an independently significant risk factor for antenatal asphyxia.

[48] Regarding prematurity, Dr Kara and Prof Cooper agreed that this might increase the risk of intrapartum hypoxia ischaemia but would not be the cause of brain injury. With appropriate foetal monitoring, the increased risk of brain injury in labour due to prematurity, could be anticipated.

[49] When it was put to him that the measurements of the baby at birth pointed more to it being born at term than 36 weeks, Dr Kara disagreed. He intimated that when the plaintiff visited the clinic on 11 March 2014, no plotting as to the measurements of the baby were made showing whether the baby was growing or not and so it could not be said that it was 34 weeks as written in the graph. That is because there was nothing to validate this opinion. Therefore, no one could say with certainty what the gestation period of the plaintiff's baby was. The plaintiff did not know when she had her last menstrual periods. Dr Kara did not necessarily accept that the pregnancy was carried to 40 weeks' gestation. He stated that if it were, the baby would have been growth restricted. He therefore did not give an opinion on the gestation period as it was unknown. He gave a scenario that if the baby was 38 to 40 weeks, then the baby was asymmetrically growth restricted, which is usually on the basis of late placental insufficiency. He accepted that when the plaintiff visited the clinic for the first

time the gestation period was estimated at 32 weeks and 36 at delivery. He said that if the plaintiff was 36 weeks at delivery, the weight of 2.5kg is on the 50th centile, the length of 49 cm is below the 90th centile and the head size of 34 cm is on the 90th centile. According to him, this is not supportive of IUGR.

[50] Dr Kara testified that it was highly unlikely that the plaintiff's epilepsy or her treatment caused ES's cerebral palsy as there was no evidence to support this. He stated that a concern of sepsis was highly unlikely to result in brain injury considering the poor management of labour. The MRI scan features are not reflective of an injury pattern of infection.

[51] Regarding sepsis, Dr Kara asserted that the records do not show evidence of sepsis although there was a comment about sepsis in the records. He asserted further that a concern of sepsis is highly unlikely to result in brain injury considering the poor management of the labour. He added that the MRI scan features were also not reflective of an injury pattern of infection.

THE DEFENDANT'S CASE

THE EVIDENCE OF DR MTSI

[52] Dr Mtsi compiled an undated report using the instruction letter from Janilite Medico-Legal, summons, hospital records, Medico-Legal reports of Dr Mugerwa-Sekawabe, a gynaecologist, Dr Kara, MRI report by radiologists, protocols and literature. She testified that the clinical notes showed that the plaintiff was a late booker. That is because she booked at Mpunzana Clinic on 11 February 2014 for the first time and went into labour on 11 March 2014. Dr Mtsi confirmed Dr Ndjapa's evidence that the antenatal graph was not plotted on 11 February 2014, when she presented to the clinic at 32 weeks of gestation. She substantiated this by saying that according to the protocol, when a patient is seen for the first time, nurses should start with SF measurements showing how big the foetus is. That was important to monitor the growth of the foetus. However, she said that it would not have made a difference because, on 11 March 2014, the baby was delivered. When it was put to her that Dr Ndjapa was adamant that proper plotting, even at that late stage would have been of assistance, she agreed. However, she asserted that to monitor the growth of a foetus, several antenatal visits are required. For instance, if in this case there were two or more visits plotted, that would have established a pattern. One spot on the graph and an attempt to do a second one would not assist because there would have been one spot. On 11 March 2014, the graph was also not plotted at the Mpunzana clinic. Dr Mtsi opined that the plaintiff did not present at the clinic due to labour pains and the graph should also have been plotted because that could have given an impression of poor growth. She opined further that failure to plot was therefore wrong.

[53] Dr Mtsi stated that although on the morning of 11 March 2014, the plaintiff was not in labour, had the nursing staff plotted the antenatal graph, they would have been able to suspect that there may have been intrauterine growth retardation. She later changed and said that '*the graph is useful if there were a number of antenatal visits, two visits will not help that's why*

I'm trying to indicate to you an error for instance already that is done in the first and the second visit.'

[54] It was difficult for Dr Mtsi to interpret the medical records suggesting bradycardia at 17h20, on 11 March 2014, saying that no indication of the duration of the condition was given. According to her, '*it was absolutely unclear*'. She said that before defining bradycardia, she would have to see the CTG that showed bradycardia at 17h20 and could not assume that it was more or less than ten minutes because she did not know. She therefore admitted that the onset of bradycardia was unknown. That is because between 15h00 and 17h00 there was no account as far as the foetal and maternal well-being was concerned. Dr Mtsi stated that the ones who managed the plaintiff's labour ought to have been alive to the fact that they were dealing with a distressing foetus, the onset of which was unknown to them. The unknown was a cause for vigilance in the management of labour. She asserted that the cause of foetal distress was not investigated. However, she maintained that when bradycardia was established, the defendant's employees acted appropriately.

[55] Dr Mtsi confirmed that once bradycardia is picked up, the cause and why it happens should be investigated in order to determine what intervention to make. Then it should be ascertained whether or not the condition responds to the correction and correct it if it does. She admitted that the investigation of the cause of bradycardia is also important as it tells the clinician that he/she must determine the timing of the delivery, the objective of which is to avoid prolonged foetal hypoxia. This confirms Dr Ndjapa's evidence. She contended that a caesarean section is indicated when there is foetal tachycardia, bradycardia, the reduced variability and late decelerations, at the same time. She contended further that it does not

happen in obstetrics that when only one parameter is diagnosed then a caesarean section is done. She later altered her version when I asked her questions to clarify certain aspects of her evidence and agreed that bradycardia is the opposite of tachycardia and that these two cannot coexist at the same time but might exist in quick succession.

[56] Regarding CTGs, Dr Mtsi stated that the quality of those that she worked on, were very poor for proper analysis. She stated further that there was an element of guess work which was unacceptable for CTGs. She had to use a lot of imagination, an incorrect way of interpreting CTGs. She contended that the fact that there was a lot of interference with the CTGs, they were not the typical CTGs that were supposed to be interpreted, especially in the management of a patient that ended up being unwell. She found it very difficult to give proper evidence due to the poor quality of almost all the CTGs. She suggested that either the doctor or nurses who saw the monitor and the CTGs progressing in front of them, had an advantage, compared to her. Otherwise, she had to speculate as she was struggling to interpret the same, which was unfair on both sides.

[57] Dr Mtsi opined that a normal CTG does not necessarily mean that there should absolutely be no decelerations, thereby disagreeing with Dr Ndjapa about his classification of a normal CTG. According to her, once there is a repetition of decelerations, the CTG falls into category 2, which is suspicious or category 3, pathological. She contended that before a caesarean section is done, a suspicious CTG should be observed for a long time and seen to be worsening. That is so because a suspicious CTG, in most cases, is not an indication for an immediate caesarean section. According to her, decelerations are an indication that there could be a problem or something worse than or equally as bad as decelerations, beat-to-beat

variability. When assessing a CTG, attention is not only given to decelerations but variability is also as important. About reduced variability, she stated that one would have to *'go back and investigate what treatment was the patient on and was she on something that is paralysing the central nervous system.'* Concerning CTG 127, 128 and 129, she identified no evidence of reduced variability and stated that same did not qualify as category 3 (pathological) CTG. She identified a slight variable deceleration in-between beat-to-beat variability and according to her, that was normal.

[58] It was difficult for Dr Mtsi to accurately assess the lateness of the decelerations or variable ones. She lamented the poor state in which the CTGs were. She disagreed that the late decelerations identified by Dr Ndjapa in the first CTG, were indeed late. She said that it *'does not show any reduced variability in the first place.'* Instead, she identified contractions which she described as those that could not really be used to assess whether or not the decelerations, she identified, were early or late. She identified one deceleration that she said would have raised questions in terms of its nature, length and time it took to recover. She noticed, however, that throughout that deceleration, beat-to-beat variability was still very normal such that there was a good response which showed a healthy baby. That is because a baby that has decelerated, would not respond with a good beat-to-beat variability. In her reading of the CTG, there was no compensation that had to be done by the foetus. She stated that she would have been extremely worried if the foetus' recovery had gone up to 160 or down to about below 100, which is 80 because then that would have been an indication of severe bradycardia and tachycardia.

[59] During re-examination, Dr Mtsi testified about degrees of foetal distress and that not every early sign of foetal distress requires intervention by way of a caesarean section. However, she accepted that foetal distress develops over time and may move from less to severe.

[60] During cross-examination, Dr Mtsi identified a variety of decelerations in the first CTG, which she said were not late but had good beat-to-beat variability. She added that in this CTG she could find an area of interference with the accuracy of the CTG. She thought that, that could have been caused by the maternal pulse rate that was picked up as it is known to cause decelerations. It was difficult for her to interpret another deceleration that she identified in the second CTG. It was further difficult for her to comment on the third and fourth decelerations. In all, she identified four decelerations and could only interpret one. Regarding the second CTG she identified five decelerations which she could not interpret and one which she classified as early.

[61] Dr Mtsi conceded during cross-examination that at 19h05 Dr Glaze issued an instruction that the plaintiff be closely monitored, which was not done. She accepted that this was not in keeping with the doctor's instructions. She further conceded that the fact that no one could account for the foetal condition and the plaintiff's progress from the time the doctor gave instructions in terms of her dilatation, was a problem and constituted substandard care. When she was asked to reconcile the fact that in her reports she said there was no substandard care, she said: "*I do not know how to respond to that.*" She conceded further that the CTG at 20h00 showed a situation that had deteriorated and at that time, nothing could be done by way of caesarean section to save the baby as that was not the onset of the problem,

thereby confirming the evidence of Dr Ndjapa. She later deviated and stated: “...*I think we are both guessing sir because I do not know, and I am unable then to say that that was the onset of labour or not the onset.*” However, she agreed with Dr Ndjapa that the CTGs remained suspicious and were not normal.

[62] When Dr Mtsi was asked whether a caesarean section would be appropriate if the CTG was suspicious, she referred to the suggested management by Figo Consensus Guidelines on Intrapartum Fetal Monitoring (referred to in the trial as the FIGO document) as well as her experience, were that when one has got a suspicious trace, one gives it enough time and action to correct reversible causes, if identified. She added: “...*[E]nough time is if you see severe deterioration from these CTGs then you know that no I’m not going to continue here but a suspicious CTG is highly unlikely to result in hypoxia or acidosis in the fetus so you’ve got time to assess, re-assess and re-assess so action to correct a reversible cause had been continuing...*”

[63] Concerning the nuchal cord around the foetus’ neck twice, Dr Mtsi explained that the baby’s head was 3/5 up as late as twenty hours and suddenly dropped to 1/5 in 40 minutes according to the nurses’ description. She thought that the sudden drop with the tight cord would subject the baby to a tightening cord and cause a little bit more lack of oxygen. She added that a cord may be two times around the neck at delivery without causing much damage but in the circumstances that she described, it would cause more danger. She did not think that the nuchal cord was tight at all stages. She admitted that if the nuchal cord was tight around the neck twice, the nurses could have picked that up that there was something wrong with the FHR if they monitored it. In her opinion, only after the plaintiff became fully

dilated did the head drop rapidly and it is when the problem was exaggerated. She confirmed that her proposition was speculative.

[64] During cross-examination, Dr Mtsi agreed that had those who managed the plaintiff's labour decided to perform a caesarean section at 19h30, they would have had enough opportunity to deliver the baby, given the fact that the actual delivery was at 20h55.

[65] When Mr Bodlani put to her that intrauterine growth restriction is a risk factor but did not cause the condition, Dr Mtsi could not answer. However, she conceded that not every baby born growth restricted has cerebral palsy. She conceded further that a growth restricted baby has problems coping with labour and would indicate with a depressed heart rate, except that in certain circumstances they respond more aggressively.

[66] When she was asked about the possibility of chorioamnionitis (inflammation or infection), Dr Mtsi testified that there was no evidence or diagnosis of chorioamnionitis but vaginal discharge. The placenta was not examined. She quoted from page 281 of an article called the South African Medical Journal which states: "*Placental mediated disease can result in hypoxia or foetal priming for hypoxia with some degree of IUGR present when labour starts.*" However, she conceded that there was no confirmed case of placental insufficiency, in the instant case. She confirmed further that the clinical finding was that the placenta was normal.

[67] Dr Mtsi confirmed that according to the clinical notes, ES was born distressed and was resuscitated at birth and given oxygen and put in a box immediately after birth. She was assisted to breathe.

[68] Dr Mtsi compiled a joint minute with Dr Ndjapa dated 11 March 2020. The importance of this joint minute were the following concessions made by Dr Mtsi that:

68.1 There was no plotting onto the antenatal graph, the growth chart was completely blank and it was unknown on which centile the pregnancy growth was;

68.2 The parthogram on page 12 of the clinical record suggested that at 14h30 the plaintiff was 3cm dilated with head level 3/5 above the pelvis membrane still intact having moderate contractions and was transferred from the latent phase of labour on to the alert line of the labour graph, suggesting that she had entered the active phase of labour as seen on the parthogram;

68.3 At 17h20 the parthogram was not plotted. It was evident that if the nursing staff had plotted it with the information as required, they would have noted that the plaintiff had crossed the action line and having in mind, on record that FHR's abnormalities were present, it was necessary that immediate action be taken by delivering the baby by the fastest route and this would have been by caesarean section and avoid unnecessary delay and further intrauterine exposure to foetal hypoxia;

68.4 The parthogram was empty, no records between 14h30 until the time of delivery. The only CTG suggesting foetal monitoring at any point available for comment were those that were performed between 18h00 and 18h30 and these were also non-reassuring therefore not only suggesting a substandard care during labour, but also suggesting that the baby may have been exposed to intrauterine hypoxia over a long period of time, long enough to have to result

in birth asphyxia and cerebral palsy. Further to that, the doctor was informed about the patient not progressing and the CTG abnormalities but did not come to assess the plaintiff personally. The parthogram was poorly filled leading to poor decision making;

68.5 At 20h50 the plaintiff was fully dilated while in the clinical record it was said that the plaintiff was fully dilated at 20h00 suggesting an incoherence in the records. Nevertheless, it appeared that five minutes later she delivered ES at 20h50 with Apgars of 5/10 and 6/10 who did not cry at birth and also had a tight cord around the neck, twice;

68.6 Perinatal asphyxia, also known as hypoxic ischemic encephalopathy (lack of oxygen), ischemic (lack of blood flow) and encephalopathy (brain damage) was characterised by clinical and laboratory abnormalities with evidence of acute or sub-acute brain injury due to asphyxia. The primary causes of this condition were systemic hypoxemia and/or reduced cerebral blood flow;

68.7 Perinatal morbidity associated with hypoxic ischemic encephalopathy could occur in the antepartum, intrapartum or postpartum period. In many cases this reflected inadequacies in the antenatal care, the decision making as well as poor intrapartum and immediate postpartum care. The quality and timing of antenatal care with delivery plan as well as an appropriate intrapartum obstetric care was key in preventing perinatal morbidity such as birth asphyxia and cerebral palsy;

68.8 There was substandard care on the labour monitoring and management of the plaintiff. There was delay in taking action when the FHR rate was noted to be bradycardic. As a result of inappropriate recording and plotting on the labour graph, the hospital staff failed to diagnose labour dystocia and therefor failed to take action when it was necessary to do so;

68.9 ES was born a premature. She did not cry at delivery and required resuscitation, had cooling and apparently seizure witnessed by the plaintiff was assessed by the paediatrician as

cerebral palsy dominantly dyskinetic. Antenatal causation including infective causes as well as infant factors had been excluded as the probable cause attributable to ES's cerebral palsy by both paediatricians and the MRI report from the radiologists suggesting that the damaging factor most probably occurred during labour and delivery. Dr Mtsi added: "*I agree that probably the insult occurred during labour but I do not have information regarding the exclusion of risk factors like prematurity, infection and epilepsy. My conclusion on that is that I will leave it to the multidisciplinary team to decide on that issue.*" (Her emphasis added)

Dr Mtsi further agreed with Dr Ndjapa that:

68.10 The foetal condition was inappropriately monitored suggesting that the foetus might have been exposed to hypoxia over a period of time, long enough to cause cerebral damage and resulting in cerebral palsy. The outcome at delivery (poor agpar score, the need for resuscitation and admission to ICU, seizures in the first 24 hours) suggested that the damaging event might have occurred during the intrapartum period. There was no evidence to suggest that the damaging event may have occurred in the antenatal or postpartum period even though the plaintiff was a known epileptic there was no scientific evidence to suggest that ES's cerebral palsy might have been attributable to her epileptic treatment; and

68.11. The poor outcome of ES might have been prevented had proper obstetric care been provided by Zithulele Hospital staff to the plaintiff and delivery of ES, expedited. Dr Mtsi again added: "*I agree that it may have been prevented but again we need a **multidisciplinary team to determine the causation.***" (Her emphasis added)

[69] In another joint minute compiled by Drs Mtsi and Ndjapa dated, 25 January 2021, unsigned by Dr Mtsi, she made a turnaround regarding her position, especially after receiving

information she claimed she did not have when she signed the joint minute dated, 11 March 2020. She said that she did not have all the information as well as CTGs and was therefore unable to make a proper assessment. Strangely, she conceded that as an expert, she has a responsibility to be independent when expressing an opinion, equip herself with all the relevant information before preparing a medico-legal report and comment on a joint minute. By appending her signature to the joint minute, she was communicating that she knew what the case was all about as she had all the relevant information at her disposal and that the contents of the joint minute were a true reflexion of her understanding of the case she was dealing with. She further conceded that, that was the position when she committed to the report that she prepared preceding the time she entered into the joint minute.

THE EVIDENCE OF PROF COOPER

[70] According to Prof Cooper, a neonatologist and head of the department of paediatrics, ES was growth restricted. That is because she was a size below the 10th percentile of babies her age, at the time of birth. He regarded ES as a 39 to 40 weeks' gestation, hence he concluded that the birth parameters of ES were asymmetrical intrauterine growth restriction which was usually on the basis of late placental insufficiency. About late booking for antenatal visits, he said:

"...I think once booking is as late as she did, the only thing one really has got to rely on is her recollection of the last menstrual period. Which I think Dr Kara and I quite agree, may or may not be accurate. But it makes it extremely difficult to assess intrauterine growth restriction because you really need to be following a pregnancy over months, not over weeks, in order to pick that up. So in terms of picking up growth restriction and a whole range of other problems, this would be compromised, severely compromised."

[71] Prof Cooper asserted that to get a good reasonable idea of the gestational period, a mother has to attend an antenatal clinic at least before 20 weeks of gestation because even sonar cannot give an accurate estimation of gestational age. He asserted further that it would be virtually impossible for the attending staff to detect IUGR as the plaintiff was a late booker.

[72] Commenting on the MRI scan, Prof Cooper stated that the partial prolonged injury sometimes occurs over at least an hour or hours or even days while acute profound occurs in just over 45 minutes. He asserted that during a contraction there is a compromise in oxygenation, nutrients and blood flow across the placenta. This is called an episode of partial hypoxia because a contraction lasts about 30 to 45 seconds. During this period there are usually enough reserves in each cell as each cell has an emergency supply of glucose which is sugar and energy. In some cases, this may occur before labour when there are episodes of poor perfusion particularly in the face in the context of IUGR or placental insufficiency. This pattern of brain injury will occur over at least an hour or many hours or sometimes even days.

[73] Prof Cooper, however, stated that in acute profound hypoxic-ischemic injury where there is sudden severe cut-off, an almost complete cut-off of blood flow to the brain, the compensatory mechanism cannot function. The whole brain gets affected and the damage may start occurring within ten minutes if it carries on for more than 45 minutes and the baby will not survive because the central parts of the brain are the most active. They will be severely affected. At times, the partial prolonged injury may have started and as time progresses, it goes into a complete shutdown in perfusion of the brain and that is when one gets the mixed pattern of both partial prolonged and acute profound.

[74] Regarding CTGs, Prof Cooper remarked that it was the domain of obstetricians to interpret and act upon what they observe. In his report dated 01 June 2020, he concluded:

“5. [ES] suffered both a partial prolonged and acute profound hypoxic ischaemic brain injury during the peripartum period around term gestation. The partial prolonged component of the injury could have occurred prior to labour in view of the fact that she had significant intrauterine growth restriction, but may also have occurred during labour. The acute profound component of the injury probably occurred during the 45-minute period prior to delivery. Expert obstetric opinion is needed to determine whether there were any signs of fetal distress and whether brain injury was avoidable.”

[75] Prof Cooper contended that the acute profound component of the injury must have occurred anywhere between 10 and 45 minutes, may be even 50 prior to delivery. He conceded that there was neither recognised sentinel event nor comment made at birth, that the baby was growth restricted. He conceded further that in order to put a baby on a particular centile, gestation in weeks should be known. At their disposal, they had an assessment of the symphysis fundal height that was said to be 32 weeks, four weeks before delivery and last menstrual periods that gave the gestation of 40 weeks. After looking at the parameters, he found that 36 weeks’ gestation period was highly unlikely and that the strong probability was that they were looking at a term baby of 39 to 40 weeks. He confirmed that there was no evidence of placental insufficiency as the placenta was not examined.

[76] It is important to note that the defendant did not lead direct evidence as far as the clinical and hospital records were concerned although both parties made extensive reference to same during the leading of evidence. Koen J in *NH v MEC for Health KZN*,³ had the following to say regarding medical records:

³ (1287/2014) [2018] ZAKZPHC 8 (4 April 2018).

[8] Statements in the medical records that are favourable to the Defendant are hearsay where the author thereof was not called to testify, and hence not admissible⁴. ...No application was made for the admission thereof in evidence in terms of section 3 of the Evidence Law Amendment Act 1998, but even if there was, it would be unlikely to have succeeded as there was no evidence that the author thereof was no longer available to give that evidence....

[9] Recordings favourable to the Plaintiff's case in establishing negligence and liability generally, and accordingly damaging to the Defendant's case, made as part of the records kept by the Defendant's servants, are however on a different footing. They constitute admissions by the servants of the Defendant made in the ordinary course of discharging their duties, which are binding against the Defendant.⁵ The Defendant's staff are obliged to make these statements by recording the medical position as it unfolds in the records. They have an obligation to speak on behalf of the Defendant and dispute what is recorded, if indeed incorrect."

[77] With the above information in mind, I have to accept the medical records favourable to the plaintiff, as admitted by the defendant. I also have to keep in mind that a medical practitioner is not expected to bring to bear upon the case entrusted to him the highest possible degree of professional skill, but he/she is bound to employ reasonable skill and care as provided for by Innes CJ in *Van Wyk v Lewis*.⁶ Importantly, the medical records show no record before bradycardia was noted at 17h20 after the plaintiff was admitted at Zithulele Hospital on 11 March 2014. This means that for about two hours there was no monitoring of either the plaintiff's or the foetus' well-being.

NEGLIGENCE AND AUTHORITIES

⁴ See DZ Zeffertt and AP Paizes Hoffman and Zeffertt's the South African Law of Evidence 4th ed, at 183ff.

⁵ DT Zeffertt and AP Paizes Hoffman and Zeffertt's the South African Law of Evidence 4th ed, at 183ff.

⁶ *Van Wyk v Lewis* 1924 AD 438 at 444.

[78] In the instant case, the plaintiff relies on negligence and must therefore establish it. If at the conclusion of the case the evidence is evenly balanced, she cannot claim a verdict, for she will not have discharged the *onus* resting upon her.⁷ In *Mitchell v Dixon* 1914 AD 519 at 525 Innes ACJ said:

“A practitioner can only be held liable in this respect, if his diagnosis is so palpably wrong as to prove negligence, that is to say, if his mistake is of such a nature as to imply absence of reasonable skill and care on his part, regard being had to the ordinary level of skill in the profession.”

[79] In *Kruger V Coetzee 1966 (2) SA 428 (A)*⁸ Holmes JA (Beyers ACJ, Van Blerk JA, Botha JA, Wessels JA concurring) held:

“For the purposes of liability *culpa* arises if -

- (a) a *diligens paterfamilias* in the position of the defendant -
- (i) would foresee the reasonable possibility of his conduct injuring another in his person or property and causing him patrimonial loss; and
 - (ii) (ii) would take reasonable steps to guard against such occurrence; and
- (b) the defendant failed to take such steps.”

[80] *In casu*, it was undisputed that the standard of care that the defendant was required to provide to the plaintiff and ES had to meet amongst others, the applicable Guidelines, as alluded. The relevant provision *inter alia*, relates to foetal distress and its management,⁹ which states:

“FETAL DISATRESS

This is suspected when the following signs are observed:

- Baseline fetal heart rate \geq 160 beats per minute
- Baseline fetal heart rate \leq 110 beats per minute
- Variability persistently $<$ 5 beats per minute on CTG, in the absence of sedating drugs
- Late decelerations of the fetal heart rate

MANAGEMENT OF FETAL DISTRESS

1. Explain the problem to the mother

⁷ Van Wyk *supra* at 444.

⁸ At 430e.

⁹ At page 55 of the 2007 Guidelines.

2. Lie the mother in a left lateral position
3. Give oxygen b face mask at 6 L/minute
4. Start an intravenous infusion of Ringer-Lactate to run at 240 mL/hour
5. Do a vaginal examination for cervical dilatation and to exclude cord prolapse:
 - If vaginal delivery is imminent (cervix fully dilated), deliver immediately, by vacuum extraction if necessary
 - If vaginal delivery is not imminent, give hexoprenaline 10 micrograms IV and prepare for immediate caesarean section. Arrange urgent transfer from a community health care centre to hospital.” Emphasis added

[81] During the trial, it was undisputed that the plaintiff was put on a CTG at 17h00 and there was no continuous monitoring. That is because according to the plaintiff, a nurse would come, look at the screen and leave. The clinical notes showed that intrauterine resuscitation was done after bradycardia was noted at 17h20. The plaintiff was 3 cm dilated at the time, which meant that vaginal delivery was not imminent. There was no indication that she was given hexoprenaline 10 micrograms IV and was also not prepared for an immediate caesarean section, as provided for in the Guidelines. During the period between 17h20 and 18h20 the plaintiff was not monitored whilst on CTG, as stated. No nursing staff was with her and therefore no one knew what was happening to her.

[82] After the doctor was again informed of the foetal condition at 18h45, he/she did not personally come to assess the condition. Instead he/she gave instructions that the then-current intervention be continued which had been applied since 17h20. At 19h00 decelerations were noted about three times, despite the management. The doctor was again informed and requested to come and assess the plaintiff, the FHR was still dropping and intrauterine resuscitation was continuously applied more than an hour later although there was clearly no progress of labour. The plaintiff and the foetus remained unmonitored after 19h05 after the doctor had given an instruction to monitor her closely. It was clear that FHR was not normal all this time. This was common cause between Drs Ndjapa and Mtsi that at no stage was the

CTG normal from the time bradycardia was noted and after intrauterine resuscitation was started. Notwithstanding this condition, the plaintiff's labour continued as if nothing wrong was noted. The fact that the foetus was suffering from hypoxia was known by the nursing staff and the doctor, who was called at 17h20 and 18h45. According to the Guidelines, they were aware that vaginal delivery was not imminent and they were supposed to have given the plaintiff hexoprenaline 10 micrograms IV and prepared for immediate caesarean section, which they did not do. There was no need to transfer her to a hospital as she was in the hospital already. Moreover, according to the Guidelines, foetal distress is one of the common indications for caesarean section, which was disregarded by the defendant's employees.

[83] During the cross-examination of Dr Ndjapa, the defendant did not challenge his evidence of what in his opinion, constituted good FHR recovery in obstetrics. It was never put to him that what he identified as late decelerations and its characterisation in the first CTG, were in fact not and no reason was advanced as to why it would not be late decelerations. It was the case even when he testified that the late decelerations were starting to be pathological and that there had been no normal CTG. He that he identified four late decelerations and elements of poor variability in the second CTG. This was also undisputed. The defendant did not put it to Dr Ndjapa that a caesarean section was indicated only when there is bradycardia, tachycardia, reduced variability and late decelerations, at the same time. Evidence that has not been challenged by the defendant, stands uncontroverted and strengthens the plaintiff's case.

[84] Dr Mtsi testified differently from what she stated in her Medico-Legal report. She adapted her evidence as the trial progressed, as shown above. She failed to answer pertinent

questions put to her. In my view, she did not assist the court but gave evidence in order to protect the defendant. Her opinion was therefore not independent. She, however, conceded that the management of the plaintiff's labour was substandard. She changed her stance after the case for the plaintiff had been closed. This conduct cannot avail the defendant.

[85] On the other hand, Prof Cooper's theory of IUGR can also not stand due to the fact that the gestational period of the plaintiff was unknown, which he conceded. This theory was intended to conclude that the baby was born at term, which fact is unsupported by the evidence. That is because when the plaintiff presented at Mpunzana Clinic on 11 February 2014 and 11 March 2014, the antenatal card was not properly completed, as plotting was not done. Moreover, the plaintiff was uncertain about her last menstrual periods, as alluded. The gestation period was therefore not established. Prof Cooper relied on hearsay. In *Mathebula v RAF (05967/05) [2006] ZAGPHC 261 delivered on 08 November 2006*, Meyer JA, as he then was, remarked:

“An expert is not entitled, any more than any other witness, to give hearsay evidence as to any fact, and all facts on which the expert witness relies must ordinarily be established during the trial, except those facts which the expert draws as a conclusion by reason of his or her expertise from other facts which have been admitted by the other party or established by admissible evidence.”

CAUSATION

[86] The relevant questions are: (i) what was the factual cause of the ultimate condition of ES; and (ii) did the negligence on the part of the employees of the defendant cause or materially contribute to the brain injury suffered by her in the sense that the respondent, by the exercise of reasonable professional care and skill, could have prevented it from developing. Dr Mtsi was long-winded in her explanations and concentrated on the maternal rather than foetal condition. This evidence was not led in her evidence-in-chief.

[87] In this regard, Nkabinde J (Moseneke DCJ, Froneman J, Jafta J and Van der Westhuizen J concurring) in *Lee v Minister of Correctional Services 2013 (2) SA 144 (CC)*¹⁰ remarked:

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

[88] The learned Judges continued at para [41]:

“[41] ...[I]n 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 the strict application of the rule would result in an injustice, hence a requirement for flexibility.”

[89] In order to apply this test one must make a hypothetical enquiry as to what probably would have happened but for the wrongful conduct of the defendant. This enquiry may involve the mental elimination of the wrongful conduct and the substitution of a hypothetical

¹⁰ At para [38]; see also *International Shipping Co (Pty) v Bentley 1990 (1) SA 680 (A)* at 700E-H.

course of lawful conduct and the posing of the question as to whether upon such a hypothesis plaintiff's loss would have ensued or not.¹¹

[90] In the instant case, for the defendant to be held liable for the act or omission committed by its employees it must be proved that the injury sustained by ES was reasonably foreseeable and the defendant's employees failed to provide the level of skill and competence that would otherwise be expected to be provided by reasonable health care employees in the circumstances. However, a plaintiff is not required to establish the causal link with certainty, but only to establish that the wrongful conduct was probably the cause of the loss, which calls for a sensible retrospective analysis of what would probably have occurred, based upon the evidence and what can be expected to occur in the ordinary course of human affairs rather than an exercise in metaphysics.¹²

[91] Dr Ndjapa testified that the defendant's employees delayed in taking action at the time when the FHR was bradycardic. Due to inappropriate recording and plotting on the labour graph, they failed to take action when it was necessary. They were therefore negligent in their treatment of the plaintiff as they failed to timeously deliver the baby by caesarean section. It was clear that the foetus was in trouble at 17h20 and they unnecessarily allowed labour to continue without appropriate monitoring, especially between 14h30 and 18h00. That is because at 18h30 the CTG was non-reassuring and delivery was essential to prevent further exposure of the foetus to hypoxia and brain damage. Dr Mtsi was of the opinion that there was no negligence in the management of the labour as monitoring was appropriate and

¹¹ International Shipping Co (Pty) Ltd v Bentley 1990 (1) SA 680 (A).

¹² Minister of Safety and Security v Van Duivenboden 2002 (6) SA 431 (SCA) at para [25].

the problem was recognised rather than ignored. However, she conceded that an abnormal CTG was discovered.

[92] Mr Bodlani, for the plaintiff, submitted that had there been immediate delivery of ES after the onset of bradycardia, in all probability, she would not have sustained cerebral palsy. Mr Ntsaluba, on behalf of the defendant, argued that the management of the plaintiff's labour was proper and not substandard. He stated that EL was growth restricted and this was not and could not have been detected during the plaintiff's pregnancy as she only booked on 11 February 2014, a mere four weeks before 11 March 2014 when she went into labour. He added that EL's IUGR primed her for birth hypoxia and rapid progression of labour from 3cm dilation to full dilatation which would have contributed to the nuchal cord around her neck twice at a time when it was too late to detect and intervene effectively. He submitted that the rapid progression from the first to the second stage of labour, and the tightening of the nuchal cord twice around her neck, most probably caused the dominant acute profound of hypoxic-ischaemic brain injury which explained the picture depicted on the MRI scan. He submitted further that even if the defendant's employees could be found to have been negligent, the causal effect of the outcome was not that of negligence but rather the fact that the child was primed by IUGR for birth hypoxia and the tightening of the nuchal cord immediately before birth.

[93] The plaintiff pleaded that the negligence of the employees of the defendant in the management of her labour and delivery of [ES] who had developed foetal heart abnormality as a consequence of foetal deoxygenation did not properly attend to the matter (condition) in circumstances where it was necessary to do so. They failed to adhere to the standard of

practice of reasonable nurses and doctors in their respective positions to ensure that foetal deoxygenation was attended to without delay to prevent ES's development of a hypoxic-ischaemic injury.

[94] Regarding the evaluation of credibility and reliability of expert evidence, Wallis JA (Fourie and Koen AJJA concurring) in *Pricewaterhouse Coopers Incorporated and Others v National Potatoe Co-operative Ltd and Another* [2015] 2 All SA 403 (SCA)¹³ referred to *Wightman v Widdington (Succession de)* 2013 QCCA 1187 CanLII) where it was remarked thus:

“Legal principles and tools to assess credibility and reliability

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

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

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

[329] With respect to its probative value, the testimony of an expert is considered in the same manner as the testimony of an ordinary witness. The Court is not bound by the expert witness’s opinion.

[330] An expert witness’s objectivity and the credibility of his opinions may be called into question, namely, where he or she:

- accepts to perform his or her mandate in a restricted manner;
- presents a product influenced as to form or content by the exigencies of litigation;
- shows a lack of independence or a bias;
- has an interest in the outcome of the litigation, either because of a relationship with the party that retained his or her services or otherwise;
- advocates the position of the party that retained his or her services; or
- selectively examines only the evidence that supports his or her conclusions or accepts to examine only the evidence provided by the party that retained his or her services.”

¹³ At page 441 para [98]; see also *Coopers (South Africa) (Pty) Ltd v Deutsche Gesellschaft fur Schadlingsbekampung MbK* 1976 (3) SA 352 at 370.

[95] *In Ruto Flour Mills v Adelson (1) 1958 (4) SA 235 (T)*¹⁴ Boshoff J stated that a party seeking to introduce expert evidence must satisfy the court that the witness not only has specialist knowledge, training, skill or experience but that he/she can, on account of these attributes or qualities, assist the court in deciding the issue, that he/she is an expert for the purpose for which he/she has been called upon to express an opinion,¹⁵ the witness does not or will not express an opinion on hypothetical facts that have no bearing on the case or which cannot be reconciled with all the other evidence in the case.¹⁶

[96] With the above in mind, I considered the various experts' evidence on behalf of the plaintiff in support of her claim. I also took into account the evidence given by the defendant's experts. I was impressed by the evidence of the experts who testified on behalf of the plaintiff as they testified in their respective fields and made conclusions founded on sound factual and evidential basis. Regarding the evidence on record, the evidence of Drs Ndjapa and Kara, in their field of expertise, leaves no doubt in my mind that the cause of the injury to ES was a consequence of negligence on the part of the defendant's employees.

[97] The evidence of the defendant's experts, Dr Mtsi and Prof Cooper were not impressive as it was unsubstantiated by the facts of this case. Dr Mtsi could not interpret the CTGs and yet she still expressed an opinion which was speculative in nature and unacceptable, which she conceded. She relied so much on literature and not on material facts upon which her opinion was based. Prof Cooper's theory of IUGR and placental insufficiency

¹⁴ At 237C-D.

¹⁵ *Goliath v Fedgen Insurance Company Ltd* 1994 (2) PH F 31 E at 83.

¹⁶ *S v Mkohle* 1990 (1) SACR 95 (A) at 100d.

was also not supported by the evidence. Diemont JA in *S v Collop 1981 (1) SA 150 (A)*¹⁷ stated that although an expert witness may refer to textbooks and a doctor to medical treatises to refresh his/her memory, or to correct or confirm his opinion, such books are not evidence *per se*. Addleson J¹⁸ remarked that when an expert relies on passages in a text-book, it must be shown, firstly, that he can, by reason of his own training, affirm (at least in principle) the correctness of the statements in that book; and, secondly, that the work to which he refers is reliable in the sense that it has been written by a person of established repute or proved experience in that field, which was not the case here.

[98] Notwithstanding her misgivings, Dr Mtsi continued and interpreted the CTGs. She asserted that the last CTG depicted a worsening scenario than the previous one. This CTG coincided with the onset of strong contractions because all along the contractions were not long and strong. However, she agreed with Dr Ndjapa that at that stage there could have been quite a significant amount of activity before delivery nothing could have been done by way of caesarean section to salvage the situation. Surprisingly, she confirmed that no normal CTG was available before 20h00 and yet refused to accept that at 20h00 the problem that had started earlier, had deteriorated. She confirmed the evidence of Dr Ndjapa that when the foetus was bradycardic and there was a drop up to 100 beats per minute up to 100 beats per minute but picked up to 120 beats per minute that was sufficient warning that the baby's heart beat was abnormal.

CONCLUSION

¹⁷ At 167B.

¹⁸ *Menday v Protea Assurance Co Ltd 1976 (1) Sa 565 (E)* at 569H.

[99] In my view, the conduct of the defendant's employees was negligent as it was clearly not according to the Guidelines. They should have foreseen the reasonable possibility of their conduct causing harm to the foetus and should have taken steps to guard against such an occurrence. They failed to uphold this standard. The cause of ES' injury resulted from the negligent conduct of the defendant's employees, acting in the course and cope of their employment at Zithulele Hospital in respects already stated above. They failed to deliver the plaintiff by caesarean section due to poor progress in labour as at the time the plaintiff was not fully dilated. Bradycardia was noted at 17h20 and resuscitation that was done as an intervention until 20h50, more than three hours later. This fell short of the required standard. Instead, the injury was prolonged when it was obvious that there was no progress to the detriment of ES who developed cerebral palsy. Had immediate delivery of ES was done, she would not have been injured. There was no evidence of IUGR suggested by Dr Mtsi and Prof Cooper, as this was not supported by the evidence. The plaintiff's claim has to succeed as she has, on a balance of probabilities, successfully proved her case.

ORDER

[100] In the circumstances, I issue the following order:

- 1. The defendant shall pay 100% (one hundred percent) of the plaintiff's agreed or proven damages in her representative capacity for and on behalf of her minor child, ES, which damages flow from the neurological injury sustained by ES during labour and delivery at Zithulele Hospital on 11 March 2014 and the resultant cerebral palsy which she suffers from.**
- 2. The plaintiff's claim in her personal capacity and issues relating to quantum are postponed *sine die*.**

2. The defendant shall pay the plaintiff's taxed or agreed party and party costs on the High Court Scale, such costs to include (but not necessarily be limited to) the following:

2.1 The costs attendant upon the obtaining of the medico-legal reports and/or addendum reports and/or joint minutes, if any, of the expert witnesses in respect of which notices in terms of Rule 36(9) of the Rules of Court, were filed;

2.2 The qualifying and appearance fees of the expert witnesses in respect of which notices in terms of Rule 36(9) of the Rules of Court, were incurred;

2.3 The reasonable and necessary air transport and accommodation costs and expenses in respect of expert witnesses in respect of which notices in terms of Rule 36(9) of the Rules of Court were filed, where such fees were incurred; and

2.4 The reasonable fees of 2 (two) counsel, where such services were engaged, including the preparation of heads of argument and running of the trial on a virtual platform and in Court.

3. The defendant shall pay interest on the plaintiff's taxed or agreed costs of suit at the prescribed statutory rate calculated from a date (14) fourteen days after agreement in respect thereof, or a date 14 (fourteen) days after affixing of the Taxing Master's *allocatur*, to date of payment.

BM PAKATI

JUDGE OF THE HIGH COURT, GQEBERHA

Plaintiff's Counsel: Adv A Bodlani with Adv Ntikinca

Instructed by: Dayimani Inc

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Attorneys

Heard on: 26, 27,28, 29, January 2021, 01 February 2021, 03, 04, 05,06,07,10,11 May
2021, 22, 23,24, 25,28,29,30 June 2021, 01,02 July 2021, 27,28 June 2022.

Judgment delivered: 28 February 2023